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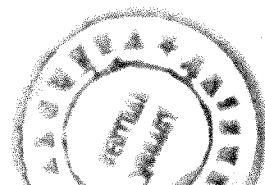
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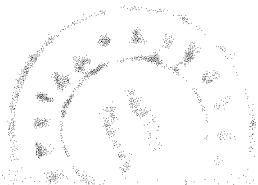
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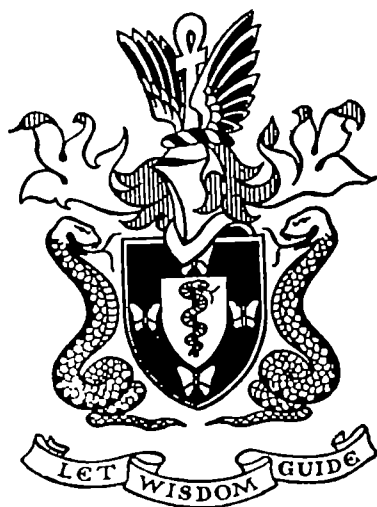
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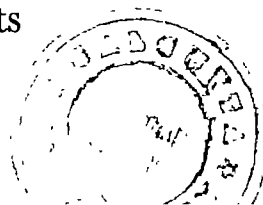
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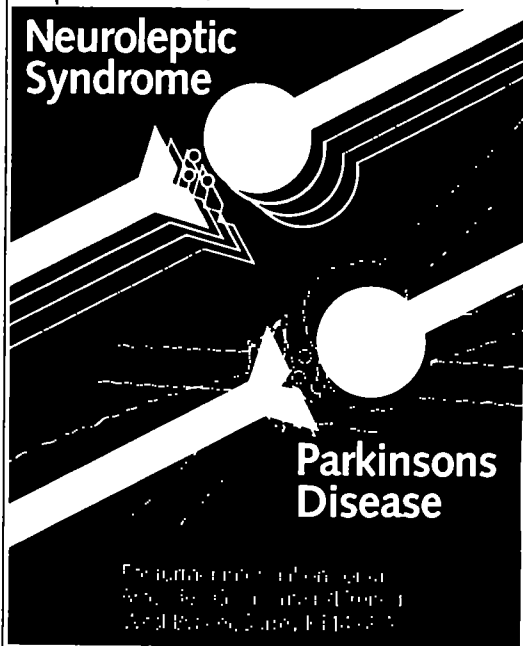
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Lecture

Different Causative Factors in Different Forms of Schizophrenia

KARL LEONHARD

Definite conclusions about the origin of schizophrenia concerning hereditary as well as psychosocial factors can be obtained when different forms of schizophrenia are investigated separately.

We still know much too little about schizophrenia. This applies particularly to its origin. Being an older psychiatrist, I am well aware of the extensive and intensive research work performed since the time of Kraepelin and Eugen Bleuler. Unfortunately, though, there has not been much progress. After the First World War, when Kraepelin and Bleuler were still alive, most psychiatrists believed they could regard schizophrenia as an inherited disease. This was generally accepted in Germany where the doctrine of Rüdin dominated research on the genetics of schizophrenia. However, he was influential not only in Germany, but in many other countries, too. For example, Kallman in the USA worked with Rüdin for several years. Rüdin and his pupils knew the extent of the gap which existed between practical observations and theoretical expectations according to Mendel's laws, and tried again and again to bridge the gap. They did so in vain. Many hypotheses were established. Some authors suggested a dominant mode of inheritance, others suggested a recessive heritability. Neither of the contrasting views held up to scrutiny. Nevertheless the belief that heredity was crucial continued, and this had disadvantageous effects on psychiatric research for, in consequence, the existence of other causative factors was completely disregarded. The result in Germany was horrible: from the alleged exclusive heritability of the endogenous psychoses Hitler derived the foundation of his laws by which he forcibly sterilised and even killed so many patients.

Partly as a reaction to these crimes, afterwards heredity was disregarded. Psychoanalytical and psychosocial theories now became completely dominant. When the North-Americans came to Germany

after the Second World War they attempted to convince us that schizophrenia would occur if a baby had been frustrated and had not been given his mother's milk in good time. This was the era of the "schizophrenogenic mother". Such psychoanalytical theories could not be upheld for long, though some of the psychosocial views arising subsequently were psychoanalytically oriented. The theories established by Lidz, Bateson, Wynne, Singer are generally known and very often cited. There has been much discussion of these theories, but after some attempts at replication, again, they did not hold up. Now many psychiatrists have returned to genetic theories and regard inheritance as decisive in causing schizophrenia. You can again find attempts at revealing a Mendelian mode of genetic transmission though all such attempts failed as long as 50 years ago. The wheel has turned full circle.

In my opinion, this regrettable state is due to the fact that schizophrenia is taken as a single entity, even though Eugen Bleuler did not speak of schizophrenia—but of "a group of schizophrenias". Indeed, definite results can be obtained concerning heredity as well as psychosocial factors when different forms of schizophrenia are investigated separately.

During the 15 years after I retired, I was able to devote myself entirely to research and was in a position to complement our knowledge of schizophrenia (through clinical and genetic examinations of more than 1400 patients). The classification on which my investigations are based have been made known in the English language from the publications by Fish, (1958, 1962, 1964), Astrup (1962), Perris (1960, 1974); and as far as my differentiation between the monopolar and the bipolar depressions is concerned, from many other publications. My classification has been presented in detail by Ban

*Opening lecture delivered at the Regional Symposium of the World Psychiatric Association, Rio de Janeiro, April 18-21, 1985

et al (1981). And my book *The Classification of Endogenous Psychoses* has also appeared in English (Leonhard, 1979).

Here, the "systematic" schizophrenias correspond to the "typical" schizophrenias, especially described by the late Japanese psychiatrist, Mitsuda. They constitute the "nuclear" schizophrenias and in almost all cases run an insidiously progressive course. The "non-systematic" schizophrenias correspond to the "atypical" schizophrenias and are predominantly remitting or even periodic. Also in their symptoms pattern they differ from the systematic forms (see Fish, 1958; 1962).

Among the endogeneous psychoses, 5 groups have to be differentiated. First: the systematic schizophrenias; second: the non-systematic schizophrenias; third: the cycloid psychoses must be separated from schizophrenia. They never lead to mental deterioration, and each phase is followed by complete recovery. The fourth and fifth groups comprise monopolar depressions and manic-depressive illness. These two forms are now generally accepted. In my opinion, there are also monopolar manias, the existence of which is mostly denied. The schizo-affective psychoses are not among the five groups. I cannot regard them as an independent group because their prognosis is indefinite. Some of these with schizo-affective psychoses recover from every phase and, in my opinion, have cycloid psychoses. Others tend to deteriorate, more and more, from shift to shift, and belong to the non-systematic schizophrenias. Whether the one psychosis is present or the other, can be identified from the distinct psychiatric picture. Thus the schizo-affective psychoses have to be separated into these two different groups (Leonhard, 1983).

The five groups of endogenous psychoses, especially the two schizophrenias and the cycloid psychoses, have quite different aetiologies. First, different inheritance: 604 systematic schizophrenics whom we examined had 1403 siblings—only 29 of them, i.e., 2.1% were ill; our 429 non-systematic schizophrenias had 744 siblings—117, i.e., 15.7% were ill. In these figures Weinberg's correction has been used. Individuals under 17 years have not been counted, individuals aged from 17 to 40 have been counted half, individuals over 40 have been fully counted.

So, in the severest forms an extremely small family history of psychotic disorder is found. In these forms psychosocial factors must be looked for. Genetic loading is also low in the cycloid psychoses, but not to so large an extent as in the systematic schizophrenias. Our 221 cycloid psychotic patients had 393 siblings—16 of them, i.e., 4.1 per cent were

ill. Thus family history of psychotic disorder is low in the severest as well as in the mildest forms of the so-called schizophrenias. It is striking that our absolute figures differ so greatly in size. This is due to the fact that we undertook our investigations in hospitals where chronic patients were prevalent. In hospitals with predominantly acute psychoses the cycloid forms are as common as the systematic and as the non-systematic schizophrenias.

I found a low hereditary loading in the systematic schizophrenias when I started my investigations into the endogenous psychoses at the beginning of my psychiatric life; but over the years I could not detect any external influences. On the basis of the examination of twins I ultimately succeeded. I have personally examined 45 monozygotic twins and 25 ill co-twins, and, in addition, 47 dizygotic twins. Among the latter all forms of endogenous psychoses occurred. By contrast, in the monozygotic twins systematic schizophrenias were completely lacking. This was extremely surprising and totally unexpected. The finding applied to the 45 index-twins and the 25 ill co-twins as well. It seems that we encountered an external factor involved in the causation of the systematic schizophrenias. The finding gives rise to the conclusion that identical twin-ship can protect the individual from falling ill with this special kind of schizophrenia. How can this be explained? I think we must give consideration to the fact that monozygotic twins being identical in their somatic and psychical constitution understand one other to so complete a degree that they cannot become entirely autistic. And we know since Eugen Bleuler that autism is a central symptom of schizophrenia. The close communication with the co-twin seems to compensate sufficiently for the little contact which may exist with other people. Following our explanation we can assume that, in turn, lack of communication, lacking even one single friend, can encourage the development of schizophrenia.

Our findings contradict the psychosocial theory according to which identical twins have difficulties in finding their identity and therefore both of them are prone to the development of schizophrenia. On the contrary they are liable to schizophrenia to a lesser degree. However, I must add that other forms of endogenous psychoses accumulate in twins. I will return to this later.

Many authors have discussed the significance of isolation and lack of communication in the origin of schizophrenia. They could not obtain definite results as they took schizophrenia as a unity. Moreover, they predominantly deal with the concept isolation as it is present in adults, whereas, according to our observations lack of communi-

cation is only predisposing towards schizophrenia when operating in childhood.

Communication between twins is psychosocial in nature, lack of communication is also a psychosocial factor; but the effect on the patient seems to be organic, for the systematic schizophrenias, which in most cases terminate in serious deterioration, certainly have an organic basis. This kind of influence is possible in childhood and adolescence when the nervous system has not matured. Isolation can result in such consequences. Let me quote the brain pathologist Seitelberger (1980):

"The maturation not only of the sensory system and the motor apparatus, but also of all parts of the brain which of necessity work as a unit, is dependent on the external demands made. It has been shown that systems which are kept from functioning by means of isolation do not attain their morphological maturation and remain non-functional. On exposure to external influences after the critical maturation period has been passed the deficit can no longer be compensated. The processes of morphological differentiation are to a continually decreasing degree demonstrable in the nerve-cells and their associative fibres until the end of the second decade of life. So, these changes accompany the processes of mental maturation in the individual."

So, the organic effect of isolation can be demonstrated morphologically. Such an influence was observed in the children described by Spitz (1945) in whom lack of communication resulted in severe somatic disturbances; many of the children died though they had been well fed, and children who survived became severely mentally retarded.

In the cycloid psychoses nothing is suggestive of an organic basis. Accordingly their origin has nothing in common with that of the systematic schizophrenias. As regards heredity, surprising results appeared when we differentiated between two sub-forms, anxiety-happiness-psychosis and motility psychosis. In both these cases the family history of psychiatric disorder is small, in siblings 4.1% and 4.2% respectively. Nevertheless the concordance rates in identical twins differ greatly. Of our six pairs with anxiety-happiness-psychosis only one was concordant; this conforms with the low hereditary loading. But of our 11 pairs with motility psychosis no less than 9 had also fallen ill. From this latter result the geneticists would derive an extremely great heritability which, in fact, does not exist. In motility psychosis no more siblings were ill than in anxiety-happiness-psychosis. In the parents, there were fewer ill individuals in motility psychosis (5.1% versus 6.3%). The old rule established by Galton and thereafter being an important guideline for geneticists, loses its validity with the cycloid psychoses. Indeed, in spite of the extraordinarily high concordance rate in identical twins the heritability of motility psychosis is low. I will give an interpretation of this finding later. In our twins with monopolar depression the concordance rate was also high though relatively few parents and siblings were ill.

Alarmed by the finding in our twin studies we focused on sibships, their sizes, and the position of the patient

among the siblings. In this regard many investigations have been carried out by other authors, again considering schizophrenia as a whole. Various and conflicting results have been obtained. We again differentiated between different forms and many definite results became apparent. I came to the conclusion that siblings by mutual influences contribute to the development of the individual in childhood and adolescence and do so not only psychologically but also biologically. On this basis the influence of siblings can prevent or, on the contrary, encourage the appearance of schizophrenia. Childhood friends may play the same role as siblings.

Let me consider cycloid psychoses in this respect. Striking results appeared when I compared motility psychosis with another sub-form, confusion psychosis. There were relatively many siblings in the first illness and relatively few in the latter. However, the difference applied only to the older siblings. In our series, 100 patients with motility psychosis had 172 older siblings, and 100 patients with confusion psychosis had only 78. The number of younger siblings differed little (127:108). This latter difference is not statistically significant. I conclude that the alleged mutual influence of siblings occurs in the first years of life when younger siblings either do not exist or are so helpless that they cannot exert any influence on older siblings. This fits into the psychoanalytic view about the first years of life. I do not discuss the question why it applies just to these cycloid psychoses whereas in other endogenous forms the external influence is operating during later childhood. And I am not able to answer the question why the existence of many siblings contributes to the origin of motility psychosis and the lack of siblings to the origin of confusion psychosis. However, I point out an interesting parallel. Motility psychosis often occurs in twins. Of our 92 twins 22 (11 monozygotic, 11 dizygotic) suffered from this cycloid psychosis. Perhaps the close relationship existing in twins corresponds to the increased relationship resulting from a greater number of siblings and thus either condition enhances the morbidity risk for motility psychosis. Probably siblings can stimulate each other too much so that the nervous system can be overstrained.

At this point I should like to return to the question of why there are some different concordance rates in identical twins with anxiety-happiness-psychosis and, on the other hand, with motility psychosis. In the latter case the influence is mutually exerted, both twins are involved. In contrast, in anxiety-happiness-psychosis there is only a one-sided influence. From other findings, I came to the conclusion that tensions in childhood can create a disposition to anxiety-happiness-psychosis and jealousy of the inferior twin toward the partner can represent the basis of such tensions. In this case for the dominant twin there is no reason to be jealous and he will remain well. So the influence is one-sided. One of our identical twin pairs gives support to this interpretation. The index twin sustained a fracture of the vertebral column at the age of 12 and had to lie on a plaster bed for several years. Moreover her father favoured the co-twin. The mother tried to compensate for this, but the father was entirely dominating in the family so that his affection was of greater importance than that of the mother. The index-twin fell ill with anxiety-happiness-

psychosis while the co-twin remained healthy. She became jealous of her sister who was somatically healthy and was favoured by the father. She continuously fought her sister in an attempt to gain ascendancy.

Concerning the non-systematic schizophrenias, I must first point out the high hereditary loading. For one of these forms (periodic catatonia) I could suggest a dominant inheritance pattern. The gap existing between the Mendelian estimates and the empirical findings could be bridged by examining many of the allegedly healthy parents. In fact, many of them were seriously abnormal. We found that in periodic catatonia 22.0% of the parents were ill and approximately as many were abnormal to a high degree. Thereby the Mendelian estimate is nearly matched. However, when the psychosis manifests itself completely external factors must operate in addition.

We found that periodic catatonics have relatively few siblings. This contrasts with the findings in motility psychosis though there are certain similarities between the two psychoses. As in periodic catatonia, many of the parents were psychotic—the time for propagation was possibly limited. But it turned out that healthy parents had only a few more children than psychotic parents. It seems then that the manifestation of periodic catatonia decreases when many siblings exist. Direct evidence of this emerged when we examined how many ill siblings came from large sibships and how many from small sibships. Many more ill siblings were found in small sibships. The figures were 29.4:17.6 in relation to the number of healthy siblings.

Some families seemed to confirm that large sibships can prevent the manifestation of periodic catatonia. This applied to the following family which contained no less than 15 siblings. Two of them had children with periodic catatonia. Thus it can be seen that the hereditary predisposition for periodic catatonia existed in the family. Nevertheless all of the 15 siblings remained healthy. I think the large sibship protected them from falling ill.

What factor might be operating to produce this effect? I suggest that a child feels protected and sheltered when living in companionship with several siblings so that he is more likely to grow up free from anxieties. Such a view fits into some psychosocial assumptions. With respect to periodic catatonia, I can confirm this view. One of our observations seems at first to contradict our interpretation, but on the contrary it supports it when the family is more completely examined. Of 10 siblings, no less than four fell ill with periodic catatonia. The existence of so many children was by no means preventive. The Mendelian estimates were nearly reached; half of the children are expected to fall ill in a dominant inheritance.

But let me describe this family thoroughly: the father was an excessive drinker and most of the time he used to sit in pubs. He used to beat his children brutally. When a little child cried he beat him or he beat an older child whom he declared responsible for the crying. As soon as the father returned home the whole family became silent; and the children made themselves scarce. The mother tried to intervene, but was rejected and was often beaten herself. The children in this family grew up under continuous anxieties; the large sibship could not prevent this.

Another psychosocial theory was not confirmed. Some

psychiatrists believe that the abnormal behaviour of a schizophrenic mother can cause an illness in the child. The assumption again has been related to schizophrenia as a whole, but periodic catatonia represents the schizophrenic form in which most frequently parents as well as children are ill, so that the validity of the theory can be tested. Heston (1966) opposed the assumption after he had examined children of schizophrenic mothers who had grown up in a home. Our patients who had catatonic mothers did not have more ill siblings than patients with healthy mothers; on the contrary they had fewer. But in spite of this the index-patients relatively often had psychotic mothers, when there was no older sister. This is astonishing and indicates that an older sister is qualified or even biologically designed to replace the mother who has been lost or is mentally incapable. Now we understand why index-patients with ill parents have not more, but fewer ill siblings than index-patients with healthy parents. In the first case the siblings could remain healthy because most of them had older sisters, for example the index individual who later fell ill. In the event of a healthy mother, the presence or absence of an older sister is without significance. If we are surprised that the existence of an older sister can reduce the risk for periodic catatonia we must remember how unselfishly older sisters often devote themselves to the care of younger siblings after the mother has been lost. According to my findings the above psychosocial theory must be corrected as follows: it is not the abnormal behaviour of a psychotic mother, but loss of the mother in association with absence of an older sister which may be important. However, in any case, the external influence does not cause periodic catatonia which is a hereditary illness, but only increases its manifestation.

Another non-systematic schizophrenia with a hereditary basis is affective paraphrenia. In this psychosis a recessive mode of inheritance is suspected because, on the one hand, we found relatively many ill siblings (13.3%) and very few ill parents (2.2%). On the other hand, I could describe a large family containing 18 affective paraphrenics of whom no less than 13 descended from consanguineous marriages. Both these findings suggest a recessive inheritance. The probability of manifestation is increased in females; in our series 95 affective paraphrenics were females and only 25 males. As far as affective form of paraphrenia is concerned, we may assume that affective lability, representing a personality trait of the women, can promote the onset of the illness.

As regards psychosocial factors, some psychiatrists believe that children become liable to schizophrenia if they cannot acquire the role belonging to them by age and sex. In most forms of schizophrenia I did not find anything fitting this theory, but in affective paraphrenia I did. Repeatedly, I was able to confirm psychosocial theories about the genesis of schizophrenia, but in no case concerning schizophrenia as a whole. Each theory applied only to one of the different forms of schizophrenia.

The affective paraphrenics are frequently the youngest of the siblings. Among our 120 affective paraphrenics there were 36 youngest and only 17 oldest siblings. We know that the youngest children are often kept in the role of a child by their parents when they should be allured to

mature. Observations in identical twins are suggestive in this sense. In one case the maturation of both twins had been retarded because the rigorous and harsh father had suppressed the activity and affectivity of the girls. This could have applied even more to the second born twin who fell ill seven years earlier than her sister. In another case the girls were never able to live an independent life. They were not allowed to undertake any activity without asking permission from their mother. In adolescence they were not allowed to make date with a young man. An incident demonstrates the high degree to which the mother dominated her daughters. When one of them was over 30 years old and had been divorced she wanted to marry another man whom her mother did not approve of. As she attempted to resist her mother she was beaten by her. Later she fell ill with affective paraphrenia. Her identical co-twin was even more submissive and fell ill with this psychosis 22 years earlier than her sister. I did not make similar observations in other forms of schizophrenia.

Maturation is retarded to the same extent when overprotection from the mother makes independent life impossible. I observed this in an identical twin recently. The mother loved one of her children very much and always wanted to keep the girl at home. While the co-twin was with friends the index-twin was with her mother. When, in adolescence, the girl wished to go out with her sister, the mother tried to prevent this and sometimes even shed tears in grief. In contrast, the co-twin had much personal liberty and was always with friends. The different attitude of the mother toward her children had developed because the index-twin was less lively and more submissive. She fell ill with affective paraphrenia at the age of 58. The co-twin remained well until the age of 74. Then she also expressed some ideas of reference; but it was difficult to determine whether she had now become psychotic or she was only susceptible to suggestion in consequence of her old age and had accepted some of her sisters ideas. In any case, the index-twin, who was overprotected by her mother, was much more severely ill. Her maturation had been hampered.

As in this case, identical twins not infrequently have different temperaments. I particularly found this with our affective paraphrenics. In three of our seven cases the one twin was lively, the other calm. In all the three cases the twin with the calm temperament fell ill. Of the partners, apart from the co-twin just mentioned, one remained healthy, the other was much milder ill than her sister. In all cases I saw both twins and personally observed that the co-twin was much more lively. One of them was even markedly hypomanic. We may enquire whether, in the patient described overprotection and submissiveness were crucial, or, perhaps the underlying temperament alone evoked affective paraphrenia. The other two twins had not been overprotected, but were submissive to their co-twins, one of them to a high degree.

Let me now turn to the third non-systematic schizophrenia, the cataphasia, which in its excited pole corresponds to the schizophasia described by Kraepelin. Here we found that the patients relatively often originated from big cities (over 100 000 inhabitants) and relatively rarely from villages (below 2000 inhabitants). The central symp-

tom of cataphasia is represented by a disorder of thought. Thus we might suspect that the function of thinking can be damaged in big cities where there is often an excess of mental stimulation. Cognitive functions development in childhood and adolescence may be overstrained. This is a very interesting aspect which arouses the idea that in our modern time the occurrence of cataphasia must have increased, for technical development has so rapidly progressed and thinking is much more strained than in former times. Indeed, we found the incidence of cataphasia had increased. We examined our 1400 patients during the course of 15 years. We made three groups and separated patients examined in the first five years, in the second five years, in the third five years and then in each group we compared the numbers of patients suffering from the different forms of schizophrenia. The number of cataphasias had increased from group to group, relative to the other forms.

Finally, I should like to discuss manic-depressive illness and "pure" depressions. The hereditary loading of these two forms is more different than other authors have found. When differentiating according to course (monopolar versus bipolar) many manic-depressives are erroneously assigned to the pure depressions as they by chance had only gone through depressive phases, which generally are much more common. The cross-sectional picture is also different in the two conditions. We have differentiated on this basis and found 20.0% ill siblings and 18.3% ill parents with manic-depressive illness; but only 3.1% ill siblings and 5.8% ill parents with pure depressions. In the first case a dominant heritability can be assumed though external factors must contribute to the manifestation of the illness. In this psychosis the abnormal behaviour of a psychotic parent seems to be significant, for probands with psychotic parents had 28.1% ill siblings, and probands with healthy parents only 15.5%, though these "healthy" parents had the full hereditary predisposition to the illness.

Among our pure phasic psychoses the euphoric form showed still fewer ill relatives than the depressive form. The figures just mentioned (3.1% and 5.8%) include the euphoric form. After separating the two psychoses we found 4.5% ill siblings and 7.8% ill parents in the depressives, whereas in the manias there were 1.2% ill siblings and 2.1% ill parents. In the euphoric form family history is less prominent. As noted above, the existence of pure manias is still predominantly denied. The low figures demonstrate that these patients belong even less than depressives to manic-depressive illness with its high hereditary loading. So monopolar mania is an independent entity, too.

Pure manics have relatively many siblings, pure depressives relatively few. However, just as we found in two of the cycloid psychoses, this difference concerns only the older siblings, so that I again conclude that a mutual influence operates in early childhood. As siblings stimulate each other we can envisage the possibility that too much stimulation in childhood may promote the occurrence of mania, while too little stimulation may promote the occurrence of depression. If this is the case, not only siblings, but also other people (above all parents) can stimulate the child too much or, on the other hand, not enough. Seven

purely depressive probands had a depressive parent (six females and one male), one purely euphoric patient had an ill mother who was euphoric, too. Perhaps manic parents stimulate their children too much, depressive parents too little. Hereditary influences are not decisive, since nearly all of the ill parents were mothers (seven of eight parents).

The finding that manics have many siblings and that depressives have few has an important transcultural aspect. In developing countries mania is relatively common, in industrial countries depression much more frequently occurs. This difference was noted by Kraepelin after he visited psychiatric institutions in Java. There is a parallel, in developing countries families are large, in industrial countries families are small. Various causes may account for the different incidence rate of manias and depressions, but a dependence on family sizes also has to be taken into account. I do not know whether in industrial countries mania was more common in former times when families were larger. In this connection, the question may be raised whether the incidence of schizophrenia has increased in our century. My opinion is: some forms of schizophrenia have increased, others have decreased. The insidiously progressive schizophrenias, i.e., the systematic

forms, in my classification, are certainly more common at the present time. Some remitting and periodic forms occurred more frequently in former times.

A similar difference in the occurrence of schizophrenia seems to exist between industrial and developing countries as many authors have described. See Jilek (1974) and Jilek & Jilek-Aall (1970). Motility psychosis is usually assigned to the schizophrenias, and runs a markedly periodic course and probably occurs more frequently in developing countries. Since in these countries the families are larger, this fits with our finding that patients with motility psychosis have relatively many siblings.

Let me close with a general remark. You may ask why I still speak of endogenous psychoses though I presented evidence of many external influences. Why do I not add "so-called"? The external events create a disposition to psychoses in childhood and adolescence. From this time onwards, the individual has an internal propensity from which later the psychosis originates. No external influence is usually operative at the onset of this illness. In the exogenous psychoses the illness immediately follows the external influence.

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The Relationship of Anxiety and Depression: A Review of the Literature

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The nature of the relationship between anxiety and depression has been much debated. The research in the past 15 years is reviewed in the context of three conceptual models: (a) anxiety and depression differ quantitatively; (b) anxiety and depression differ qualitatively; and (c) combined anxiety and depression syndromes (anxious depressions) differ both quantitatively and qualitatively from either pure anxiety or pure depression. The major areas of research—phenomenological, treatment, course and outcome—are considered and findings in support of each position reviewed.

In recent years, there has been an increased emphasis on the phenomenology of the various disorders of mood. This has received its impetus from attempts to construct a clinically relevant taxonomy of the affective disorders by identifying operationally defined criteria to delineate one disorder from another. This is difficult because of complex symptom patterns in which there is considerable overlapping of symptomatology between two disorders. Such is the case with anxiety and depression. There are many anxious patients who present with concurrent symptoms of depression, and many depressed patients who present with concurrent symptoms of anxiety. How these patients are diagnosed has implications for both treatment and prognosis.

This paper reviews the literature on the relationship between anxiety and depression over the last 15 years. It is not an exhaustive review but an attempt to consider the major research areas for future study. An overview of the nosological problems inherent in understanding how anxiety disorders and affective disorders are related can be found in Snaith (1981).

Review of Existing Literature

Currently there are two opposing conceptual models, as well as a third intermediary position defining how anxiety and depression are related. These focus on anxiety and depression as; (a) variants of the same

disorder differing quantitatively (unitary model); (b) distinct disorders differing qualitatively (pluralistic model); and (c) a mixture of the two syndromes, phenomenologically different from either primary anxiety or primary depression (anxious depressive position). The following is a description of each position, the variables on which anxiety and depression have been compared, and the research findings on which the model is based.

Unitary Position

Clinical variables: The overlapping of clinical symptomatology between anxiety and depression has been cited as the greatest source of support for the conceptualisation of the two disorders as represented by a continuum. Gersh & Fowles (1979) conceive of them as two symptomatic stages of affective disorder with the ratio of anxiety and depressive symptoms varying over time such that the diagnosis depends upon when in the course of illness the evaluation is made. This was supported by Kendell (1974), who studied the stability of clinical diagnosis over a period of five years: comparing the diagnoses of 2,000 patients for two separate hospital admissions, there was a change in diagnosis from anxiety to depressive disorder for 24%, while for 2%, the change was in the opposite direction.

Studies of patients suffering from anxiety neurosis have found a high prevalence of depressive symptoms, severe enough to qualify for a secondary diagnosis of depression, while secondary anxiety has been found to occur in a comparable percentage of depressed patients (Fawcett & Kravitz, 1983). Estimates of depressive symptoms in anxious patients are as high as 65% (Roth *et al*, 1972): irritability, mild agoraphobia, anxiety, agitation, and ideas of guilt were common in both the anxious and depressed patients in their sample. Clancy *et al* reported that 44% of their sample of 112 anxious patients, interviewed six years after their key illness, were diagnosed as having a secondary depression in

TABLE I
Unitary position

<i>Study</i>	<i>Subject population</i>	<i>Variable(s) examined</i>	<i>Results</i>
Roth <i>et al</i> , 1972	Anxious and depressed inpatients	Clinical symptoms	65% of anxious patients had concurrent depressive symptoms
Fawcett & Kravitz, 1983	Research Diagnostic Criteria for major depression	Clinical symptoms	42% of major depressive disorder patients had moderate worry; 29% had history of panic attacks; 19% had moderate phobic symptoms
Dealy <i>et al</i> , 1981	General anxiety or panic attacks	Clinical symptoms	33% had secondary depression
Clancy <i>et al</i> , 1978	Experimental-anxiety neurotic and general medical patient control—surgical patients	Clinical symptoms	44% of anxious neurotics had secondary depression
	Secondary depression	Family history	No differences in family history for anxiety and/or depression between pure anxiety group and mixed anxiety depression group
		Course/outcome	Average of five years from onset of anxiety to development of depression
Lesse, 1982	Anxiety patients	Course/outcome	Stress—anxiety—depression axis
Kelly & Walter, 1969	3 clinical groups, 1 normal group	Personality traits	No difference in neuroticism score (Maudsley Personality Inventory) of anxious and depressed patients
Johnstone <i>et al</i> , 1980	Unselected non-psychotic outpatients with scores of 2+ on observed anxiety, depression, or both	Treatment response	Amitriptyline resulted in improvement for both anxiety and depression
		Rating scales	Ratings of anxiety and depression did not discriminate between the two groups; correlation between two sets of scales increased over time
Mendels <i>et al</i> , 1972	Female psychiatric inpatients	Rating scales	Failure to find separate anxiety depression factors, high correlation between anxiety and depression rating scales
Schapira <i>et al</i> , 1971	Anxious and depressed inpatients	Prognosis	Anxiety states of long standing tended to acquire depressive symptoms

addition to the primary diagnosis of anxiety, compared to 7% of a surgical control group. Their findings were replicated by Dealy *et al* who reported that 33% of their patients had a diagnosis of secondary depression. An association between panic attacks (anxiety neurosis) and depression has also been found: Dealy *et al* reported that 64% of their anxious patients who experienced panic attacks had secondary depression, compared to 29% without secondary depression, while analysis of the data of Fawcett & Kravitz revealed that 29% of their depressed sample had concomitant panic attacks.

Both Clancy *et al* and Dealy *et al* in their studies of anxious neurotic patients who were either diagnosed as having a secondary depression or not, found no difference between the two groups in terms

of mean age of onset, duration of illness, suicide attempts, or family history of psychiatric illness. This led them to conclude that depression and anxiety were clinical variants of the same disorder.

Treatment and treatment response: Further support for an unitary orientation comes from treatment studies in which anxious patients have responded favourably to antidepressants (Kelly *et al*, 1969; King, 1962; Sargant & Dally, 1962), and for which success has been reported with anxiolytics in depressed patients (Henry *et al*, 1970; Hollister *et al*, 1967; Overall *et al*, 1966). Johnstone *et al* (1980) found no significant drug effect which could differentiate patients on the basis of either clinical or

self-ratings of anxiety and depression: amitriptyline proved to be superior to diazepam in the treatment of both anxious and depressed patients.

Course and outcome: The concept of anxiety and depression as a continuum has been further supported by research which has hypothesised the aetiological factor of anxiety in depression. Roth *et al* (1972) reported an early age of onset for anxiety compared with depression, and noted that long-standing anxiety states tended to acquire predominant depressive characteristics with the passage of time (Schapira *et al*, 1972); Clancy *et al* found an average length of five years between the onset of anxiety and the development of secondary depression. In agreement with this position is the stress→anxiety→depression axis postulated by Lesse (1982) to explain the relationship between the two disorders. While not ruling out the possibility that in some situations, depression may be the primary response to stress, Lesse stated that he had not been able to document an acute or subacute severely depressed state in a patient who had not manifested symptoms and signs of anxiety prior to the onset of depression. Hays (1964) also reported that many depressive reactions are preceded by prodromal periods of chronic anxiety.

Rating scales: The failure to find separate factors of anxiety and depression (Mendels, 1972; Johnstone *et al*, 1980) in anxiety and depression rating scales, and the high correlation between the two sets of scales have also been cited as evidence for the quantitative model. Mendels *et al* (1972) factor-analysed the scores of six self-rating mood scales; analysis of the data for 76 female psychiatric inpatients resulted in the extraction of two factors accounting for 68% of the total variance. All of the scales, except for two of the MMPI, loaded highly on the first factor, suggesting that it constituted a dimension of general psychiatric disturbance. They concluded that in a psychiatric sample, homogeneous as to sex, self-rating scales of anxiety and depression do not separate different sets of symptoms.

This failure to find separate factors might have been dismissed as being due to the relative unreliability of self-reports of behaviour, but it was also demonstrated to be true of observer-rated scales. In their study of treatment response to anxiolytics and antidepressants, Johnstone *et al* rated their patients on the Hamilton Depression Inventory and the Hamilton Anxiety Inventory at several specified

time-intervals during the trials. For all patients, the correlation between ratings of anxiety and depression increased over time; at week one, it was +0.53, while at week five it had increased to +0.77.

Thus, the unitary position for the relationship of anxiety and depression is supported by the overlapping of symptomatology between the two syndromes, by the lack of stability of the clinical diagnosis between anxiety and depression, by the similarity of anxious patients with and without secondary depression on several variables, by the tendency for patients suffering from long-standing anxiety states to develop depressive symptoms, by the failure to find separate dimensions of anxiety and depression in both self-rated and observer-rated scales, and by the lack of a specific response to drug treatment.

Pluralistic position

The most widely published advocates of the position that anxiety and depression are separate and distinct entities have been Roth *et al* (Newcastle Group). Their studies showed a separation between clinical groups of anxious and depressed patients in terms of a number of variables—clinical symptoms, personality, treatment and treatment response, course and outcome, prognosis, and rating scales. This Group, while cognisant of the overlapping of symptomatology between depression and anxiety, suggested that with the use of appropriate statistical methods, the two disorders could be shown to differ on certain dimensions. Tentative support for their viewpoint has been achieved, using the statistical techniques of principal components analysis and discriminant function analysis (Prusoff & Klerman, 1974; Derogatis *et al*, 1972a), although others have failed to replicate their results (Mendels, 1972; Johnstone *et al*, 1980).

The Newcastle Group published a series of studies establishing the independence of anxiety and depression. A sample of 145 inpatients, diagnosed as anxious or depressed on the basis of their predominant mood state, were evaluated—at the key illness and at follow-up—in terms of clinical variables, prescribed treatment and treatment response, personality and an outcome component used to assess prognosis. Mountjoy & Roth (1982a & b) replicated the earlier research on clinical variables, and extended the analysis to rating scales, but unlike the previous sample of inpatients, this one excluded patients with endogenous depression and included daypatients and outpatients.

TABLE II
Pluralistic position

<i>Study</i>	<i>Subject population</i>	<i>Variable(s) examined</i>	<i>Results</i>
Mountjoy & Roth, 1982(b)	Anxious and depressed inpatients, outpatients, and day patients	Clinical symptoms	Principal components identified a bipolar factor; discriminant function analysis separated the two groups
Newcastle Group, 1970-72, Roth <i>et al</i> , 1972, Schapira <i>et al</i> , 1971, Kerr <i>et al</i> , 1970, 1972, Gurney <i>et al</i> , 1970, 1972	Anxious and depressed inpatients	Personality traits	Anxiety characterised by personality traits of dependence, immaturity, hysteria, neuroticism, and poor social adjustment; depressed more stable, mature and independent
		Treatment/treatment response	Anxiety states high neuroticism scores on Maudsley Personality Inventory; depression higher extroversion scores
		Course/outcome	Depressed group more frequently prescribed ECT and tricyclics; anxiety group treated with sedatives & tranquillisers; better response to ECT and tricyclics for depressed group at discharge and at six month follow-up
		Prognosis	Anxiety states had an earlier onset and a longer duration
		Family history	Poorer prognosis for anxiety states
Zitrin <i>et al</i> , 1980	Agoraphobics	Treatment response	Greater neurotic illness in families of anxious patients
Mountjoy & Roth, 1982(a)	Anxious and depressed inpatients, outpatients, and day patients	Rating scales	Negative relationship between depression in agoraphobics and response to imipramine
Prusoff & Klerman, 1974	Anxious and depressed outpatients	Rating scales	Separate anxiety and depression dimensions
Derogatis <i>et al</i> , 1972(a)	Outpatients with primary anxiety or depression	Rating scales	Discriminant function analysis of Symptom Checklist factors discriminated between anxious and depressed groups
Crowe <i>et al</i> , 1980	DSM III criteria for panic disorder	Family history	Identified depression factor in Symptom Checklist ratings of anxious and depressed groups; could not identify anxiety factor in depressed group
Van Valkenburg <i>et al</i> , 1984	Anxious neurosis and depressed patients	Family history	Morbidity risk for panic attacks but not for other anxiety disorders or depression higher in anxious neurotics
Kelly & Walter, 1969	Experimental-nonagitated depression control-normal	Response to stress	Greater family history for depression in major depressive disorder patients
			Anxious patients had mean basal forearm blood flow significantly greater than that of depressed patients

Clinical symptoms: Analysis of clinical symptoms for the anxious and depressed groups of the Newcastle studies demonstrated an earlier age of onset, greater prevalence of psychiatric disturbance in first-degree relatives, poorer social adjustment, personality traits of dependence and immaturity, and longer duration

of the illness for the anxiety states, compared with the depressive illness (Roth *et al*, 1972).

Personality characteristics: Personality differences between the groups on the Maudsley Personality

Inventory (Kerr *et al.*, 1970) related to higher neuroticism scores and lower extraversion scores for anxiety states, compared to depressive states, both at key illness and at follow-up; it was concluded that the extraversion dimension represents a more stable aspect of personality, while neuroticism is primarily a measure of anxiety. These results were not replicated by Kelly & Walters (1969), who found no difference between their anxious and depressed patients in terms of the neuroticism score on the Maudsley Personality Inventory.

Treatment and treatment response: The anxious and depressed groups of the Newcastle studies also differed both in the prescribed treatment and in their response to two of four treatment strategies. Both ECT and tricyclic antidepressants were significantly more beneficial for the depressed group, in contrast to the anxious group; 24 out of the 28 depressed patients improved with ECT, compared with two out of the nine anxious patients; 23 out of the 27 depressed patients benefited from treatment with tricyclic antidepressants, whereas only five out of the 18 in the anxious group showed improvement.

Prognosis: The differentiation between anxiety states and depressive illnesses made on clinical grounds was upheld by differences in prognosis (Schapira *et al.*, 1972): there was a consistent tendency for the whole range of symptoms studied at follow-up to be more frequent and more severe in the anxious group of patients. A principal components analysis, carried out on 53 items used in the assessment of mental state at follow-up, showed a better prognosis for depressive patients in terms of global adjustment.

Rating scales: Mountjoy & Roth (1982a) factor-analysed the scores from both observer-rated (Hamilton Scales for Anxiety and Depression, Newcastle Anxiety Depression Scale) and patient-rated scales (Zung Depression Scale, a scale for anxiety, phobic scales for agoraphobia and for school phobia). All of the scales loaded positively on the first component, which accounted for 44% of the variance; it was interpreted as a general factor of severity, and there was no significant difference between the diagnostic groups in their mean scores on this component. The second component was bipolar, and a subsequent discriminant function analysis separated the two clinical groups at a statistically significant level, resulting in a miss-classification rate of 32%.

Prusoff & Klerman (1974) analysed the Symptom Checklist, a 58-item self-report inventory. Their sample consisted of 364 anxious outpatients and of 364 depressed patients, matched with the anxious group for race, sex, age, and social class. Previous factor analysis of the Symptom Checklist had identified five separate factors: the patients' scores on these factors were analysed by discriminant function analysis. The depression and somaticism factors contributed to the discrimination between the groups, while the anxiety factor failed to make a significant contribution to the discrimination. Similar results were reported by Derogatis *et al.* (1972a), who examined the factorial invariance for the same scale. A factor analysis of the scores of 641 anxious neurotic outpatients and 251 neurotic depressed patients identified a depression factor in both groups, but the anxiety factor was only identified in the anxious group. It was concluded that the different pattern of factor loadings of the two groups on the depression dimension were evidence for differences between anxious and depressive syndromes.

Family history: Several investigators have cited differences in terms of family history between the two disorders as supportive of the pluralistic model. Roth *et al.* found a greater prevalence of neurotic illness and personality disorder in first-degree relatives of anxious patients, whereas van Valkenburg *et al.* (1984) found a greater family history for depression in patients diagnosed as primary affective disorder. Crowe *et al.* (1980), in a sample of patients with anxiety neurosis (panic attacks), reported a higher morbidity risk in first-degree relatives for panic disorder but not for other anxiety disorders or for depression.

Physiological: There is physiological evidence in support of the separate entity hypothesis (Kelly & Walter, 1969): when subjected to experimentally induced stress, anxious patients had a mean basal forearm blood flow significantly greater than that of depressed patients. Mountjoy & Roth (1982b) also found a tendency for anxiety states to show increased physiological response, while depressive states were frequently associated with psychological response to stress.

In summary, the differentiation of anxiety states from depressive states in adults has been made on the basis of clinical items, rating scales, prognosis, personality, treatment response, course and outcome, physiological response to stress, and family history data.

TABLE III
Anxious depression position

Study	Subject population	Variable(s) examined	Results
Van Valkenburg, <i>et al.</i> , 1984	1) Primary anxiety (panic attacks) 2) Primary depression 3) 2 mixed groups of anxiety and depressive syndromes	Clinical symptoms Treatment response Course/outcome Family history	In general, differences between the "pure" and "mixed" groups were found on the variables under examination "Mixed" groups showed increased agitation, hypochondriasis, depersonalisation, derealisation, chronic depression Increased proportion of "mixed" group failed to improve when given spectrum of pharmacological and psychosocial intervention "Mixed" groups more likely to have poor psychosocial outcome; more chronic course "Mixed" groups did not differ significantly in family history of depressive or anxiety disorders
Clancy <i>et al.</i> , 1978	Anxious patients with and without secondary depression	Course/outcome	Anxious neurotics with secondary depression have a more severe and chronic illness
Schapiro <i>et al.</i> , 1971	Anxious and depressed inpatients	Prognosis	Anxiety symptoms in depressed patients and depressive symptoms in anxious patients indicated poorer prognosis
Paykel, 1971	Depressed inpatients, out-patients, day patients, emergency patients	Rating scales	Factor analysis delineated a subgroup of depression characterised by anxiety and tension

Anxious-depressive position

In addition to anxiety and depressive disorders, a group of patients has been identified with coexistent anxiety and depression; while seeming to support the unitary position, this group show phenomenological differences from either generalised anxiety disorder or major depressive disorder. Overall *et al.* (1966) factor-analysed the Brief Psychiatric Rating Scale scores of a sample of male inpatients; this divided the sample into three sub-groups, with one (anxious-tense) characterised by anxiety, tension, and depression. Similarly, Paykel (1971) identified by cluster analysis a group suffering from both anxiety and depression.

Downing & Rickels (1974) compared target symptomatology, illness history, and prognosis for a sample of patients with a mixed anxiety-depression diagnosis assigned to treatment with either anti-depressant or anxiolytic drugs. They concluded that

the group with mixed symptomatology demonstrated differences in terms of clinician and patient ratings of anxiety, depression, and treatment response; and that the diagnosis of mixed anxiety depression was not representative of a homogeneous group.

Van Valkenburg *et al.* (1984) examined the validity of the anxious-depressive syndrome in a sample of 114 patients who met the diagnostic criteria for anxiety neurosis (DSM-III panic disorder) and/or for depression. This differed from the study by Downing & Rickels (1974) in the use of panic attack as a clinical marker for anxiety syndromes and in the use of operationally defined criteria to delineate their groups, thereby greatly enhancing the homogeneity of the diagnostic groupings. The assessment of the groups was on several variables—clinical symptoms, course and outcome of the illness, and family history. In terms of clinical symptoms, the groups in which there was a mixture of anxiety and depression showed a marked similarity, with observed

differences attributed to severity; both groups had a similar family history of psychiatric illness.¹ In contrast, significant differences between the groups of anxious depressives and those with primary anxiety or primary depressive disorder were reported for treatment response, psycho-social adjustment, and prognosis. The finding that functioning is most impaired in those patients in whom anxiety and depression co-exist corroborated earlier research. Clancy *et al* compared anxious neurotics with and without secondary depression: their anxious-neurotic group who subsequently developed secondary depression had a more serious course and outcome, leading to the conclusion that anxious patients who develop a secondary depression have a more severe and chronic anxiety illness, which predisposes them to depression.

In summary, research indicates that when the two syndromes coexist, there is increased chronicity of the illness, reduced response to conventional therapies, and a poorer prognosis. Anxiety and depression in combination appear to represent both a quantitatively and qualitatively separate syndrome, unlike either anxiety disorder or affective disorder in many important respects.

Critical analysis of previous research

There are many sources of discrepancy in the studies examining the relationship between anxiety and depression. These range in scope and severity from the relatively minor inconsistencies in scoring procedures to major issues concerning semantic, methodological and interpretative differences. These discrepancies could, directly or indirectly, give rise to the observed results, thereby helping to explain some of the apparently contradictory findings.

Semantic inconsistencies: The largest stumbling block to the study of the relationship of anxiety and depression has been one of semantics, and relates to the multiplicity of the concepts of both anxiety and depression. Semantic inconsistencies between the various studies arise from two sources—discrepant use of the terms by investigators, and a lack of differentiation between the terms by patients in reporting on their symptoms. The introduction of operationally defined criteria for delineating the disorders has alleviated some of these difficulties, but confusion over the use of the two terms is still common among both clinicians and patients. 'Psychotic/endogenous' and 'neurotic/reactive' depressions are vague and ill-defined: they can either represent the two ends of a continuum, thus differing quantitatively, or two separate entities, differing qualitatively. In addition, the complaint of anxiety

can denote a normal response variance, a trait, or a state, and has been used interchangeably with depression.

Methodological differences: The next largest source of variance between the studies has been methodological differences in sample populations, sources of data, range of symptomatology assessed, and level of analysis.

Sample selection: The procedures for selecting subjects have varied from preselection by clinical diagnosis to recruitment through radio advertising (Dealy *et al*, 1981) so that patients one investigator may exclude are the subjects for another study. Different exclusion and inclusion criteria have resulted in varied demographic as well as clinical patient characteristics of the samples—differences in terms of degree of psychopathology, sex, and socio-economic level. Studies included only outpatients, only inpatients, or a combination of both. Based on the assumption that inpatients are 'sicker' than outpatients or day patients, a study composed only of outpatients may not find the same association between anxiety and depression as one composed of inpatients. There is a similar lack of generalisability for samples homogeneous as to sex: three studies (Prusoff & Klerman, 1974; Mendels *et al*, 1972; Zitrin *et al*, 1980) included only female subjects, while in three others (Downing & Rickels, 1974; Derogatis *et al*, 1972a; Dealy *et al*, 1981), there were more women than men. Since data indicate that females report symptoms of anxiety and depression in greater numbers than males, results obtained in all female samples can only apply to other samples of females.

Samples have also differed in socio-economic level: both Prusoff & Klerman (1974) and Derogatis *et al* (1972a) excluded patients from the lowest social class because earlier research had indicated that the factor structure of a lower-status anxious group is qualitatively distinct from that of the other social classes. To what extent the results from research which has included lower class subjects are distorted is not presently known.

There are also inconsistencies in the homogeneity of the samples: sometimes heterogeneous groups have been intentionally selected (Mendels *et al*, 1972), while other samples have varied in their degree of homogeneity from the absence of diagnostic groupings (Johnstone *et al*, 1980) to the use of a clinical marker for delineating groups (Van Valkenburg *et al*, 1984). The success of the various selection procedures in achieving homogeneity

depends to a great extent on the degree to which empirically defined criteria are used in the selection and diagnosis of the clinical groups. However, even this is not enough to assure comparable samples, since the claim that diagnoses were made according to DSM-III criteria is not sufficient to establish that these criteria were used as intended or as others have used them (Helzer & Coryell, 1983). In addition, many of the clinical diagnoses of the present studies were made prior to the advent of operationally defined diagnostic criteria.

Procedural differences: Procedural differences between studies also help to account for some inconsistent results. Differences in focus, in the nature and source of the data, and how the data are analysed vary across studies:

- (i) Focus: The focus of studies on the relationship of anxiety and depression has ranged from discriminating anxiety from depression (Newcastle studies) to examining the etiological role of anxiety in depression (Clancy *et al*, 1978), to an assessment of clinical, family, and outcome variables in anxious-depressive states (Van Valkenburg, 1984).
- (ii) Sources of data: Studies differ in the source of their data and in the degree of reliability inherent in those sources; in those reviewed, the source of information includes clinical interviews, observer-rated scales, patient-rated scales, physiological measures, and retrospective recall. Many have used clinical interviews for diagnosis, for obtaining clinical histories, or for rating the presence and severity of psychopathology. The interviews have varied in their degree of structure, whether the interviewer is the same person or a different one from the clinician who made the original diagnosis, whether the interviewer is 'blind' to the diagnosis, and to the number of raters present: all of these factors have a bearing on the reliability of the data obtained.

Observer-rated scales have generally been found to be a more reliable source of information about severity of psychopathology than self-reports of behaviour, which are subject to distortion either through the respondent 'faking bad' in a bid for attention or 'faking good' to appear mentally healthy. Denial of symptoms, especially in the acute phase of illness, has been cited as one of the major reasons for the low concordance between clinician and patient ratings (Prusoff *et al*, 1972). There is a similarly low concordance between patient and clinician ratings for bipolar depression (Donnelly *et al*, 1980). It becomes clear that the reliability of self-reports depends both on the type of depression

(unipolar/bipolar) and on the stage in the illness at which the patient is assessed. Self-reports of behaviour also presuppose that the patient has the ability to analyse and make accurate judgement on his internal states, but this varies considerably from patient to patient, depending on such factors as intellect, verbal ability, and severity of the disorder. Clinician-rated scales give a more accurate picture of the disorder for several reasons. Firstly, they have the advantage of a far greater variety of behaviour on which to make judgements, e.g. facial expression, degree of tearfulness, amount of agitation or retardation, and body language. Secondly, the clinician has considerably more knowledge of the whole range of psychopathology, and thus a larger perspective on which to base his judgement.

The reliability of data in studies which have used retrospective recall of childhood events (Roth *et al*, 1972) or required the patient to reconstruct the course of an illness (Shapira *et al*, 1972) is questionable, as these reports are subject to errors of memory. Without substantiating evidence, they are generally an unsatisfactory source of information.

Range of psychopathology assessed: The selection of clinical items is also variable across studies, as is the choice of rating scales. Clinical items and rating scales which assess a wide range of symptoms have greater chance of finding significant differences between groups than fewer items which assess a narrower range of symptoms. Derogatis *et al* (1972a) cite the small number of items relating to anxiety on the Symptom Checklist as a plausible explanation for the failure to find a dimensional construct of anxiety in their sample of depressed neurotics.

Timing of disorder in relation to course of the disorder: Studies have also varied in the timing of the study: in some, the focus is on the current mental state while in others, it involves a follow-up assessment. Since the symptoms of anxiety and depression vary during the course of the disorder, different results could be a function of the length of time after the initial illness until the follow-up assessment.

Statistical analysis: There are also wide variations in the use of statistical techniques to analyse the data; these vary from univariate frequency counts, t-tests, and analysis of variance, to the sophisticated multivariate techniques of multiple regression, factor analysis, and discriminant function analysis. Many of the statistical tests have certain assumptions such as normality of the distribution and homogeneity of variance, which must be met if the results are to be

valid. Frequently, insufficient information is provided to assess whether these important assumptions have been met or not.

One misuse of statistical methods in several of the studies is evaluation of the differences in means between groups by multiple t-tests. When these are computed on the same data, the range of scores increases at a faster rate than the standard deviation, so that non-significant results will be erroneously reported as significant.

Level of interpretation: The level of interpretation is also variable between studies, with some examining the relationship of anxiety and depression at the symptom level (Dealy *et al*; Clancy *et al*), while others examine differences at the level of factors (Newcastle studies; Prusoff & Klerman; Mendels *et al*; Derogatis *et al*, 1972a). Discussion at one interpretational level is not necessarily comparable with discussion at a different level.

There has also been some selectivity in what individuals choose to interpret as being significant: in the factor analysis of rating scales by Mountjoy & Roth (1982a), their first component accounted for 44% of the variance; all the scales loaded substantially on this component, and it was described as a general dimension of severity of psychiatric disturbance. This is basically the same result that Mendels *et al* obtained when they factor-analysed self-rated scales, but whereas Mendels *et al* chose to interpret this as evidence for the fact that anxiety and depression are variants of the same disorder, Roth & Mountjoy go on to interpret the second component, and use it in a discriminant function analysis to demonstrate that anxiety and depression are separate entities. Thus, we have similar results interpreted very differently.

These semantic, methodological, and interpretational disparities between studies evaluating the relationship of anxiety and depression make it difficult to compare the results of one with another. Indeed, inconsistencies inherent in the various research designs may explain why one investigator gets one set of results while another, doing a similar procedure, gets the opposite set of results. The need for replication of the crucial studies is obvious, if the ambiguity in the present findings is to be resolved.

Discussion

Anxiety and depression are classified as separate disorders—clinically through DMS-III and statistically through discriminant function analysis. However, certain issues such as the presence of mixed anxiety/depression patients, the relatively high miss-classification rate (which casts some doubt as to the feasibility of being able to discriminate between anxiety and depression in clinical practice), and contradictory research findings suggest that there is no unequivocal solution to the problem of how anxiety and depression are related. Several issues are in need of clarification and resolution, including replication of research findings, cross-validation of discriminant function coefficients, assessments of biochemical markers as a means of separating the two groups, relationships within the sub-categories of each major disorder (e.g. endogenous depression and generalised anxiety disorder), and the relationship between anxiety and depression in children. Based on the assumption that childhood and adult psychopathology are continuous, an examination of the nature of this in children might help to clarify the relationship in adults.

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A Follow-Up and Family Study of Briquet's Syndrome

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For many years we and other colleagues in the Department of Psychiatry at Washington University School of Medicine in St Louis, Missouri have been concerned with the need to develop widely accepted, valid diagnostic criteria for psychiatric disorders. The aim has been to identify patient groups as homogeneous as possible. Diagnostic criteria, such as those used in this study, were therefore developed with the goal of maximising specificity (i.e. reducing false positives) while keeping sensitivity as high as possible (i.e. keeping false negatives low).

This paper deals with some issues concerning Briquet's syndrome (hysteria, somatisation disorder): in particular, the validity of specific diagnostic criteria (measured by consistency of diagnosis over time) and the pattern of illness seen in first-degree relatives of index cases. This report is part of an extensive investigation of patients attending the Washington University Psychiatry Clinic in St Louis, carried out under 'blind' conditions. Different individuals studied the patients at index and at follow-up and examined the patients' first-degree relatives. The follow-up and family studies were performed without any knowledge of the findings concerning any individual in the other component, or of the findings concerning any individual at index.

The overall investigation was designed to evaluate and improve diagnostic criteria for a variety of psychiatric disorders by studying the longitudinal course and by identifying patterns of illness in close relatives of index cases. The study resulted from an initial intention to repeat and refine a series of previous investigations concerning Briquet's syndrome by selecting a new population of patients. It quickly became apparent that in order to obtain a group of patients with Briquet's syndrome it would be necessary to screen many other patients, and it was decided that the study should be enlarged to include patients with a wide variety of psychiatric disorders.

Method

Selection of subjects

The selection of the 500 index patients has been described elsewhere. The selection process was designed to identify a representative cross-section of patients attending the Washington University Psychiatry Clinic. The patients were examined initially during a period of 28 months, from July 1, 1967 to November 1, 1969. Data from this phase of the study have already been presented (Guze, Clayton & Woodruff, 1975; Guze, Woodruff & Clayton, 1971*a,b,c*, 1972, 1974; Liskow *et al.* 1977; Woodruff, Clayton, Cloninger & Guze, 1976; Woodruff, Clayton & Guze, 1969, 1971, 1972, 1975, 1976; Woodruff, Guze & Clayton 1971*a,b*, 1972*a,b*, 1973, 1974; Woodruff, Guze, Clayton & Carr, 1973).

Between 1969 and 1973, 1249 interviews with first-degree relatives of the index subjects were carried out. We had intended to interview an average of three first-degree relatives for each index case. It became apparent, however, that progressively more time and effort were required to locate and interview relatives as the number of completed relative interviews grew. It was decided to stop before the full 1500 interviews had been completed, because we concluded that it would require another two years to interview the additional 250 relatives.

The follow-up interviews were carried out from 1973 to 1979. This resulted in a 6-12 year follow-up for individual patients. Of the 500 index patients, 6 were unavailable for follow-up because they could not be located, 41 were known to be dead, 31 refused to be interviewed or were repeatedly uncooperative, and 422 were interviewed.

Research interviews

The same standardised, structured interview was used in all three phases of the study: index, family, and follow-up (Guze, 1976). In each phase the interviews were carried out by different individuals, who were blind to information concerning individual patients obtained in the other phases.

Diagnoses

The general approach to psychiatric diagnosis has been described previously (Guze, 1976). The specific criteria used in this study (Martin *et al.* 1985) were very similar to those of Feighner *et al.* (1972).

The interviews with the relatives were conducted, after appropriate training, by psychiatry residents and fourth

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year medical students; the latter were selected from those with a strong interest in psychiatry who had already completed their psychiatry clerkships. The protocols were reviewed by Robert A. Woodruff and two of the authors. The protocols from the follow-up interviews (conducted by CRC and RLM) were reviewed by PJC and SBG. Following each protocol review, diagnoses were made according to the specified criteria, and appropriate forms were completed for coding and keypunching.

In view of the large number of patients (500), relatives (1249), and total interviews (nearly 2200), and the long interval involved in the overall study (14 years, as protocol review continued until 1981), it is highly unlikely that the protocol reviewers were any less blind than were the individual interviewers.

At follow-up a second review of each case was carried out a few weeks or months after the initial protocol review, in order to consider additional information obtained from physicians, clinics, and hospitals visited by the index patients, and from other informants in those cases where a follow-up interview had not been feasible. A second follow-up diagnosis was then made for each case. This was based on all available information except for the index interview and the relative interviews. These second follow-up diagnoses were used for the analyses in this paper. Comparisons between the follow-up interview information and that obtained from records and other informants will be presented in future publications for the entire range of psychiatric diagnoses.

Previous work

The present investigation of Briquet's syndrome follows a number of earlier studies (Arkonac & Guze, 1963; Bibb &

Guze, 1972; Cloninger & Guze, 1970*a,b*, 1975; Cloninger *et al*, 1975, in press; Farley *et al*, 1968; Gattfield & Guze, 1962; Guze, 1963, 1964, 1966, 1967, 1970*a,b*, 1975, 1983; Guze & Purley, 1963; Guze, Woodruff & Clayton, 1971*b*, 1972; Liskow *et al*, 1977; Martin *et al*, 1979; Perley & Guze, 1962; Woerner & Guze, 1968; Woodruff, Clayton & Guze, 1969, 1971). This previous work suggests that the diagnostic criteria used selected patients (nearly all of whom were women) whose clinical course and familial illness patterns provided strong evidence for the validity of the diagnosis. These patients show a consistent clinical picture over time, with very few turning out to have other disorders that might, in retrospect, account for the original clinical picture. (This is in striking contrast to the findings when patients are selected primarily on the basis of conversion symptoms). The same disorder occurs with considerably increased frequency among first-degree female relatives, while antisocial personality is encountered at increased frequency among first-degree male relatives and spouses. Furthermore, Briquet's syndrome and antisocial personality are encountered together in the same individual much more frequently than would be expected by chance. This personal and familial aggregation of the two disorders has led to the hypothesis that Briquet's syndrome and antisocial personality result from similar combinations of aetiological factors and that the clinical manifestations of these underlying aetiological factors depend largely on the sex of the individual: women are more likely to show the pattern of Briquet's syndrome, while men are more likely to show the pattern of antisocial personality.

Results

Table I shows the follow-up diagnoses given to those

TABLE I
Follow-up diagnoses of Briquet's syndrome according to index diagnoses

<i>Follow-up diagnoses</i>	<i>Index diagnoses</i>	
	<i>Briquet's syndrome (Definite or probable) (n = 36)</i>	<i>Undiagnosed ? Briquet's syndrome (n = 26)</i>
Briquet's syndrome (definite or probable)	23	15
Undiagnosed: ? Briquet's syndrome	5	6
Other diagnoses		
Primary affective disorder	3 ¹	1
Primary anxiety neurosis	2 ²	2 ⁴
Antisocial personality	1 ³	
Primary alcoholism	1	
Undiagnosed	1	2
Total	22	5

1. Unipolar depression 2 (secondary anxiety neurosis 1 and mental retardation 1), bipolar disorder 1

2. With secondary depression in both and with phobic neurosis in 1

3. With secondary alcoholism plus other undiagnosed features

4. With secondary depression in 1

TABLE II
Index diagnoses according to follow-up diagnoses of Briquet's syndrome

Index diagnoses	Follow-up diagnoses	
	Briquet's syndrome (Definite or probable) (<i>n</i> = 71)	Undiagnosed ? Briquet's syndrome (<i>n</i> = 42)
Briquet's syndrome (definite or probable)	23	5
Undiagnosed: ? Briquet's syndrome	15	6
Other diagnoses		
Primary affective disorder	9 ¹	15 ¹
Primary anxiety neurosis	11 ²	7 ⁴
Schizophrenia	1	
Primary alcoholism	3 ³	
Antisocial personality	1	1
Epilepsy	1	
Mental retardation	1	
Undiagnosed	6	8
Total	33	31

1 All unipolar depression

2. With secondary alcoholism 1, with secondary depression 6, with ? mental retardation or ? brain syndrome 3

3. With associated drug dependence 1, with secondary depression and secondary anxiety neurosis 1

4. With secondary depression 4; with secondary alcoholism 1, with associated epilepsy 1

patients with index diagnoses of Briquet's syndrome; Table II shows the index diagnoses that had been given to those patients with follow-up diagnoses of Briquet's syndrome. At both index and follow-up, a number of patients did not meet the criteria for either definite or probable Briquet's syndrome; nor did they meet the criteria for any other diagnosis. Since their symptoms resemble Briquet's syndrome more than anything else, they were designated as 'undiagnosed, ? Briquet's syndrome'.

About two-thirds of the patients who received the diagnosis of definite or probable Briquet's syndrome at index received the same diagnosis at follow-up (Table I). Another 14% of such index cases were considered to be undiagnosed, ? Briquet's syndrome at follow-up, so that nearly four-fifths of index Briquet's syndrome patients continued at follow-up to present with either the same diagnostic picture or one resembling it enough for them to be suspected of suffering from the same condition. On the other hand, over 20% of these index patients received other diagnoses at follow-up. A similar pattern was seen in those patients receiving a diagnosis of undiagnosed, ? Briquet's syndrome at index.

The data indicate that more patients received diagnoses of Briquet's syndrome or of undiagnosed, ? Briquet's syndrome at follow-up than at index, and that the retrospective consistency was much less than the prospective consistency (Table II). Only one-third of patients with follow-up diagnoses of Briquet's syndrome had received the same diagnosis at index, and only 12% of patients considered undiagnosed, ? Briquet's syndrome at follow-

up had received the diagnosis of Briquet's syndrome at index. In total, only about half of the patients with follow-up diagnoses of Briquet's syndrome had had the diagnosis made or suspected at index; the comparable figure for the follow-up cases considered to be undiagnosed, ? Briquet's syndrome was about one-quarter. Whether prospectively or retrospectively, primary affective disorder and primary anxiety neurosis were the most frequent diagnoses made in the inconsistent cases.

Tables III and IV present the diagnoses in first-degree relatives according to the proband diagnoses. In Table III the probands are characterised in terms of the follow-up diagnoses; in Table IV, in terms of either index or follow-up diagnoses. The latter analysis was carried out because of the inconsistency observed in the proband diagnoses over time. The results are very similar. They indicate that the diagnostic criteria for Briquet's syndrome select a group of patients whose first-degree female relatives show a severalfold increase in the frequency of the same syndrome, as well as in the frequency of antisocial personality, and whose first-degree male relatives also show a significantly increased frequency of antisocial personality. It should be noted here that the probands who had never received any diagnosis suggesting Briquet's syndrome included many with antisocial personality. It would be reasonable to assume that their first-degree relatives include many with the same disorder, so that the frequency of this condition in the overall sample of relatives is probably especially elevated, tending therefore to obscure the full significance of the elevated prevalence of antisocial personality among

TABLE III
Disorders in first-degree relatives classified by follow-up proband diagnosis of Briquet's syndrome

Diagnosis at index ¹	Probands Diagnosis of follow-up	No.	No.	Female relatives No. with Briquet's syndrome	No. with antisocial personality	Male relatives No.	No. with antisocial personality
+	+	23	33	2	3	25	6
?	+	15	27	1	3	26	6
0	+	33	45	4	3	45	6
Total	+	71	105	7*	9**	96	18***
+	?	5	4	0	1	4	1
?	?	6	8	0	0	3	0
0	?	31	47	2	1	30	3
Total	?	42	59	2	2	37	4
+	0	8	9	2	0	3	1
?	0	5	9	0	0	3	1
0	0	374	514	11	14	414	42
Total	0	387	532	13*	14**	420	44***
Total sample		500	696	22	25	553	66

1. + = definite or probable Briquet's syndrome, ? = undiagnosed, ? Briquet's syndrome; 0 = other diagnoses

*Difference between + and 0 significant ($P < 0.05$)

**Difference between + and 0 significant ($P < 0.01$)

***Difference between + and 0 significant ($P < 0.05$)

the relatives of probands with Briquet's syndrome. Our strategy for breaking the blind diagnostic codes prevents us from opening up the data concerning the relatives of probands with antisocial personality until we are ready to study the probands directly.

No cases of Briquet's syndrome and only three cases of undiagnosed, ? Briquet's syndrome were found in male relatives. No increased frequency of either Briquet's syndrome or antisocial personality was found among the relatives of probands considered undiagnosed, ? Briquet's syndrome. No familial association was seen between Briquet's syndrome in the probands and either schizophrenia, primary affective disorder, alcoholism, or anxiety neurosis in the relatives

Discussion

Several features of this study deserve emphasis. The overall research design emphasised 'blindness' to a degree seldom achieved in such clinical investigations. The index subjects represented a broad cross-section of psychopathology, so that a wide range of diagnoses had to be considered in evaluating the follow-up data and the study of relatives, thus

adding to the diagnostic difficulty. All of the interviews were carried out by clinically trained individuals. The same interview protocol and explicit diagnostic criteria were used in all phases of the investigation, and these are related conceptually and historically to widely used instruments and diagnostic systems (Feighner *et al*, 1972; Guze, 1976; Robins *et al*, 1981; Stoltzman *et al*, 1981). The results of the overall project attest to the value of careful follow-up and family studies in improving diagnostic validity (Robins & Guze, 1970).

Overall, the results confirm previous work concerning follow-up and familial illness patterns associated with Briquet's syndrome. Briquet's syndrome runs in families. There is no familial association with schizophrenia, primary affective disorder, primary anxiety neurosis, or alcoholism in the absence of antisocial personality. The association with antisocial personality is especially noteworthy, because the diagnostic criteria in the two conditions are independent and without overlap. Furthermore, as noted previously (Cloninger & Guze, 1970a,b; Guze *et al*, 1971a,b; Cloninger *et al*, in press, Guze,

1976), the repeated observation that Briquet's syndrome is seen predominantly in women while antisocial personality is encountered predominantly in men emphasises the importance of the association confirmed in this report, indicating that the two disorders must share important aetiological factors.

The diagnostic inconsistency observed is compatible with our clinical experience in treating patients with Briquet's syndrome. Many patients, especially early in the course of our contact with them, prove to be inconsistent historians. This was demonstrated in a previous follow-up study in which a major source of diagnostic inconsistency was attributable to inconsistent reporting of symptoms (Martin *et al*, 1979). It is often not until we have followed individual patients for some time that the full clinical picture becomes evident. We have learned that this pattern is itself a characteristic of the disorder. It accounts in part for the confusing clinical course, the overtreatment, and the therapeutic frustrations encountered in the care of these patients. Experience with such patients underscores our conviction that

it is very important to talk to relatives and friends and to seek information from other physicians, hospitals, and clinics whenever faced by any difficult or puzzling case. In fact, in the previous follow-up study diagnostic consistency was appreciably improved when independent medical records were considered as well as interview data.

Our data suggest that once a diagnosis of Briquet's syndrome is made, however, a reasonable diagnostic consistency may be expected (about 80% in this sample), but that other psychiatric diagnoses will be made or seriously considered in patients who ultimately meet the criteria for Briquet's syndrome, but who fail to meet them initially (about half the cases in this sample).

The failure to find any characteristic familial illness pattern associated with probands considered undiagnosed, ? Briquet's syndrome but who had never received a definite or probable diagnosis requires comment in view of the fact that an overlap over time between this diagnosis and the diagnosis of Briquet's syndrome was seen. The data suggest

TABLE IV
Disorders in first-degree relatives classified by follow-up index or follow-up proband diagnosis of Briquet's syndrome

Diagnosis at index ¹	Probands Diagnosis of follow-up	No.	No.	Female relatives		Male relatives	
				No. with Briquet's syndrome	No. with antisocial personality	No.	No. with antisocial personality
+	+	23	33	2	3	25	6
?	+	15	27	1	3	26	6
0	+	33	45	4	3	45	6
+	?	5	4	0	1	4	1
+	0	8	9	2	0	3	1
Total ever +		84	118	9*	10**	103	20***
?	?	6	8	0	0	3	0
0	?	31	47	2	1	30	3
?	0	5	9	0	0	3	1
Total ever ?, never +		42	64	2	1	36	4
0	0	374	514	11*	14	414	42***
Total sample		500	696	22	25**	553	66

1. + = definite or probable Briquet's syndrome; ? = undiagnosed, ? Briquet's syndrome; 0 = other diagnoses

*Difference between + and 0 significant ($P < 0.01$)

**Difference between + and 0 significant ($P < 0.01$)

***Difference between + and 0 significant ($P < 0.05$)



that the proper explanation is simply one of relative probability. Individuals considered undiagnosed, ? Briquet's syndrome at any one time include some who are really cases of Briquet's syndrome, which will become evident on another occasion, while others are suffering from other disorders which will also be evident on other occasions, but which sometimes resemble Briquet's syndrome. In any particular sample, the relative proportions of the two groups will determine whether or not a familial association with Briquet's syndrome will be found. In this study, after a 6–12 year follow-up, the failure to present with the full Briquet's syndrome indicates that they were false positive cases. Our findings confirm our clinical experience that careful follow-up of uncertain cases of Briquet's syndrome will usually clarify the diagnosis. If, however, the clinical picture remains uncertain, our family data indicate that such patients should not be classified with Briquet's syndrome.

Summary

The study began with the systematic clinical evaluation of a cross-section of 500 of the clinic's patients. This was followed by a 'blind' follow-up of the index subjects and a 'blind' study of first-degree relatives. The present report deals with the diagnosis of Briquet's syndrome (hysteria, somatisation disorder) at index, at follow-up, and among first-degree relatives. The data indicate that the criteria used for the diagnosis of Briquet's syndrome select patients who show a high degree of diagnostic consistency over many years, although not all patients who meet these criteria at follow-up receive the diagnosis of Briquet's syndrome initially. Most importantly, the diagnostic criteria select cases associated with a strong familial increase in the risk of Briquet's syndrome and in the risk of antisocial personality. Both are increased among first-degree female relatives, while only antisocial personality is increased among first-degree male relatives.

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The Importance of Hysteria

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Hysteria has been a topic of interest throughout the history of medicine; those who have been concerned with it include Galen, Paré, Sydenham, Charcot and

Freud. Anyone who chooses to proclaim its importance, therefore, might be asked to provide some reason for gilding the lily. Controversies have

always attended the subject, and different disciplines still disagree over it. The diagnosis, which occurs in the International Classification of Diseases (ICD-9, 1978) has been deprecated on both sides of the Atlantic (Slater, 1965; DSM-III, 1980) and also advocated with varying degrees of fervour (Walshe, 1965; Lewis, 1975; Merskey, 1979). It is a subject of historical study (Veith, 1965; Walker, 1981; Shorter, 1984); there have been at least nine monographs on it since 1977 (Horowitz, 1977; Krohn, 1978; Jakubik, 1979; Merskey, 1979; Roy, 1982; Riley & Roy, 1982; Colligan *et al*, 1982; Weintraub, 1983; Ford, 1983), and there is a steady flow of paper on the topic of hysteria or its major subdivisions (eg, hysterical personality, conversion symptoms) or pseudonyms and partial pseudonyms (eg, somatisation disorders, borderline personality, and operant pain).

Furthermore, the whole field of chronic pain is a prominent theme in which at least some aspects of hysteria require consideration (Merskey, 1980). Nevertheless, it is probably true that the volume of publications on hysteria is less than on other topics, in relation to its supposed importance.

For convenience of definition, I shall follow the ICD-9 classification of hysterical neurosis (300.1) and hysterical personality (300.5). For the subtypes of hysteria, I have suggested the following (Merskey, 1979):

- (a) hysteria with one or two symptoms, usually motor or dissociative (as in amnesia), sometimes pain
- (b) polysymptomatic hysteria, especially hypochondriasis and Briquet's syndrome as described by Guze and his colleagues (the St Louis group) and now elaborated in DSM-III under the heading of somatisation disorder
- (c) hysterical elaboration of organic complaints
- (d) symptoms of self-induced illness or self-damage in abnormal personalities ranging from anorexia nervosa to hospital addiction
- (e) psychotic or pseudopsychotic disorders (Ganser's syndrome, hysterical psychosis)
- (f) hysterical personality
- (g) culturally sanctioned endemic or epidemic hysteria.

Since different aspects of this list will occur in many different situations, we have a hint that the theme might be an important one both in medicine and in society. I will start by considering the epidemiology of somatic complaints not due to physical illness and probably not due to psychophysiological causes.

Frequency

If we look only for the florid symptom picture described by the St Louis group, we find that 1–2% of parous women (Farley *et al*, 1968) or women under investigation in a medical ward (Woodruff, 1968) present this pattern of illness. There are similar findings in other surveys (Majerus *et al*, 1960; Murphy *et al*, 1962). About 10% of psychiatric in-patients in university hospitals (Bibb & Guze, 1972) and 5–11% of psychiatric out-patients (Farley *et al*, 1968) are similarly affected. Folks *et al* (1984) found, in 1000 consecutive psychiatric consultations in a general hospital, that the prevalence of conversion disorder was about 5%; one third of these cases had somatisation disorder. Katon *et al* (1984) have reviewed the prevalence of somatisation in primary care and have shown that it is at least relevant, in the majority of general practice cases, whether the patients are seeking treatment for minor somatic complaints because of major psychological problems, or whether it is of psychophysiological origin rather than the result of a conversion process. Many of these symptoms may also be related to depression, but it is impossible to read their survey, or to consider this field in any depth, without reaching the conclusion that an understanding of psychological mechanisms in the production of bodily complaints is fundamental to an understanding of medical practice as a whole.

Chronic pain has a frequency of 11% in a fairly typical population (Crook *et al*, 1984). When it is a subject for treatment in pain clinics there is always a group in whom anxiety and hysterical mechanisms are believed to play an important role (Merskey, 1980). Ford (1983) provides evidence from various sources to show that physical complaints on the basis of psychological illness (somatisation) affect 50–60% of medical clinic patients; 27% of patients with symptoms had no evidence of disease (Garfield *et al*, 1976). Spear (1967) found that 66% of psychiatric in-patients and day patients had pain. If the complaints are hysterical, even in a minority of these patients, this is a matter for concern in clinical practice.

Diagnostic problems

Hysteria keeps recurring as an issue in difficult diagnostic cases. Whenever we are at the margin of our ability to decide on a diagnosis, hysteria is a diagnostic possibility. If a somatic symptom has a psychological cause, and if benign physical methods of investigation are not enough to rule out organic disease, the patient may be heavily and even dangerously over-investigated. Alternatively, a lack of

adequate technology and a proper unwillingness to undertake hazardous investigations for obscure symptoms may encourage a false attribution of psychological symptoms to a physical illness.

I have emphasised the frequency of somatic symptoms of psychological origin. At the same time, we have to recognise the existence of a group of symptoms for which advances in clinical knowledge have enabled an organic diagnosis where formerly the label 'hysteria' was applied; if such cases are not correctly diagnosed the psychiatrist will be expected to cure a physical disorder with antidepressant drugs or by psychotherapy.

I should like to offer some examples of this dilemma. Firstly, there is a form of paroxysmal hemicrania which is now a well-defined syndrome (Sjaastad & Dale, 1974), but which was earlier diagnosed as headache of psychological origin. Secondly, the thoracic outlet syndrome, particularly if it is relatively mild and accompanied only by modest changes, has often been called 'neurotic'; recently I have seen six such patients whose symptoms were precipitated by a flexion-extension ('whiplash') injury of the cervical region, in whom physiotherapy had failed to provide relief and in whom first rib resection was a satisfactory procedure.

Globus hystericus provides a third example. Sydenham (1697), following earlier writers, spoke of "suffocation of the Womb in which the Belly and Entrails rise upwards towards the Throat". Purcell spoke of the symptom of difficulty in swallowing as if "a hard ball pressed against the outside of their throats; sometimes as if it were a Stick thrust down their Throats...". Traditionally, the symptom was regarded as hysterical. However, it is plausible that anxious patients may swallow air or have trouble with anorexic sensations and have difficulty in swallowing. A disorder of the oesophagus, thought to be a spastic phenomenon, was in fact demonstrated by Jacobson (1924) in an early radiological investigation. Malcolmson (1966, 1968) showed, in a series of 307 patients, that only 21% were completely normal on physical and radiological examination; 30% had a physical lesion (eg, cervical spine osteophytes, overactive cricopharyngeus, hyperplastic tonsils, goitre, postcricoid web or enlarged lymph nodes) which explained the symptom, and 49% had a gastrointestinal disturbance below the pharynx, for example hiatus hernia or duodenal ulcer. More evidence in the same direction has been produced by Delahunty & Ardran (1970), Hunt *et al* (1970), Watson & Sullivan (1974), and Lehtinen & Puhakka (1976). There may still be some patients with globus, now best termed globus pharyngis (Malcolmson,

1966, 1968), whose symptoms are psychological in origin either on the basis of anxiety or a conversion mechanism. However, most cases have a physical basis.

Facial dyskinesias and torticollis provide a fourth example. The diagnosis of psychological disorder has to a considerable extent been superseded by an organic diagnosis. I first learned to think of these as more likely to be of organic rather than psychological origin through the influence of my late colleague Dr R. T. C. Pratt. He observed that neurologists seemed to have more difficulty in making up their minds about organic or psychological causes in relation to torticollis and abnormal movements than on any other topic, and also that the symptoms tended not to respond so well to the usual treatments for anxiety or hysteria compared with other more recognisable somatic anxiety or hysterical symptoms. On one occasion, in 1969, with the knowledge of all participants we showed a patient with abnormal facial movements to six neurologists; three said her symptoms were psychological, and three said that they were physical in origin. All six gave impressive arguments to back up their opinions, but the one whose views seemed to me to be most cogent was Dr M. J. McArdle, who observed a retrocollis and abnormal movements of the soft palate. Since that time neurologists have more readily diagnosed facial dyskinesias as organic, for two reasons: firstly, they may be provoked in Parkinsonism by levodopa, and secondly, phenothiazine drugs used in the treatment of schizophrenia may produce such movements.

A fifth condition which I have seen diagnosed as hysterical, but know to be organic, is that of painful legs and moving toes (Spillane *et al*, 1971). Nathan (1978) has now shown that this is a characteristic syndrome with a specific organic basis in damage of either the dorsal nerve roots or the spinal cord.

The final example is the whiplash syndrome. It is well established that people who undergo rear-end collisions in motor vehicles typically show a pattern of development of cervical pain, headache and muscle tenderness. Animal experimentation has shown that there are recognisable and distinct soft tissue lesions, some of them severe, like retro-oesophageal haematoma, which can even be demonstrated in accident victims if the patient complains of difficulty in swallowing and an X-ray is taken at the right time (McNab, 1973). Evidence (Merskey, 1984) supports an organic aetiology in most of these cases. Moreover, patients who have had such injuries may not recover merely because litigation issues are settled (Mendelson, 1982; Merskey, 1984). What used to be called hysterical or compensation

neurosis is now better considered to be a symptom based on physical disability. It may carry in its wake some secondary depression and secondary psychological manifestations, but when a condition is poorly understood and the patient is referred from one medical department to another, it is understandable that psychological symptoms should develop; these will include anger, frustration, irritability, and some criticism of the medical profession. In such conditions hysteria should not be diagnosed in the absence of positive grounds for the diagnosis.

Social consequences

The figures quoted above for the frequency of somatic symptoms in medical practice give some indication of the importance of the production of bodily symptoms. The numerous social implications of 'illness behavior' or 'abnormal illness behavior' (Pilowsky & Spence, 1975) cannot be considered here in detail. Some of the many cases of attempted suicide, some disability claims and financial settlements, and perhaps many changes in family relationships will be influenced by hysterical symptoms. In military practice, particularly when the danger is great and the opportunity for a 'respectable' hysterical illness exists, the numbers increase considerably. By April 1918, according to Hurst (1940), there were over 20 000 cases of 'war neurosis', who received invalid pensions. It is not known what proportion of these patients suffered from hysteria and what proportion from anxiety states or depression, but the frequency of hysteria in that era is well attested to by the numerous books and other publications on the topic of 'shell shock' and 'war neurosis' (Merskey, 1979). Since the First World War such cases have been less frequent; during the Second World War the organisation of the Allied military psychiatric services was such that the development of hysterical symptoms was discouraged. However, hysterical manifestations have always been found in men under stress. Likewise, epidemics of hysteria occur, most often in girls' schools (Sirois, 1974).

It has been suggested that one of the important theoretical consequences of the recognition of combat neurosis and hysteria in the First World War was the strength it gave to the argument that, since so many men who often seemed otherwise normal were affected, hysterical symptoms were not necessarily sexual in origin.

Symptoms from thoughts

The single most important aspect of hysteria is its contribution to our understanding of the mind. To

place this in context it is worthwhile considering the 17th century concept of hysteria, about which I have written in more detail elsewhere (Merskey, 1983). Sydenham (1697) included two major ideas under the rubric of hysteria. Firstly, he indicated that emotional stress disturbs the function of the nervous system; even before the time of Galen others had recognised an influence of the mind upon the body (Veith, 1965, and Hunter & McAlpine, 1963, give several illustrations from writers such as Bartholomeus Anglicus, Elyot, Langton and Paré).

Secondly, Sydenham provided a catalogue of bodily changes presumed to result from this disturbance. Some of these are clearly organic: apoplexy with hemiplegia, epilepsy, vomiting bile, and swollen legs, for example; others, such as excessive laughter, tears and labile emotions, reflect general emotional disturbance, or more specifically hysteria. Some, such as diarrhoea, may be psychosomatic in the strict sense of being related to peripheral somatic changes induced by psychological causes. Sydenham did not elucidate these matters. The critical point is that hysteria was considered to be a mixture of symptoms that we would now call organic, psychosomatic or perhaps hysterical; at the time, they were all thought to result from emotional causes. Gradually, the organic symptoms have been clarified (as the examples given earlier indicate), but it was still not really possible for the distinction between a hysterical symptom representing an idea and a physiological symptom presenting an autonomic disturbance to be made before the days of modern physiology and anatomy. Only with Sir Benjamin Brodie (1837) did some of these possibilities begin to emerge, although precursors existed. These were reviewed by Wright (1980), who remarked upon the fact that Descartes explained strange aversions by past experience. For instance, a cat might have jumped into a baby's cradle, thus causing a dislike of cats without the adult retaining any memory of it afterwards. Descartes also discovered that he was attracted to cross-eyed women, and attributed this to his love, as a young child, for a girl who looked after him and who had this defect. It is noteworthy that the recollection and analysis of the memory cured him of that particular fixation. We see here some recognition that thoughts might cause symptoms, although the symptoms which are caused are also psychological ones.

Others saw physical symptoms being transmitted by example. Robert Boyle is cited by Wright as noting that it is common for one hysterical woman, when she observes another in a fit, to be "infected with the like strange discomposure" and Blackmore (1725) wrote: "Terrible ideas, formed only in the

imagination, will affect the brain and the body with painful sensations. . .". These authors recognised the way in which ideas could produce physical symptoms, but until the age of modern neurology and neurophysiology it was probably impossible for a consistent distinction to be established in respect of the topic, except perhaps for epidemic hysterical complaints, which were well reviewed by Hecker (1844).

The new conception really began with Sir John Russell Reynolds, who was Professor of Medicine at University College Hospital. At a meeting of the British Medical Association Reynolds described "three cases of paralysis dependent upon idea". The report was published the same year (Reynolds, 1869) and seems to have been the first systematic discussion of the theme. Certainly Charcot (1889), who said that the idea was long known, gave Reynolds full credit for his comments. Charcot was then inspired to produce symptoms in patients by the implantation of an idea under hypnosis, and this constituted the second step in the recognition of the special characteristics of the hysterical symptom. The third step we owe to Freud (Breuer & Freud, 1893-5) and his explanation of the origin of the symptom in conflict or in repression. This is so well known that I will not dwell upon it; nor upon the fact that it was the foundation of psychoanalysis, although I also take that to be an important contribution of hysteria to mankind.

The significance of hysteria and its interest lie above all in this reflection of the way in which the mind may transform a person's concept of himself or a part of his body. It has become an avenue to the discussion of personality types, a theme for evaluation in social relationships, a key to motives, and a highway to the unconscious. It is difficult to ask more of a single condition.

The demand on the doctor

However, this condition (whether single or multiple) gives us yet another theme to tackle, and one which is personally demanding for all doctors. Hysteria imposes a test on our professional skills. We can, if we wish, dismiss it as counterfeiting, none of our business, and something with which physicians need not be concerned once they have established the true situation. For reasons which deserve longer discussion elsewhere, I think that this would be an inappropriate professional response. The physician should be as committed to understanding and treating patients with hysteria as those with any other psychological or physical disorder. In order to provide that understanding and treatment, he needs an extensive background in traditional organic medicine and, equally, a tolerant and flexible approach based on psychiatric skills. That combination offers a challenge to the personal qualities of every doctor who aims to treat patients.

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Epilepsy and Hysteria

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Charcot's term ‘hysteroepilepsy’ implies brain hysteria. No such direct link between these two mechanisms in common between epilepsy and conditions exists. Nevertheless, there are a number of

indirect associations between epilepsy and hysteria that can lead to confusion in the differential diagnosis and management of the patient with seizures.

Hysterical pseudoseizures are attacks of sudden unconsciousness, usually (but not invariably) associated with dramatic motor manifestations, which simulate epileptic attacks to a varying degree. The term 'pseudoseizure' is not ideal, since it conveys the impression that the event is not really a seizure. The term 'pseudoepileptic seizure' is more appropriate, since it acknowledges the occurrence of seizures and their resemblance to genuine epileptic fits, but avoids the implication of physiological mechanisms in common between epilepsy and hysteria.

The other terms used to describe hysterical fits ('simulated epilepsy' and 'psychogenic seizures') should not be used. 'Simulated epilepsy' implies malingering; this is rarely the case, since the patient with hysterical fits is not aware of the underlying psychological motivation for them. Psychological stress is important in precipitating frank epileptic attacks in some epileptic patients. Indeed, a recent survey of unselected primary care patients suffering from epilepsy reported that 72% of the sample of 300 found emotional upset to be accompanied by an exacerbation in seizure frequency (Dowds *et al.*, 1983). Hence the term 'psychogenic seizure' should be avoided.

Pseudoepileptic seizures are not an infrequent manifestation of hysteria, being the second most common form found in 381 patients with hysteria (Ljungberg, 1967); 20% of this series presented with hysterical fits. More recently, Reed (1975) found a 9% prevalence among patients at the Maudsley Hospital admitted between 1949 and 1964 with a diagnosis of hysteria. Rather more surprising is the paradoxical observation that genuine epilepsy and pseudoepilepsy can coexist in the same patient. A present or past history of epilepsy is found in a significant number of patients with pseudoepileptic seizures referred to hospital for investigation, the prevalence varying from 12% to around 65% (Merskey, 1978a; Fenton, 1982; Lesser, 1985). Doubtless the wide variance reflects the heterogeneous nature of the series of small groups of patients reported in the literature from different centres.

It may seem strange for people with genuine fits to display pseudoepileptic attacks as an hysterical manifestation. One would think that genuine epileptic fits would provide a more than adequate locus for the illness-rewarding behaviour of the patient who responds to stress using hysterical mechanisms. However, the real epileptic attacks of the patient

with pseudoepileptic seizures are often well-controlled by anticonvulsant therapy. Indeed, there is evidence that the development of classical hysterical fits can be facilitated by anticonvulsant drug toxicity (Niedermeyer *et al.*, 1970), but this would only account for pseudoepileptic seizures in a minority of patients with epilepsy. Previous episodes of unconsciousness, either experienced personally or observed within the family or work environment, often provide a model for the choice of pseudoepileptic seizures as the hysterical manifestation. Roy (1980) found a personal history of epilepsy or an experience of working in a medical environment in about one-third of a series of patients with hysteria, the majority of whom had hysterical convulsions. Therefore, if the person with epilepsy is predisposed to hysterical mechanisms as a means of coping with a stressful situation, the previous experience of genuine epileptic attacks will have a major influence in determining the selection of fits as the hysterical manifestation.

Pseudoepileptic seizure patients also display many other features indicative of psychopathology: a family history of psychiatric illness, a past personal history of psychiatric disorder with suicidal attempts, a diagnosis of personality disorder, sexual maladjustment, disturbed interpersonal relationships with parents or heterosexual partner, an unsatisfactory current life situation, and concurrent affective symptoms. They also tend to have significantly higher scores on the General Health Questionnaire, the Wakefield Self Assessment Depression Inventory and the Hamilton Rating Scale for Depression (Roy, 1977; Lesser, 1985).

Epileptic and pseudoepileptic seizures: differential diagnosis

Clinical manifestations

In the differential diagnosis between epileptic and pseudoepileptic seizures, the clinician should always supplement the patient's account of the seizures by obtaining an accurate history from a reliable observer and be thoroughly familiar with the clinical features and the classification of the wide variety of epileptic seizure phenomena and their pathophysiological mechanisms (Commission on Classification, 1981).

Typical of the pseudoepileptic grand mal attack is the marked involvement of truncal musculature, with some opisthotonos (a complete *arc de cercle* is now very rare) and random struggling or thrashing movements of the limbs, trunk or head. Side-to-side throwing or rolling of the head or body is common.

The pseudoconvulsive movements may increase if restraint is imposed, and the patient may struggle to free himself from the restraint and even become combative; this is in marked contrast to the stereotyped tonic-clonic convulsions of the grand mal epileptic fit.

Cyanosis, an invariable feature of grand mal convulsions, is rare in pseudo-grand mal attacks, although it may occur as a result of breath-holding. The pupillary reaction to light and the corneal reflexes are retained during a pseudoepileptic seizure, but may be difficult to elicit due to the patient actively moving about or to upward deviation of the eyes. Indeed, during the pseudoepileptic attack the patient may actively resist attempts to open his eyes. Pressure on the supraorbital notch may cause head withdrawal, indicating that the patient has some perception of pain. Responsiveness to painful stimuli is entirely lost during the tonic-clonic phase of an epileptic grand mal attack. The facial expression during a grand mal seizure is characterised by trismus in the tonic phase and symmetrical twitching during the clonic phase. In the pseudo-grand mal fit, the face reflects either repose or a variety of emotions, including ecstasy.

The level of consciousness may fluctuate during the pseudoconvulsive phase, so that the patient may be able to recall some or all of the events of the fit. However, the presence or absence of diminished awareness during the attack can be difficult to determine; moreover, some epileptic attacks of partial origin can occur in clear consciousness. Nevertheless, the demonstration of retained awareness of the environment while bilateral or whole body convulsive movements are in progress is strongly in favour of the diagnosis of a pseudoepileptic seizure. A marked emotional display either after, or (on rare occasions) during an attack, with the patient in floods of tears or shouting obscenities, is again suggestive of an hysterical fit.

Following a genuine grand mal attack, the plantar responses become extensor for a short time. However, the responses must be elicited carefully, since it has been shown that healthy adults can learn to dorsiflex their toes in response to plantar stimulation of the foot (Lesser, 1985). If the tongue is bitten at all in pseudoepileptic fits the lacerations tend to be minor, involving the tip rather than the side of the tongue, or the lips rather than cheeks. Loss of bladder control and injury by falling during pseudoepileptic attacks may occur, but are uncommon.

The epileptic aura signals the transition of the episodic interictal firing of epileptogenic tissue into the continuous discharge that recruits additional neuronal circuits and provokes the clinical manifes-

tations of the fit as it spreads to involve large areas of brain. Unless the patient has multifocal disease, which occurs only in cases of extensive brain damage, the aura of the epileptic patients, if present at all, will be identical or very similar during different attacks. Neither the presence nor absence of an aura, nor the type of the aura, provides useful differential diagnostic information.

Subjective phenomena herald the onset in a significant minority of pseudoepileptic fits. Specific symptoms include palpitations, malaise, choking, dizziness, paraesthesiae in the face or extremities, pain, olfactory and gustatory hallucinations, and visual illusions and hallucinations (Lesser, 1985). Pain, either in the body, extremities, or head, is rare as an epileptic aura. The only exception is a focal seizure of parietal origin, in which there is usually contralateral paraesthesia with spread of a seizure activity to the motor cortex so that the affected extremity will show clonic convulsive movements. On the other hand, a prodromal complaint of headache is quite common in pseudoepileptic seizures (Finlayson & Lucas, 1979). Auras that contain a great number of somatic or visual symptoms which vary on different occasions suggest pseudoepilepsy.

Epileptic attacks usually begin abruptly. Although many pseudoepileptic fits do so also, a consistently gradual onset favours the latter diagnosis. Precipitation by shock, surprise or emotional distress must be interpreted with caution, since such factors may trigger epileptic attacks in susceptible individuals. Pseudoepileptic seizures may be provoked by hyperventilation, which will on occasion precipitate a true ictal event. The seizure duration is the same for the two types of attack, though a gradual termination is more often observed in pseudoepilepsy (Lesser, 1985).

Pseudoepileptic attacks usually occur when other people are present, especially those of significance in the patient's life, but there are exceptions. Unlike true epileptic seizures, pseudoepileptic seizures never occur during sleep. Careful enquiry about the person's state of awareness at the time of the onset of the seizure is necessary, since patients may develop pseudoepileptic attacks immediately on waking from sleep. Pseudoepileptic seizures are sometimes frequent, attacks recurring many times per day; this is unusual in real epilepsy, with the exceptions of status epilepticus, petit mal absences and some patients with minor complex partial seizures. Indeed, the recurrence of persistent and frequent major fits despite adequate anticonvulsant drug dosage, evidence of satisfactory compliance, and serum concentrations within the optimal range should raise the suspicion of pseudoepileptic seizures.

Classification of pseudoepileptic seizure manifestations

Analysis of videotaped pseudoepileptic fits has led to attempts to classify the range of clinical phenomena. In a study of 27 patients (Gulick *et al*, 1982), motor manifestations were found to be bilateral in 15 and unilateral in 3, while 8 displayed multiple behavioural phenomena and 3 decreased responsiveness only. Luther *et al* (1982) observed generalised convulsive-type attacks in 15 out of 37 attacks in 30 patients; 21 resembled elementary partial seizures with secondary generalisation, and one a complex partial seizure.

Lesser (1985) has proposed the following main categories of manifestation: sustained or repetitive muscular contractions, either unilateral or bilateral; muscle inactivity or loss of tone; unresponsiveness alone; and unresponsiveness in association with quasi-purposeful or automatic behaviours. Episodes may occur either with or without an aura and, in any one patient, more than one of these patterns may coexist.

Some unusual presentations of pseudoepilepsy

Repeated pseudoconvulsive seizures without recovery of consciousness between attacks, with incontinence of urine or tongue biting during some of the fits, may simulate status epilepticus, leading to transfer to an intensive care unit and exposure to the potential hazards of apnoea and hypotension due to the vigorous pharmacological treatment of the apparent status. Toone & Roberts (1979) described three such patients, and made the point that some patients with pseudoepileptic seizures have attacks that bear a striking resemblance to genuine epilepsy. This is particularly true of patients with some experience or knowledge of epilepsy, a problem among nurses with pseudoepileptic seizures; two of the patients described by Toone & Roberts were nurses. I vividly recall a trained psychiatric nurse who had a series of hospital admissions for control of status epilepticus. It was only when the hemiconvulsions suddenly and inexplicably changed from one side of the body to the other after the intravenous cannula and drip set delivering the medication and nutritional fluids had been inserted in the opposite arm that the suspicion of pseudoepilepsy was aroused! This diagnosis was confirmed during a prolonged period of in-patient observation, including gradual withdrawal of anticonvulsants and replacements by a placebo. In organic status epilepticus the patient does not regain consciousness in the postictal period, and has further seizures separated by intervals of not more than 15–30 min.

As the status progresses the seizures become shorter and less vigorous, but there are never any thrashing or throwing movements. The diagnosis should be verified by electroencephalography (EEG); there will always be seizure discharges in association with the attacks in status epilepticus. In pseudostatus epilepticus the EEG tracing shows only muscle movement and electrode artefacts during the pseudoconvulsive episodes.

On rare occasions focal neurological signs can be manifest during pseudoepileptic fits. One patient has been described who produced pupillary dilation and an extensor plantar response by pressing his thumb into the left side of his neck (Hammond, 1948). Another rare but potentially confusing manifestation of pseudoepilepsy can be recurrent injury. I recall a young student nurse with pseudoconvulsive seizures; after each attack she invariably had bruising over the face, limbs and trunk. Such extensive bruising is rarely seen after a grand mal convulsion. Hence, if the nature and extent of the injuries received during a fit are inappropriate for the circumstances of the seizure, the epileptic nature of the attacks should be open to critical re-examination.

Although the most common type of pseudoepileptic seizure is the pseudoconvulsive type, less dramatic forms do occur. Some patients may present with transient episodes of unconsciousness associated with pallor and slumping to the ground without convulsive movements resembling syncope. However, in the latter condition the patient is usually responsive by the time he or she falls to the ground, the face is pale, and there is no resistance to eyelid opening or to testing the corneal reflexes. In the pseudoepileptic seizure patient the period of unresponsiveness will usually be longer, and active resistance to eye opening may be manifest. The facial colour is either normal or flushed.

Some brief attacks of pseudoepilepsy may resemble simple partial seizures of the motor type, with twitching of one side of the face or jerking of limbs down one side of the body. The cortical motor representation of the face, thumb and hand occupies the largest proportion of the lateral surface of the motor cortex (Penfield & Jasper, 1954); the trunk has a small representation, and the leg areas are on the mesial surface. For this reason, epileptic attacks originating in the motor area affect mainly the face, thumb and hand. Jerking of shoulder or elbow in the absence of jerking of face and hand is therefore unlikely to be of epileptic origin. Since in most right-handed people and in 60% of left-handed people speech is dependent on the function of the left hemisphere, a right-sided motor seizure is usually associated with speech arrest; if speech is

preserved during a clinically right-sided seizure and the patient is not left-handed, an epileptic basis is therefore unlikely. The movements of the focal motor attack are either tonic or clonic in nature, in contrast with the trembling or grossly irregular shaking of the pseudoepileptic variety. Sensory manifestations may occur as part of a minor pseudoepileptic attack. However, the location and spread of the sensory phenomena do not usually fit into a consistent and orderly pattern that reflects progressive involvement of adjacent areas of the brain by the spreading seizure discharge.

Hysterical fugue states, complex partial seizures and transient global amnesia

An hysterical fugue may be misdiagnosed as a complex partial seizure with automatic behaviour. However, automatic behaviour of epileptic origin is of brief duration, lasting minutes rather than hours. A classical temporal lobe aura may precede the attack. During an epileptic automatism there is usually evidence of clouding of consciousness, poor appreciation of the environment, and purposeless, inappropriate automatic behaviour. The duration of the hysterical fugue is hours or days. The patient's behaviour is usually well structured and co-ordinated and not inappropriate to the circumstances of the environment, despite the lack of knowledge of personal identity. Indeed, the behaviour seems goal-directed, the unconscious motivation being to escape from an intolerable situation.

Transient global amnesia is another disorder that can be confused with both hysterical fugue states and epileptic automatism. It differs from the latter condition in that the patient continues with routine activities but fails to lay down any permanent memory traces throughout the episode. Knowledge of personal identity is retained during the attack. The behaviour is well-structured and consistent with what is going on in the environment at the time. Memory gaps are not restricted to matters of personal concern nor specific emotionally loaded themes, and there are no inconsistencies in memory performance.

Other episodic organic disorders that simulate hysteria

The discussion of differential diagnosis has so far emphasised the need to avoid the mistake of diagnosing true epilepsy, when the attacks are actually those of pseudoepilepsy. Less frequent, but just as important, is the risk of wrongly attributing the

diagnosis of pseudoepilepsy to episodic disturbances of behaviour that have a truly epileptic basis.

Patients with paroxysmal choreo-athetosis suffer from episodes initiated abruptly by restless movements of upper and lower limbs which progress to typical choreiform activity. The movements may be unilateral with generalisation, symmetrically bilateral or strictly unilateral. The patients remain conscious, usually do not fall, and are acutely embarrassed by the occurrence of such attacks, which may last between half an hour and several hours. They tend to be precipitated by emotional stress, coffee, or beer, and may be aggravated by anticonvulsant medication—especially phenytoin, which is sometimes prescribed when a seizure disorder has been mistakenly diagnosed. There is frequently a positive family history for this condition.

In startle or movement-induced seizures the patients suddenly drop to the ground with associated loss of consciousness as a result of a sudden startle stimulus or of suddenly rising from the sitting position. The attack is short-lived, and the patient returns to consciousness immediately. It usually results from a myoclonic jerk, and the EEG frequently shows spike wave discharge. Myoclonic jerks occur mainly in patients with primary generalised tonic-clonic seizures, but can be a manifestation of such progressive brain syndromes as Lafora's disease, subacute sclerosing panencephalitis and Creutzfeldt-Jakob's disease. Sometimes (in primary generalised epilepsy and Lafora's disease) the myoclonus is severe and causes gross uncoordination of movement and gait. In some patients, emotional stress greatly aggravates the myoclonus. The link between the patient's emotional state, the jerking and apparent ataxia can lead to an erroneous diagnosis of hysteria, especially as the disorder may have an episodic quality and may lead to the avoidance of situations. Myoclonus tends to be worse in the mornings. The jerks often come in rapid succession, and herald a tonic-clonic seizure. EEGs frequently reveal that they are associated with bursts of generalised spike wave activity, and photic stimulation may elicit the myoclonus and associated generalised spike wave complexes.

The psychic auras that often initiate the complex partial seizures of temporal lobe epilepsy can be a bewildering and distressing experience for some patients. Those people whose habitual way of coping with psychic pain is denial may dissociate into a fugue state in response to the distressing subjective experience of the aura. Alternatively, they may respond by manifesting disinhibited, explosive behaviour in an attempt to escape the stressful

event. In my experience such reactions are rare, but undoubtedly do occur, especially in patients of low intelligence and those predisposed to using hysterical mechanisms to deal with stress. For instance, a 30 year old man of low intelligence and limited social and verbal skills had problems in relating to the opposite sex; at a time when he was having difficulties with a girlfriend, he experienced several attacks of panic followed by disinhibited behaviour. The attacks responded to chlordiazepoxide, and were considered to be an hysterical response to stress. It was only when the same sequence of events was repeated several years later, when he was having difficulties with another girlfriend, that the attacks were shown to be of temporal lobe origin with secondary fugue states.

Aetiology of hysterical pseudoepileptic seizures

Kendell (1974) and Smith (1978) have formulated essentially similar aetiological models for hysteria, in which hysteria develops as a response to emotional stress or conflict when a series of environmental, biological and personal predisposing (vulnerability) factors are present within the individual or as part of the current life situation. These are an illness-rewarding situation (when the advantages of being sick outweigh those of health), and individual variables such as female gender, youth, inferior social status, immigrant status, low intelligence, hysterical or passive-dependent-immature personality, suggestibility, difficulty in the overt expression of feelings, a tendency to dissociate and use denial mechanisms as a means of coping with stress, and personal experience of physical illness in relatives, self, peers or acquired as a member of one of the health care professions. The symptoms and signs of the resulting hysterical syndrome mimic organic disease, and permit the patient to adopt the sick role with consequent relief from the precipitating stress or conflict.

One important element missing from this model is organic brain disease. This seems to facilitate the use of hysterical mechanisms (Slater, 1965; Whitlock, 1967; Merskey & Buhrich, 1975; Merskey, 1978*a, b*; Roy, 1980, 1982) and is reported in 40–63% of patients with hysteria. Although Slater made the important observation that hysteria can herald the onset of the central nervous system disease, pre-existing organic brain disease is also a common predisposing factor to the development of hysterical symptoms.

This modified model clarifies the genesis of hysterical pseudoepileptic seizures. Precipitating stress factors are frequently present (80% of those studied

by Standage & Fenton (1975)–53% psychological and 27% physical). An illness-rewarding situation is often apparent. Anxiety and depressive mood change is common, as is other evidence of psychological maladjustment (Roy, 1977, 1980, 1982). The sex distribution is predominantly female, personality disorder is not infrequent, and organic brain disease is reported in between one-quarter and one-half of patients. The brain dysfunction tends to be acquired early in life, usually before the age of 15, and is often accompanied by cognitive impairment (Standage, 1975; Standage & Fenton, 1975; Roy, 1977). Side-effects of anticonvulsant drugs can be another factor. Previous episodes of unconsciousness, including epileptic fits either experienced personally or observed within the family or work environment, often provide a model for the choice of pseudoepileptic seizures as the hysterical manifestation. Unconscious symbolic processes are not important in symptom choice (Standage & Fenton, 1975).

Investigation

As already explained, the correct diagnosis of patients with pseudoepileptic seizures can be difficult, especially those who present with a history of both well-controlled epilepsy and frequent pseudoepileptic seizures. Many such patients will have been treated with large doses of anticonvulsant medication in the mistaken view that uncontrolled epilepsy is the problem. Even when the differential diagnosis seems straightforward, it is often impossible on an out-patient basis to elicit any underlying psychological difficulties to account for the pathogenesis of the attacks. The use of denial mechanisms in response to conflict or stress inevitably leads to defensiveness and denial of emotional difficulties during initial diagnostic interviews. Indeed, even the mere suggestion of an emotional basis for the attacks will often be angrily rejected. Similar attitudes are usually present in the patient's close relatives. To overcome these difficulties it is best to admit the patient to hospital, where the clinical pattern of the seizures can be carefully observed and described, preferably by staff who have special experience in the observation of fits.

Electroencephalography

EEG abnormalities are not infrequently recorded, but must be interpreted with caution. The presence of EEG paroxysmal abnormalities of the type associated with epilepsy does not necessarily confirm that the observed attacks have an epileptic basis. The

former can occur in the pseudoepileptic patient for one or more of the following reasons:

- (a) underlying epilepsy, usually well controlled
- (b) bursts of generalised spike wave complexes or paroxysmal theta activity, due to anticonvulsant drug withdrawal. Such withdrawal effects may persist for as long as 21 days after the end of the withdrawal phase
- (c) generalised fast spike and wave complexes are found in nearly 3% of apparently healthy people, presumably being a reflection of an unduly low convulsive threshold (Fenton, 1974).

Background activity abnormalities such as focal slow waves, a marked alpha or beta rhythm asymmetry or diffuse slowing of the dominant activity frequency are evidence of organic brain dysfunction. However, their presence in a patient with seizures of uncertain origin cannot be regarded as unequivocal evidence that the attacks are epileptic, since organic brain disease is an important predisposing factor in the genesis of pseudoepileptic seizures. Anticonvulsant drug intoxication produces diffuse EEG background dominant activity frequency slowing, and can also cause pseudoepilepsy to develop. Hence, the routine EEG is of limited help and may even be misleading in the investigation of pseudoepileptic attacks.

Prolonged EEG recording over hours or days, using either conventional leads, a videotape telemetry system, or ambulatory monitoring with a portable tape recorder that can be carried about by the patient, often makes it possible to record an actual seizure (Oxley *et al*, 1981; King *et al*, 1982). Muscle and movement artifacts caused by the convulsive movements may make the presence of ictal discharge during the seizure difficult to identify. However, scrutiny of the recording just prior to seizure onset and after cessation of the clinical seizure can be fruitful. An epileptic seizure of partial (focal) origin may be accompanied by an EEG seizure discharge lateralised to the hemisphere origin prior to onset of the clinical manifestations of the attack. In addition, during the postictal phase there may be an asymmetry of EEG background activity, with apparent reduction in normal rhythms and the appearance of slow waves over the hemisphere of origin of the seizure discharge. After a generalised convulsion there are always postictal EEG changes; transient electrical silence is followed by the appearance of irregular slow waves which, over the next few hours, are gradually replaced by normal activity. Hence, if the recording of an actual seizure is neither possible nor convenient, it is still useful to record an EEG within an hour of the occurrence of the seizure, so

that the postictal changes (if any) can be studied. However, their absence cannot exclude epilepsy, since the changes even after a real grand mal attack may be only present for a few minutes. An attack with major convulsive movements without EEG changes is almost certainly non-epileptic in nature, but in true partial epileptic seizures there may also be an absence of any scalp EEG change.

If the patient is receiving anticonvulsant therapy on admission, the medication should be continued until there has been sufficient time to observe the nature of the attacks. If the evidence in favour of an epileptic basis is not convincing, the anticonvulsant drugs can then be gradually replaced by placebos. As well as depressing cognitive function, heavy anticonvulsant medication can suppress the EEG paroxysmal abnormalities associated with epilepsy. However, it is often difficult to obtain appropriate placebo tablets. If so, the reasons for the proposed drug withdrawal will have to be discussed with the patient and the drug withdrawal procedure can then be carried out with the patient's knowledge and consent. In the event of placebos being used, the placebo nature of the medication will have to be disclosed to the patient; this is best done at an appropriate time during the parallel programme of psychotherapy. If, following withdrawal, real epileptic attacks recur, anticonvulsant medication will have to be reintroduced. The dosage required is usually small.

Other useful investigations

Serum prolactin levels rise sharply after many, but not all, generalised tonic-clonic convulsions to 1000–2000 mUnits/litre; this may be helpful in differentiating epileptic grand mal seizures from non-epileptic pseudo-grand mal attacks (Trimble, 1978; Lesser, 1985). A major difficulty is that both false-negative and false-positive results have been reported after major convulsive attacks. Furthermore, significant elevations are less common and less consistent following complex partial seizures, and do not occur after simple partial seizures (Dana-Haeri *et al*, 1983; Lesser, 1985). Therefore, postictal serum prolactin changes must be interpreted cautiously in the light of the associated clinical and EEG manifestations. Since organic brain disease is a common finding in patients with pseudoepileptic seizures as well as epilepsy, neurological investigation is not as helpful as one might predict. However, a computerised tomography scan will often identify underlying pathology of the central nervous system, and is useful in excluding progressive brain lesions that present with hysterical manifestations.

Treatment

The psychological management of the patient should run in parallel with the programme of observation and investigation. Rapport will be gradually established during the initial interviews. As rapport and mutual trust between doctor and patient develops, the façade of denial will gradually be penetrated and a process of clarification and non-directive discussion of the patient's symptoms and current life problems and their inter-relationship can begin. As the link between the fits and their emotional state begins to be understood by the patient, the acceptance of the psychogenic origin of the attacks becomes possible without loss of face. If a videotape recording of the pseudoepileptic attack is obtained, it can be used to show the patient what he looked like during a seizure. The non-epileptic nature of the movements or behaviour can be pointed out, along with a demonstration of the normal EEG data to both patient and family. This procedure can facilitate the development of insight into the nature of the seizures (Feldman *et al*, 1982).

Providing the patient's intelligence level is appropriate, and motivation for self-examination adequate, the exploratory psychotherapy can progress to an examination of the psychological meaning of the illness and the habitual defence mechanisms used against anxiety. The patients should be encouraged to acknowledge and discuss their emotional difficulties and tensions as a healthy alternative to denying their existence. Such a psychotherapeutic approach will tend to focus on an examination of the relationship between the therapist and patient (transference) and how this reflects relationships in his or her formative years and current life situation. The number of sessions of such focal psychotherapy will depend on the amount of available time and the therapist's assessment of the patient's capacity to benefit. A minimum of ten and a maximum of thirty one-hour sessions will be required. If this psychodynamic approach is inappropriate because of low intelligence, poor motivation for self-examination, or inability to cope with the inevitable transference problems, a more supportive psychotherapeutic role is still helpful, the aim being to permit abreaction, reduce emotional tension, and provide encouragement, support and guidance in dealing with current life problems.

One of the basic principles of learning theory is that patterns of behaviour that are rewarded tend to increase in frequency. When the pseudoepileptic nature of the attacks is firmly established, the staff should be encouraged to ignore their occurrence, but to respond to the patients when they are free

from seizures. The patient should be encouraged by all members of staff to express and discuss emotions when distressed. Events in the ward environment that upset the patient and are followed by attacks can be discussed and used as examples of the operation of dissociation and denial mechanisms. More healthy ways of coping with such potentially stressful events can be encouraged and rehearsed. It is important that all members of the therapeutic team adopt a consistent approach to the patient. This must be sympathetic but firm. Clear guidelines about how to handle the varied repertoire of behaviour must be formulated so that the patient is not given the opportunity to manipulate individual staff members.

As patients' awareness of their emotional difficulties grows during the psychotherapy, the fits tend to subside. However, the patients may react by becoming depressed or by developing other conversion symptoms; alternatively, they may show their distress by self-damage, damage to property, or by various types of demanding histrionic behaviour. Such behaviour should be ignored as much as possible, but if it becomes seriously disruptive firm boundaries defining the limits to be tolerated by the staff should be drawn up and communicated to the patient by the therapist. A consistent approach is facilitated when all members of staff can meet regularly to discuss management, either during regular staff meetings, ward rounds, or clinical conferences.

Work with the patient's parents or spouse is always required. Simple explanation about the nature of the attacks and their relationship to emotional conflict and tension is essential. Advice to avoid rewarding their occurrence with too much attention, sympathy or concern is necessary; again, the need for a consistent approach should be stressed. Family or marital psychotherapy may be necessary if problems exist. A further strategy of management should be based on an attempt to minimise the disadvantages of health and the advantages of sickness. This will require careful and detailed inquiry to elucidate the full implications for the patient of both states. If the stress which caused the patient to adopt the sick role is a temporary one, little is needed beyond a firm prediction of recovery and the provision of a treatment setting in which this can take place gracefully. If the advantages of the sick role are substantial and enduring, however, a carefully planned regime will be needed. This will involve the patient's relatives.

After discharge from hospital, a long-term supportive relationship with the therapist is helpful. Contact should be relatively frequent (once every few weeks) for the first few months, in order to cope with crises that may arise, followed by a gradual

lengthening of the intervals between interviews (to once every few months). The follow-up should be continued for at least two years. Contact with relatives should also be maintained after the patient leaves hospital, in order to provide support and advice about management. It is also important to inform all medical personnel concerned with the patient's care of the management strategy, especially the general practitioner. This will avoid the reintroduction of, or increase in, anticonvulsant medication and further unnecessary investigation should the pseudoseizures recur. It is best if the management is supervised by one person.

Should the patient prove unresponsive to the combined dynamic and behavioural psychotherapy approach, an alternative is to use a seemingly paradoxical procedure with the consent and co-operation of the relatives: the patient is informed that the fits are likely to continue unabated, since all attempts to understand and treat them have failed, and that their frequency may even increase. Because conventional treatment methods have been unsuccessful, strict bed rest with a minimum of sensory

stimulation is recommended: a bed with cot-sides in a single darkened room with only basic amenities and no books, television or visitors. The restrictions on activities and other amenities are systematically lifted as the seizure-free intervals lengthen, but are reimposed in reverse order should relapse occur. In resistant patients this simple behavioural approach can lead to a dramatic reduction or even abolition of seizures, but must be followed by continued supportive psychotherapy and work with key relatives.

The long-term outcome for pseudoepileptic seizures is still subject to debate and disagreement. Reported improvement rates have varied from 30–80% (Lesser, 1985). The widely differing views about progress probably reflect the nature of the unit the patient is referred to and the programme of management used. Having had personal experience of the management of a substantial number of such patients, it is my view that the majority respond to a carefully structured programme of psychological management. However, some may continue to react at times of stress with occasional clusters of pseudoepileptic seizures. A few remain a chronic problem.

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Hysteria, Play-acting and Courage

DAVID C. TAYLOR

Levi-Strauss (1977) describes what happened to a child, in a primitive tribe, accused of sorcery—a crime punishable by death. The child invented more and more complex stories by way of self-justification. In the end his accusers believed his outlandish explanation because “the choice is not between this system and another but between this system and no system at all—chaos.” The child’s plight can represent the close affinity between magic and hysteria; his accusers might represent those contemporary psychiatrists who would prefer any system of classification of hysteria, however absurd, to no system at all (Sheehan & Sheehan, 1982a, b; DSM-III, 1980).

Commenting on similar issues, Mayou (1984) explained how lack of agreement and precise definitions cause great difficulties for psychiatry. Underlying the imprecision is a general failure to appreciate the importance of the fact that the different sicknesses to be classified are in different universes of discourse, and that any attempt to develop one system to embrace processes, things, and states of affairs is bound to fail (Taylor, 1979, 1982). Within this chaotic classification hysteria holds pride of place for ambiguity. Doctors wishing to dispose of the problem by abandoning the diagnosis, however, are mortified by the thought of how

otherwise to describe their next patient with those symptoms.

This mortification will persist until the concept of ‘hysteria’ is replaced by a better explanation. Some people believe that this will come from the biological sciences, others that it will come from psycho-social case analysis and epidemiological techniques. Some believe that if sickness is not physical it must be psychological, or again that if sickness is not physical it is not our business. I believe that hysteria is generated as a defence mechanism and propagated as a consensual phenomenon, as is magic. Like magic, it has no real properties beyond belief. Granted that it is a belief, the physiological and psychological characteristics of the believer are probably irrelevant. But beliefs, convictions and avowals, however, are powerful phenomena.

Hysteria persistently imposes itself upon medicine by threatening the implicit contract of trust between doctors and their patients. A group of people, referred to by Bayliss (1984) as ‘the deceivers’, pose problems not just of diagnosis but also of management, resource allocation and ethics. Having mentioned the Munchausen syndrome, illness by proxy and factitious disorders, but having studiously avoided the word ‘hysteria’, Bayliss remarked that “these patients are seldom malingerers” and that

"quite often, before the diagnosis is firmly established, an experienced psychiatrist will declare the patient as perfectly normal and unquestionably suffering from organic disease". This formulation, by a physician, is an important index of the contemporary standing of psychiatry as well as of hysteria and its group of inextricably associated phenomena. His point was that it is crucial for biomedicine not to be deceived, because it is potentially dangerous, wasteful and deprecatory.

Consider the emergence of a hysteria: a person in a predicament lays claim to sickness. This is probably a commonplace event, and can be regarded as tantamount to 'faints' (Daymas, 1982) or 'pax' in children's games. This particular claim, however, in this predicament, finds the opportunity for validation among the family or living group instead of being politely ignored or being offered temporary token regard. The sickness is now presented to the family physician; the physician attempts a rational validation, which naturally fails. If failure is an unacceptable outcome, a higher authority must be invoked. Since specialists specialise in organ systems, one system must be defined as 'sick' by the family doctor in order to identify the appropriate specialist. This process helps to refine the presentation, and of itself partly validates it. Rather than providing an elucidation, the specialist usually merely confirms or denies the putative disease. Not validating the disease does not invalidate the claim (Taylor, 1982); the patient still lays claim to it, since little alternative has been made possible. The medical view is that it must be due to 'something else', and if nothing else offers itself then 'hysteria' is the diagnosis that will be made.

Hysteria, the laying of claims to illnesses and diseases the presence of which cannot be corroborated, thus becomes in itself an illness. In effect, what doctors are saying is that claiming to be sick when not sick is a variety of sickness. However contradictory that may seem, doctors feel justified in that belief by the manifest distress that can be evident in the original predicament and the subsequent development of the sickness. What we have hitherto lacked the courage to say is that a good deal of sickness is attributable to the enactments of distress and the playing-out of scenarios in which people exhibit distress through whatever scope is left to them. The body speaks what the tongue cannot utter. From this common situation, without any useful reference to physical or mental illness, some people are led astray and persuaded into severe sickness.

The alternative viewpoint, dignified by a thousand years of medical tradition, is to treat hysteria like any other sickness. For example, Breuer (1895)

regarded it as "a clinical picture which has been empirically discovered and is based on clinical observation in just the same way as tubercular pulmonary phthisis". This is a false postulate, because 'clinical pictures' are not in the same universe of discourse as 'pulmonary tubercles'. It leads to the false proposition that the cause of hysteria will be found by detailed investigation of people deemed to be sufferers, in much the same way as is tried for schizophrenia and was successful for *Legionnaires' disease*.

My most frequent connection with hysteria comes from young people who are persuaded that they suffer from epilepsy and wish to encourage others to the same belief. They are identified to me by their partial success, for if they failed or completely succeeded I should not notice them. The difficulty to which they give rise was characterised by Worster-Drought (1934): 'It is extremely difficult, in fact almost impracticable, to draw a hard and fast line between hysteria and epilepsy'. His paper was written just before the advent of EEG, at the watershed of ancient and modern epileptology.

Since 1934, the late 19th century concept of epilepsy as an electrical discharge from neurones spreading by progressive recruitment has been vindicated and developed down to neurochemical levels. With hysteria, however, we have been encumbered with ideas based on psychic energy, or flux, which might be blocked, diverted, or converted; these ideas have led nowhere. The contrast might be seen as an aspect of the relative failure of psychiatry, but it is more usefully seen as suggesting that hysteria is not biomedical in nature. This does not mean that it does not exist at all. As Widlocher (1982) wryly noted: "The last trick that the Great Neurosis plays on medicine will be to slip away down the rift caused by the separation of neurology from psychiatry". Hystero-epilepsy is a way of acting in a way which makes people wonder if one is having a fit. Most people who do this really are having a fit. They see doctors who believe them and investigate them for disease.

But 'disease' is only one component of sickness, which is also composed of 'illnesses' and 'predicaments' (Taylor, 1982). These terms owe something to Karl Popper's conception of three worlds (Popper & Eccles, 1977) to which we attend; world I, a world of material bodies or 'things'; world II, a psychological world of mental states; and world III, a world of human artefacts such as novels, plays, and enactments, which although abstract are equally real. 'Illnesses' cover most of the psychiatric disorders (world II), and 'predicaments' are those situations which are human constructs which can be

seen as excruciatingly painful and problematic (world III).

There is little evidence to support the idea of psychopathology in children with hysterical symptoms, so there is little to suggest that the explanation of hysteria will prove to be in world II. I think that hysteria is a world III phenomenon – an artefact like a story or play, a fictive consensual belief. This is easiest to understand in the ‘mass’ hysterias which are frequently reported throughout the world (Small & Borus, 1983; Mohr & Bond, 1982; Dhadphale & Shaikh, 1983). It is ludicrous to suppose that personal psychopathology predisposes the actors in these dramas in any meaningful way. It does not mean that the effect is trivial: nine hundred people were persuaded to commit synchronous suicide by James Jones in Guyana. Even in the most trivial incidents there are always some who are reluctant to relinquish their new convictions. The forms and the victims, the situations and the causal explanations are so regular as to have the air almost of a ceremony (McEvedy & Beard, 1970). It is just more difficult to see the social and group dynamics which produce the individual cases. The casting and the auditioning have occurred elsewhere, and in secret. Even more importantly, when sickness emerges in an individual it is not necessarily realised that it is a World III phenomenon, and the social group, as well as physicians, will quite properly regard it at first as being in World I: a potential disease.

The problem is that the enactments may come from the depths of the behavioural repertoire, from protective and adaptive mechanisms which we share with other animals. Many ground-nesting birds such as the curlew will lure birds of prey away from attacking their young with a display of feigned incapacity. Kretschmer (1961) perceived a range of reactions by which animals effect their own removal from overwhelming stress. One, translated as ‘violent motor reaction’ (Kretschmer, 1926) or ‘instinctual flurry’ (Kretschmer, 1961), describes uncoordinated frantic attempts to flee. It is typified by the behaviour of a bee or a bird in a room, or people at the scene of a disaster. Another is the ‘death feint’ or ‘playing possum’. These are allaesthetic displays designed to alter the behaviour of other animals. *Peromyscus* (the deer mouse), for example, maintains in a small proportion of its population a gene for convulsing if shaken, which helps reduce risk from predators. (Chance & Russell, 1959). Neither of these ‘hysterical’ reactions is entirely without risk; ill-organised attempts to escape disaster may carry a survivor towards rather than away from danger, and ‘playing possum’ or ‘playing hurt’ may occasionally just make things easier for the predator. The study

of these important reflex defences provides the best option for biomedical study of hysteria, but they are probably biomedically transient and ‘hysteria’ is suffered by people who have become caught up in an extended enactment.

In man, these behaviours may be interpreted as ranging from the reflex to the contrived. As such, they are subject to moral evaluation. They are judged more or less negatively depending upon the degree to which they seem personal to the patient and demanding of others, or are desirable and clearly consensual beliefs. At one end of the scale, extreme deliberate abuse of the ill-health system is called malingering; at the other, placid concurrence with therapeutic intent is called a placebo response. Being persuaded is more or less reflexic. A mother who had abused her child was speaking to social workers while her infant was screaming beside her. She raised the infant to within inches of her face and bellowed “Shut up”. The child lapsed immediately into a stupor which lasted several hours. This is an extreme case of the well-known phenomenon of an ‘over-tired’ child lapsing suddenly into sleep at a sharp remonstrance. Similar reactions have been described in people who have just been told that they have cancer (Beecher, 1962; Milton, 1973; Stedeford, 1984). At their extreme they bring us back to magic and to hystero-epilepsy.

Cannon (1942), in his article on voodoo death, quoted Basedow (1925): “... the expression on his face becomes horribly distorted ... He attempts to shriek, but the sound chokes in his throat, and all that one might see is froth at his mouth. His body begins to tremble and the muscles twist involuntarily ...”. The victim is persuaded to die. It is a consensual phenomenon, because everyone knows that he will die and they withdraw their social support. (What if the social supporters ‘know’ that someone is ‘ill’?) Cannon observed that one of the interesting characteristics of this mode of dying was its astonishing reversibility from states near to death, given adequate counter-suggestion. The important finding relevant to hysteria is that those who have recovered from these states say that they did not wish to die but recognised that they had been victims of forces which had made death inevitable. Cannon adds: “the phenomenon is characteristically noted among aborigines – among human beings so primitive, so superstitious, so ignorant that they feel themselves bewildered strangers in a hostile world.” I suggest that he meant they were child-like, in an animistic phase of personal as well as cultural development, under the influence of others more powerful – rather like Charcot’s patients in the Salpêtrière.

This view of hysteria turns upon persuasion: the

strength of the persuasive forces, the liability to be persuaded, the capacity to engage in the drama, and the effectiveness of the escape. In it 'conversion' is appropriate in its religious rather than its Freudian sense. It may serve a purpose, or it may be a trap; it may be transient, or it may ensnare. The sorts of extreme situations described by Woodruff *et al* (1982) imply people having been trapped into a life of hysteria, just as other people are trapped into living through epilepsy or other chronic sicknesses.

The elements of an hysteria are discernible in most serious writing on the subject, but deserve to be clearly stated. The first requirement (Taylor, 1979, 1982) is a predicament which is intolerable but for which all apparent solutions are blocked. The traditional example of this, from Freud's own observations, follows sexual abuse, but there are equally painful alternatives. The predicament, or the blocked solutions, may concern the psychopathology of the 'hysteric', but in childhood hysteria most authors now agree that little abnormality is detected (Dubowitz & Hersov, 1976; Maloney, 1980; Goodyer, 1981). Secondly, an ally who is helping to promote the sickness is required. This person acts like a manager; he too may have been innocently trapped, or else have his own psychopathology. Allies may believe only in world I sickness, quite justifiably deny world II problems, and have no conception of world III. These allies will vigorously defend the 'right to be sick', and pursue disease explanations relentlessly. Thirdly, a model of the sickness has to be available, whether current or historical, real or contrived, chronic or ephemeral. Thus one patient who twitched in a faint on being given an injection to protect her against sicknesses of equally uncertain provenance was asked if there was epilepsy in the family. (Taylor, 1985). Finally, hysteria can only be promoted by persons with the necessary social skill or by those who derive that skill through their allies. This con-

straint, among others, precludes young children from hysteria and probably defines the border with malingering and Munchausen's syndrome.

Doctors in particular, and other health care workers to some extent, can provide well for all these elements. They can block alternative explanations by patients, or by failing to take an adequate history of their predicament fail totally to discern it. They are powerful allies in sickness promotion, and can be sucked into the system quite unwittingly, especially if they have an investment in a biomedical diagnosis. They provide a variety of models and can offer suggestions which improve the credibility of the sickness. They have the requisite social skills. They are consistently intermittently reinforced in their belief in the organic substrates of hysteria (Slater, 1965) by the literature and by their own experience. They seem never to have been told that the emergence of organic disease in no way invalidates the hysteria, which often uses a different body system and in any case is a phenomenon in a different domain. It needs courage for physicians to help their patients most by making a decision on the balance of benefit, not to exclude every possible organic cause for a sickness.

This view of hysteria is similar to part of that recognised by Kretschmer (1926), Rabkin (1964), Kendell (1982) and Slater (1982), and well argued by Mayou (1975). Their view is that hysteria is a social construction set up in the doctor-patient relationship. Hysteria, the laying claim to sickness for which there is no objective evidence, is a commonplace reaction, and those who become dignified by a formal diagnosis are a severe, extreme, or fortuitous selection. Treatment requires detailed analysis of the predicament, negotiation with the allies, uncovering of the model (especially from fantasy), and the selection of a dignified alternative by a figure powerful enough to alter firmly rooted beliefs.

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A Controlled Assessment of Propranolol in the Treatment of Neuroleptic-Induced Akathisia

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Twelve patients with neuroleptic-induced akathisia were treated in a randomised, double-blind, cross-over design with propranolol and matching placebo. Propranolol caused significant decrements in both subjective and objective ratings of akathisia, but not in anxiety scores. This confirms prior findings of the efficacy of propranolol in akathisia induced by neuroleptic treatment.

Akathisia is a frequent and often disturbing side-effect of neuroleptic therapy; its literal translation is an inability to remain seated (Braude *et al*, 1983; Raskin, 1972). It occurs most frequently with high-potency neuroleptics, but can occur with any of these agents. Braude *et al* delineated mild, moderate, and severe forms of the syndrome. In mild akathisia, the symptoms are mainly subjective in nature and involve an uneasiness and inner restlessness, more often sensed in the legs. In addition to these complaints, patients with moderate akathisia have: rocking from foot to foot, walking in place, a coarse tremor, and myoclonic jerks in the feet. The severe form further involves an inability to maintain one's position, also known as *tasikinesia*; this leads to frequent arising from a seated position or pacing.

The frequency with which akathisia occurs is between 12.5% (Freyhan, 1958) and 45% (Van Putten, 1975); the most common estimate of prevalence is about 20% (Braude *et al*, 1983; Ayd, 1961; Marsden & Janner, 1980). The symptoms of akathisia can be so distressing as to hinder compliance with treatment or exacerbate psychopathology (Van Putten, 1974).

The treatment of akathisia is often difficult. Therapeutic approaches include: anticholinergic medications (Van Putten, 1974, 1975), benzodiazepines such as diazepam (Donlon, 1973; Gagar *et al*, 1978) and lorazepam (Germino, 1985), and the anti-parkinsonian drug amantadine (DiMascio *et al*, 1976). Clinically, these treatments are often only partially effective and, particularly with anticholinergics, side-effects frequently limit the dose. However, recent evidence indicates that beta-adrenergic blocking medication can be effective. Lipinski *et al* (1983, 1984) and Zubenko *et al* (1984) have reported open studies in which propranolol, a beta blocker

with both central and peripheral effects, in low doses (30-80 mg/day) led to marked improvement of akathisia. We have also found, in a prospective, single-blind study, that propranolol (20-30 mg/day) was superior to lorazepam (2 mg/day) (Adler *et al*, 1985). Ratey *et al*, (1985) have also described several patients in whom this condition was effectively treated with nadolol, a beta-adrenergic blocking drug with mainly peripheral effects.

To further examine the effect of beta blockers on akathisia, we conducted a randomised, double-blind study of propranolol.

Method

In-patients and out-patients receiving neuroleptics, who had akathisia-like movements, were identified. Patients were enrolled if these movements were of sufficient intensity to be rated at least '1' on a modification of the original Simpson/Angus EPS Scale (Simpson & Angus, 1970) ('Mild; occasional restlessness observed during examination and/or subjective report of definite restlessness'). A physical examination and ECG were done; patients were excluded if they had any contraindication to receiving beta blockers, such as asthma, insulin-dependent diabetes mellitus, or any cardiac conduction abnormality or concurrent cardiovascular illness which required medical treatment.

Patients were randomised to a 6-10 day treatment period with either propranolol, in 10 mg capsules, or matching placebo. The initial dosage was one capsule twice daily; the maximal daily dosage was six capsules. Side-effect monitoring, blood pressure assessment, and adjustment of the dose of medication were carried out for several days into the treatment period. At the end of this period, side-effects and blood pressure were again assessed and akathisia ratings were carried out. Patients were then crossed over to the alternate medication, with treatment and assessment as in the first stage. Neuroleptics and anti-

TABLE I
Characteristics of patients

Age	Sex	Diagnoses	Medications	Maximum dose of propranolol
35	M	Schizophrenia	Perphenazine 32 mg/day Benztropine 4 mg/day	20 mg
35	M	Bipolar affective disorder, depressed; alcohol dependence; grand mal epilepsy	Perphenazine 56 mg/day Trihexyphenidyl 4 mg/day Diphenylhydantoin 400 mg/day Lithium carbonate 1800 mg/day	40 mg
32	F	Schizophrenia	Fluphenazine decanoate 62.5 mg every 10 days Fluphenazine 15 mg/day Trihexyphenidyl 6 mg/day	60 mg
44	M	Schizophrenia	Fluphenazine decanoate 200 mg every 7 days Fluphenazine 5 mg up to 6 hourly p.r.n. Trihexyphenidyl 8 mg/day Temazepam 30 mg node p.r.n.	60 mg
40	F	Schizophrenia	Fluphenazine decanoate 125 mg every 10 days Haloperidol 5 mg up to 3 hourly p.r.n. Trihexyphenidyl 8 mg/day	60 mg
23	M	Schizophrenia	Chlorpromazine 400 mg/day	60 mg
34	M	Schizophrenia	Fluphenazine decanoate 37.5 mg every 7 days Fluphenazine 5 mg daily p.r.n. Trihexyphenidyl 8 mg/day	40 mg
33	M	Schizophrenia; alcohol dependence	Perphenazine 112 mg/day Temazepam 30 mg node p.r.n.	20 mg
39	M	Schizophrenia	Fluphenazine decanoate 62.5 mg every 7 days Diphenhydramine 150 mg/day Trihexyphenidyl 15 mg/day	60 mg
43	F	Schizophrenia	Fluphenazine 30 mg/day Benztropine 6 mg/day Lorazepam 1 mg node p.r.n.	60 mg
29	M	Schizophrenia	Fluphenazine decanoate 25 mg every 7 days	60 mg
26	M	Schizophrenia	Perphenazine 52 mg/day Amantadine 400 mg/day	50 mg

parkinsonian medications were held constant throughout the trial. Both doctors and patients remained blind to the order of treatments.

Assessments of akathisia and anxiety were performed at baseline and on the last day of each treatment period. These consisted of: (1) the Simpson/Angus EPS scale (the objective rating); (2) a subjective rating in which the subject was asked to mark a 100-millimetre line corresponding to perceived restlessness; and (3) the Hamilton Anxiety Scale (HAS). All subjects gave informed consent.

Results

Twelve subjects participated; their ages, sex, neuroleptics, diagnoses, anti-parkinsonian medication and final dose of propranolol are shown in Table I. The mean number of capsules per day was 5.1 for propranolol and 5.3 for placebo. The final HAS rating during propranolol treatment was not obtained on one patient.

Six of the 12 subjects received placebo first; the remaining six were initially treated with propranolol. Baseline

TABLE II
Ratings at baseline and on last day of treatments

	Baseline	Mean (S.E.M.) Propranolol	Placebo
Objective* akathisia	1.75 (0.13)	0.75 (0.18)†	1.50 (0.23)‡
Subjective** akathisia	56.0 (5.0)	22.8 (5.0)††	45.3 (7.1)‡‡
Anxiety***	11.8 (1.6)	9.6 (2.0)	11.7 (2.0)

*Clinical ratings for pooled sample ($n=12$), statistical analyses by ANOVA for completely repeating measures with rating period and drug as repeated factors. *Post hoc* planned comparisons made by paired *t*-tests

*Overall: $F(2,22)=13.00$, $P<0.001$

†Propranolol vs baseline: $t(11)=5.74$, $P<0.0001$

‡Propranolol vs placebo: $t(11)=3.45$, $P<0.006$

‡‡Placebo vs baseline: $t(11)=1.15$, N.S.

**Overall: $F(2,22)=13.74$, $P<0.001$

††Propranolol vs baseline: $t(11)=5.87$, $P<0.0001$

‡‡Propranolol vs placebo: $t(11)=3.15$, $P<0.009$

‡‡‡Placebo vs baseline: $t(11)=1.64$, N.S.

***Overall: $F(2,21)=1.31$, N.S.

scores were similar in both of these groups; order of treatment did not significantly affect akathisia or anxiety ratings (Student's *t*-test (independent), all $t<1$). Therefore, statistical tests were performed on the pooled sample of 12 subjects (Table II).

Data were analysed by repeated measures analyses of variance (ANOVA), with rating period and drug (propranolol and placebo) as the repeated factors, to control for multiple, non-independent comparisons. *Post hoc* planned comparisons were performed by paired *t*-tests.

Propranolol caused a significant overall improvement in both objective and subjective ratings of akathisia. This effect was due to significant decrements in akathisia ratings on propranolol versus ratings done both at baseline and on placebo. Placebo caused no significant change of akathisia

ratings from baseline. No significant effect of either propranolol or placebo was seen in HAS. Statistical comparisons are shown in the legend to Table II.

Ten of the 12 patients were able to select the active medication, and indicated that they preferred it (one patient could not discriminate between the treatments and one believed that placebo was the active medication). This result was significant by the test for significance of a proportion ($Z=2.29$, $P<0.05$). There were no significant differences in the nine EPS measures other than akathisia after treatment with propranolol versus after placebo treatment. Mean arterial pressures during treatment with propranolol (97 ± 3 mm Hg) and with placebo (100 ± 3 mm Hg) did not significantly differ.

Discussion

The results of this double-blind, placebo-controlled, cross-over study confirm that propranolol in doses of 20–60 mg/day is an effective treatment of neuroleptic-induced akathisia. Efficacy was further suggested by the fact that most of the patients were able to select and indicated they preferred the active medication. Furthermore, six patients requested and have received propranolol as a maintenance medication, and have not since experienced a return of their akathisia. As in prior studies by Lipinski *et al* (1983, 1984), Zubenko *et al* (1984) and ourselves (Adler *et al*, 1985), propranolol in this dose range did not significantly affect mean arterial pressure.

Anxiety is often considered to be part of the akathisia syndrome. However, the fact that propranolol improved akathisia ratings, but did not affect anxiety ratings suggests that akathisia and anxiety may be dissociated.

Further investigations as to the central and peripheral effects of beta-blockers may clarify the role of dopaminergic and beta-adrenergic systems in neuroleptic-induced akathisia and perhaps in other conditions where motor restlessness is a problem.

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The Prediction of Abnormal Evoked Potentials in Schizophrenic Patients by Means of Symptom Pattern

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The case notes of 23 schizophrenic patients who participated in a study of somato-sensory cortical evoked responses were examined for descriptions of the clinical features of their mental state at the time of first illness or most recent relapse. A comparison of syndrome scores derived from Present State Examination with those derived from case notes showed that the latter was a reliable method of mental state description for the purposes of this study. A group of syndromes similar to that described in electrodermal research was identified from the case note review and found to be highly correlated with the presence of an abnormal response in the somato-sensory evoked potentials study.

In a recent paper, Gruzelier & Manchanda (1982) suggested that two syndromes of schizophrenia could be identified which correspond to two types of asymmetrical electrodermal activity following auditory orienting stimuli. They proposed that these two syndromes occur as a result of differential over- and under-activity of the two cerebral hemispheres, so that left hemisphere overactivity produces a florid, delusional and hypomanic clinical picture, while right hemisphere overactivity produces a slow, withdrawn and self-neglected clinical picture. We investigated whether these two syndromes could also be found in a group of schizophrenic patients in a study of somato-sensory cortical evoked potentials.

The design of the evoked potential study, and the methods and equipment used are described in greater detail elsewhere (Cooper *et al*, 1985). Briefly, the cortical potentials were evoked by a percussive stimulus to each index finger by means of a modified loud-speaker cone, recorded from the vertex and over each parietal lobe 2 cm posterior and 6 cm lateral to the vertex, and averaged over either 512 or 1024 stimulus presentations. All electrodes were referenced to linked ears, and the audible click from the stimulus apparatus was masked by popular music played through headphones for the duration of the experiment. The recording epoch was the first 100 ms post-stimulus, and in each case it was demonstrated that the order of stimulus presentation had no effect on the potentials recorded, and that the auditory mask was effective.

The expected pattern of evoked responses from such a stimulus in the first 100 ms is that the parietal cortex contralateral to the stimulated finger is activated, and that little, if any, activity is recorded over the ipsilateral cortex. This expected lateralisation

of response was lost in about half of the 21 schizophrenic patients reported in our previous study (Cooper *et al*, 1985), but was not lost in either the manic depressive patients or the normal volunteer comparison group; this suggested that those schizophrenics with the clearly abnormal response may be different in some clinically relevant aspect of their illness. The earlier paper reported a trend associating the abnormal response with a poorer clinical outcome. In this paper, we report the results of testing the hypothesis that the syndromes derived from the Present State Examination (PSE) (Wing *et al*, 1974) which characterise the right hemisphere-overactive syndrome described by Gruzelier & Manchanda (1982) are associated with the abnormal evoked potential response. The reasons for deciding to test this association were firstly that those right hemisphere-overactive syndromes (slowness, self-neglect and withdrawal) are likely to be correlated with the poorer social outcome that had already been noted; and secondly, if two different electrophysiological measurements can be shown to correlate with a clinically salient distinction between two presentations of schizophrenia, then they might be produced by the same underlying physiological abnormality. Such a straightforward correlation between two different measures would have both diagnostic utility and a number of research implications.

Method

Subjects

The patients described are 23 out of 24 with a clear and unequivocal diagnosis of schizophrenia who participated in an on-going study of somato-sensory cortical evoked

TABLE I

Ages and hospital admissions for patients in the SSEP study

	Male	Female
Average age	35.5	31
Range	55-25	48-21
Average age when first taken ill	26	26
Range	53-18	44-17
Average number of admissions*	4.6	4.1
Average total months in hospital†	11.4	6.4

*Excluding one male with 17 brief admissions.

†Excluding rehabilitation admissions of more than one year (one male & one female).

potentials (SSEPs). Our findings in the first 21 patients studied are reported elsewhere (Cooper *et al.*, 1985). The 24th patient lacked sufficiently detailed clinical documentation and was excluded for that reason.

The group comprised 12 men and 11 women, whose ages and hospital admissions are shown in Table I; the men were older at the time of recording and had spent more time in hospital. Two patients were adopted male monozygotic twins, and two were father and son. Of the remaining 19, four had a history of severe mental illness in a first-degree relative: schizophrenia in the case of two, manic depression in another, and unspecified psychosis in another.

In this group, 16 patients had the abnormal response (either bilaterally or unilaterally symmetrical) and seven had the normal response (bilaterally asymmetrical). The types of response are reported and discussed elsewhere (Cooper *et al.*, 1985).

Experimental design

In outline the study had four stages: firstly, two clinical syndrome categories were outlined, and category scores for

each patient were obtained from case note ratings; secondly, the case note ratings were compared for reliability with separately obtained PSE ratings in some of the patients; thirdly, the category scores were used as the basis for a blind prediction of abnormality of SSEPs; and fourthly, an association between certain clinical syndromes and SSEP abnormality was sought. These stages are outlined in greater detail below.

Derivation of the syndrome ratings from the case notes: The items and factors described by Gruzelić (1983) were translated into the nearest equivalent PSE syndromes of which they form a whole or part. This essentially involved using the terminology of PSE syndromes to describe the clinical phenomena under consideration, in a similar manner to that used in the derivation of the Syndrome Check List (Wing *et al.*, 1974). The syndromes were grouped to produce two 'categories', approximating to Gruzelić's left hemisphere-overactive syndrome (Category 1) and the right hemisphere-overactive syndrome (Category 2). These categories are designated 'active' and 'withdrawn' respectively, for the purpose of further discussion (Table II).

The psychiatric case notes of all 23 patients were examined by one of us (AOH, an experienced and trained PSE interviewer), and the presence or absence of these PSE syndromes in a substantial part of the index illness was recorded, as in the Syndrome Check List. The rating of the presence or absence of each syndrome in each patient was undertaken blind to the SSEP results, and blind to the PSE-derived syndrome scoring.

Reliability of the case note-derived ratings compared to PSE interview-derived ratings: Nine of the 23 patients had PSE interview-derived data for the index illness, made for other reasons, the percentage agreement between the ratings from the two sources was calculated as a way of assessing the reliability of the case note ratings. The PSE interviews and ratings had been undertaken before the present study was started and were therefore blind to the purpose of this study and to the SSEP findings.

TABLE II
PSE Syndrome list used in case note review of 23 schizophrenic patients

Category 1	PSE mnemonic	Category 2	PSE mnemonic
Hypochondriasis	HY	Neglect	NG
Depressive delusions	DD	Affective flattening	AF
Grandiose delusions	GR	Slowing	SL
Hypomania	HM	Lack of energy	LE
Overactivity	OV	Social unease	SU
Ideas of reference	IR	Simple depression	SD
Situational anxiety	SA	Specific features of depression	ED

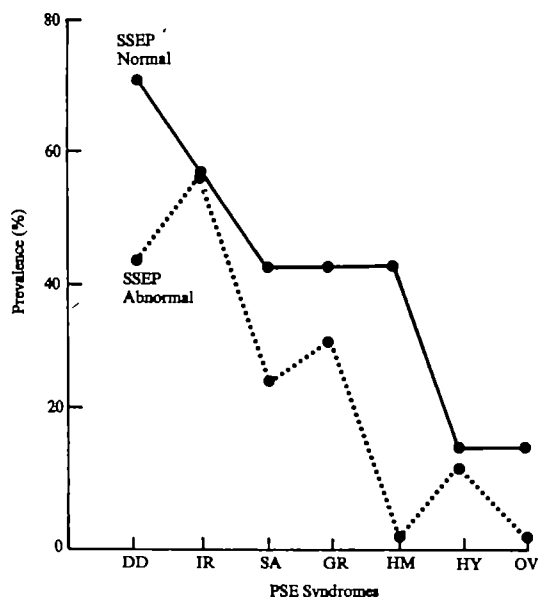


FIG. 1 The percentage prevalence of "active" syndromes in schizophrenic patients with normal and abnormal SSEPs. For abbreviations see Table II.

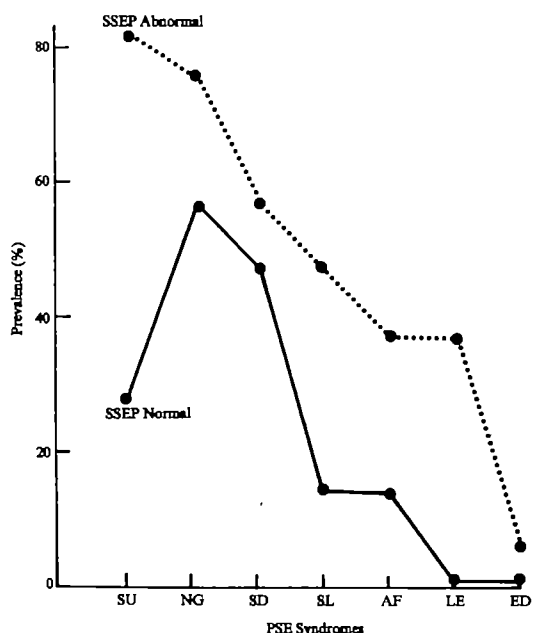


FIG. 2 The percentage prevalence of 'withdrawn' syndromes in the two groups. For abbreviations see Table II.

Prediction of the type of response in the individual patient: Each syndrome was scored '1' or '0', depending on its presence or absence in the case note record, and these amounts were added up to give the number of syndromes present in the two categories for each patient. Where there were more 'withdrawn' category syndromes present than 'active' category syndromes, an abnormal response was predicted. The predictions were made by AOH, blind to the actual SSEP results.

Correlation between SSEP-type and the syndrome category scores: The percentage prevalence of each syndrome in the two categories for both the normal and the abnormal responders was estimated (Figs 1 and 2). Differences were calculated between the patient groups (abnormal responders $n=16$, normal responders $n=7$) in terms of their total category scores, and between the percentage prevalence of the individual syndromes across the two groups.

Results

Comparison of the case note and PSE interview ratings

There was less than 70% agreement on only four syndromes, self-neglect and ideas of reference were more commonly rated as present from the case notes; lethargy and poor concentration were more commonly rated as present in PSE interviews.

The discrepancies are not large and may be easily explained. For instance, self-neglect is more likely to be recorded in case notes than rated as present during the PSE interviews because of the more prolonged period of observation involved in the former; ideas of reference, which are largely situation-dependent and evanescent, may be present in the ward environment but not in the interview room; lethargy and loss of concentration are symptoms elicited at PSE interviews, often with no expression in behaviour that may be observed in the ward, and hence are likely to be under-recorded in case notes.

The prediction of SSEP abnormality

In two patients, no prediction was made because the scores in each category were the same. The abnormalities were predicted correctly in 19 of the 21 cases in which a definite prediction could be made (Fisher's exact test two-tailed, $P=0.002$; Table III).

SSEP type and syndrome category scores

Patients with abnormal SSEPs had significantly different 'active' and 'withdrawn' category scores (Wilcoxon matched-pairs test, $t=3$, two-tailed, $P<0.05$). They also had significantly higher 'withdrawn' category scores (Wilcoxon matched-pairs test, $t=3$, two-tailed, $P<0.01$). Abnormal responders were more likely to have three or more 'withdrawn' syndromes each, while normal responders were more likely to have less than three (Fisher's exact test, two-tailed, $P<0.05$). The syndrome

TABLE III

Comparison of actual and predicted results of somatosensory evoked potentials (SSEP) in 23 schizophrenic patients

Actual results of SSEPs	Predicted results of SSEPs from category scores		
	Abnormal	Normal	Equivocal
Abnormal	14	0	2
Normal	2	5	0
Equivocal	0	0	0
Total	16	5	2

'social unease' (SU) and the syndrome 'slowness' (SL) were significantly more common in the patients with abnormal SSEPs (Fisher's exact test, two-tailed, $P < 0.05$ in both cases).

Discussion

Although the number of patients in this study is small, it has been possible to demonstrate a correlation between a physiological abnormality found in some schizophrenic patients and certain features of their mental state. The features which we have found to be relevant show similarities with those highlighted in the 'right hemisphere-overactive' syndrome of Gruzelier & Manchanda (1982). Any discrepancies may be explained by differences in the selection of patients and in the instruments used for clinical description. The main difference between our syndrome groupings and those of Gruzelier & Manchanda appears to lie in the lack of discriminating power of the depressive syndromes (Figs 1 and 2). As clinical practice suggests that PSE depressive syndromes may represent a non-specific dysphoria, associated with many varieties of major mental illness, it is not surprising that they cannot be readily associated with electrophysiological abnormality.

A corresponding ability to identify a syndrome grouping equivalent to Gruzelier's left hemisphere-overactive has not been demonstrated—perhaps because the 'active' and hypochondriacal syndromes are rare in the evoked potential group, while ideas of reference, depressed mood and anxiety are too widespread to be of discriminating value.

The syndromes associated with the evoked potential abnormality in this study are closely related to the descriptions of the primary symptoms of schizophrenia given by Bleuler (1911), and it is of interest that Gruzelier refers to the distinction pro-

posed by Kety (1980) between 'classical Bleulerian schizophrenia' and 'acute functional psychosis', when discussing the possible clinical relevance of his electrodermal study. The 'withdrawn' category also bears comparison with the features of 'true' (poor prognosis) schizophrenia, as suggested by Langfeldt (1960): blunting, lack of initiative, altered behaviour and motor disorder.

Although the 'withdrawn' syndromes are generally considered part of the clinical poverty syndrome, and therefore indicators of poor outcome, their intensity can vary considerably (Hirsch *et al*, 1973). The evoked potential abnormality bears no relation to the presence or absence of these or any other syndromes at the time of recording; it is their presence characterising the patients' mental state when ill which gives the 'withdrawn syndromes' their predictive value. Indeed, since the evoked potential abnormality is stable over time and mental state is not, it could not be otherwise. If confirmed in other studies, this suggests that the SSEP abnormality is an indication of a persisting vulnerability, which disposes towards a certain sort of mental state when ill, and towards a certain kind of outcome.

As the correlation between syndromes and physiological abnormality which was found in this study is similar to those found following investigation of different physiological parameters, it is reasonable to speculate that the physiological abnormalities are themselves related. The accumulating evidence for intra- and interhemispheric disorganisation, and for abnormalities of cortical arousal has been reviewed recently (Walker & McGuire, 1982; Gruzelier, 1983; Flor-Henry, 1983). The difficulty in discussing the relationship between hemispheric arousal, hemispheric disorganisation and early SSEP abnormalities rests principally on the uncertainty as to which cortical or subcortical disorder might produce them. However, a brief discussion of general cortical-subcortical relationships is worthwhile.

The sensory volley from the stimulus apparatus used in the SSEP recording is likely to be produced by muscle spindle activity as a result of the percussion from the cone of the loudspeaker (Pratt *et al*, 1979). The earlier of the two peaks we identified is generally considered to be the arrival of the first thalamic component at the primary receiving area (Allison *et al*, 1980). By 80 ms post-stimulus, the activity at the two parietal recording sites tends to equality; there is no suggestion of a persisting abnormality of cortical activation at those sites after the second peak which we identify at 50 ms has passed. The relevance of this early cortical abnormality to any later cortical abnormalities that may reflect handling of higher

order sensory information is uncertain. However, the fact that there is a bilateral component, one to each receiving area from the thalamus, suggests some disorder of thalamo-cortical interaction where the cortex ipsilateral to the stimulated finger is activated by the thalamus, as well as the more normal contralateral activation. The mutual inhibition of the hemispheres by each other is a well established feature of the large-scale control of cortical activity (Geschwind, 1965), and it is not unreasonable to suppose that hemispheres which are abnormally bilaterally activated by somato-sensory thalamic volleys continue to inhibit each other in the usual way. The early abnormalities described here may be taken to be produced by some disorder of thalamo-cortical interaction, which may, in its turn, produce the overactive hemispheres that are described in electrodermal and dichotic listening studies.

The reasons for considering that abnormalities in visual cortical evoked potentials may be produced by abnormal cortico-thalamic activity are discussed by Jutai *et al* (1984). This and the finding of a loss of the normal lateralisation of auditory evoked potentials in a proportion of schizophrenics reported by Connolly *et al* (1985), taken together with the results reported here, suggest that the explanation of the difference between normal and schizophrenic evoked potentials across the sensory modalities may be due to the presence of a general disorder of cortical—sub-cortical relationship, rather than of a disorder specific to any one sensory modality. The possibility that such abnormal relationships may be of relevance in schizophrenia merits further investigation.

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The Formation of Maintenance of Delusions: A Bayesian Analysis

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This paper argues that recent research on normal-belief formation is relevant to our understanding of the establishment and maintenance of delusions. Bayesian theory provides a normative model of the way in which evidence relevant to normal beliefs may be evaluated: this makes it possible to classify delusional beliefs in terms of deviations from optimal Bayesian inference. Some hypothetical forms of deviation appear to correspond closely to cognitive processes observed in some groups of deluded patients. Theories of the precise nature of the abnormal judgemental processes also have implications for psychological approaches to treatment of deluded patients. The role of hallucinations in the formation and/or maintenance of delusions and the extent to which the distortions of cognitive processes associated with delusions are content-specific or mood-specific are also considered.

This article aims to indicate ways in which a recent paper on the formation of *normal* beliefs (Fischhoff & Beyth-Marom, 1983) may aid our understanding of the establishment and maintenance of *abnormal* belief systems (delusions). We also argue that a consideration of such literature suggests a number of experiments to clarify the nature of abnormal beliefs.

Although, in a general sense, delusions may be viewed as abnormal beliefs, the definition of "delusion" continues to present problems. Mullen (1979) has suggested that abnormal beliefs have the following characteristics:

- (i) They are held with absolute conviction
- (ii) They are experienced as self-evident truths, usually of great personal significance
- (iii) They are not amenable to reason, or modifiable by experience
- (iv) Their content is often fantastic or at best inherently unlikely
- (v) The beliefs are not shared by those of a common social or cultural background (Mullen, 1979, p. 36).

The difficulties inherent in such standard definitions have been examined by Moor & Tucker (1979). Even individually the criteria present problems; in conjunction, the definition becomes further confused. We hope to clarify the issues involved by an examination of the various ways in which the processing of information by deluded subjects may deviate from normality.

It appears to be essential to distinguish between the

establishment and the maintenance of belief systems. Establishment may result from abnormal perceptual experiences, such as previously ignored and/or preconscious information becoming salient (Frith, 1979). Authors have differed in their assessments of the role of anomalous experiences in the formation of delusions. Some, such as Maher & Ross (1984), have argued that the processes whereby the beliefs are formed are normal. In their theory, a normally-formed belief corresponds to a Jasperian "secondary delusion" (Jaspers, 1913) which is explicable in terms of the patient's background, personality or experiences: it is distinguished from a "primary delusion" which is not understandable and represents a disturbance of symbolic processing or attribution. Winters & Neale (1983) note that this distinction between explicable and inexplicable beliefs is not generally accepted or reliable, and question its validity as a diagnostic marker.

It may also be argued that normal processes are involved in the *maintenance* of abnormal beliefs, it being apparent that even "normal" strongly held beliefs are highly resistant to change. Here one is forced to confront the question of whether the statement "strong beliefs are resistant to change" is a tautology. The problem obviously lies in the definition of "strong belief", which involves both resistance to change and certainty of belief. In addition, resistance to change may be specified either in terms of the likelihood of spontaneous alteration of the belief, or in terms of the effects of confrontation with evidence inconsistent with the belief. Clearly we need to examine the relationship between resistance to change and initial intensity of belief, and to

ascertain whether this is different for abnormal and normal beliefs. If the relationship were identical, this would support the view that normal processes may be involved in the maintenance of delusions: it would then be important to examine how, when and why strong beliefs are formed, rather than why they are difficult to change.

The alternative position is that the processes which maintain delusions are abnormal. Cameron (1951) has suggested that deluded individuals disregard information incongruent with their delusional systems. However, as a paper by Alloy & Tabachnik (1984) indicates, similar processes are operative in the maintenance of *normal* strongly held beliefs. Alloy & Tabachnik consider organisms' ability to detect the relationships or covariations among stimuli, behaviour and outcomes in the environment: information about such covariation provides a means of explaining the past, controlling the present, and predicting the future. They propose that this "covariation perception" is determined by the interaction between two sources of information: (a) the organism's prior expectations about the covariation between two events; and (b) current information provided by the environment about the objective contingency between events. The relative impact of each factor is dependent on the strength of the other: the same level of current information regarding covariation of events has less influence on an organism's perception of contingency when the organism has strong prior expectations about the relationship between the events. Thus if deluded subjects take little account of information incongruent with the delusional system, this may simply reflect strong prior expectations.

A normative theory of how people *should* evaluate evidence relevant to their beliefs can provide a conceptual framework for a consideration of how they do *in fact* evaluate it. Fischhoff & Beyth-Marom (1983) argue that Bayesian inference provides a general framework for evaluating hypotheses (beliefs) and that it is possible to describe a person's consistency with, or departures from, the theoretical model. They identify a set of logically possible deviations from Bayesian behaviour and review the research literature to see whether these possibilities correspond to observed behaviour in the normal population. It is argued below that this approach may usefully be applied to understanding the cognitive distortions shown by patients regarded as deluded.

A Bayesian Analysis of Delusional Beliefs

From a Bayesian perspective, knowledge is represented in terms of statements or hypotheses, H_i , each

of which is characterized by a subjective probability, $P(H_i)$, representing one's confidence in its truth. Bayes's theorem then governs the way in which the strength of one's belief in a hypothesis should be revised in the light of new information or data (D). In its simplest form, the theorem deals with the implications of the datum (D) for the relative likelihoods of a hypothesis (H) and its complement ($\bar{H}$). In such cases Bayes's theorem states that:

$$\frac{P(H/D)}{P(\bar{H}/D)} = \frac{P(D/H)}{P(D/\bar{H})} \cdot \frac{P(H)}{P(\bar{H})}$$

Reading from right to left, the three terms in the formula are: (i) the *prior odds* that H, and not $\bar{H}$, is true in the light of all that is known before the receipt of D; (ii) the *likelihood ratio*, representing the information value of D with respect to the truth of H; (iii) the *posterior odds* that H is true in the light of all that is known after the receipt of D. This approach has the advantage, for our purposes, of incorporating the level of prior belief explicitly in the inferential process. Fischhoff and Beyth-Marom point out that Bayesian inference is a normative scheme and that it is logically possible to deviate from optimal Bayesian performance in a number of ways. This provides us with a scheme for categorising different types of judgemental processes and considering whether a particular form of deviation is characteristic of a given group of people (e.g. deluded subjects). In the clinical setting, a consideration of the precise nature of the abnormal judgemental processes may have implications for attempts at modification of abnormal beliefs, such as those reported by Milton *et al* (1978).

Fischhoff & Beyth-Marom suggest a number of potential biases that can occur in hypothesis evaluation. These may occur at the stages of (a) hypothesis formulation (b) assessing component probabilities (c) assessing prior odds (d) assessing the likelihood ratio (e) "aggregation" (f) information search, or (g) action. For a given belief (hypothesis), a deluded subject may demonstrate deviation from the Bayesian model at one or more of the above stages.

(a) Hypothesis formulation

Fischhoff & Beyth-Marom indicate two possible sources of difficulty relevant to our discussion: failure to formulate a hypothesis at all, or formulation of an untestable hypothesis. They argue that for normal subjects the failure to formulate hypotheses is rare, but this may not apply to the transient beliefs of those traditionally described as hebephrenic schizophrenics: such patients appear at times to translate

abnormal experiences directly into belief statements, with no intervening stage of considering evidence which might be relevant to the related hypothesis.

Patients whose beliefs involve untestable hypotheses are frequently encountered in clinical practice. Perhaps the most common of such beliefs are those concerning knowledge of future events, e.g. the certain belief that the world will end in the year 2093 A.D.

(b) *Assessing component probabilities*

Beliefs must be translated into subjective probabilities, and the assessment of component probabilities is the second stage at which deviations from the model may occur. One such deviation would be the violation of the probability axioms, for example $PH + PH = 1$. Thus a subject might express 100% confidence that the IRA were plotting against him, but also a one-in-five chance that no such plot existed. Alternatively, poor assessment of component probabilities may result from miscalibration, i.e. the failure of one's confidence to correspond to reality. This might be viewed as an essential characteristic of delusional beliefs: however, normal subjects are frequently over-confident in their probability estimates (Lichtenstein *et al* 1982), even to the extent of frequent expression of absolute certainty (Fischhoff *et al*, 1977). The special peculiarity of deluded subjects is not the absolute certainty which is often apparent but rather the improbability of the events about which they express such beliefs.

A further well-known form of deviation at this stage is an over-reliance on the availability and representativeness of the data used as a guide to probability estimation (Kahneman *et al*, 1982). Subjects judge an event as likely to the extent that exemplars are easy to recall or imagine, and thus unusually salient events may be judged to be more frequent than is actually the case. This process appeared to occur in a young paranoid patient treated by one of the authors. He was preoccupied with his appearance and as a result felt that 80% of the people in the street were laughing at him. Accompanying him for a walk and counting those people who were smiling, not necessarily at him, produced a score (agreed with the therapist) of about 15%.

(c) *Assessing prior odds*

The assessment of prior odds represents the calculation of the ratio of the probabilities of competing hypotheses. Difficulties at this stage are largely

dependent on the earlier stages of hypothesis formulation and assessment of component probabilities. However, there are problems specific to this stage, e.g. the neglect of one of the hypotheses. When a person acts as though a hypothesis that is probably true is absolutely true, they have effectively neglected its complement. This aberration appears to be relevant in the case of certain delusions where the question of the probability of $\bar{H}$ is totally ignored: deluded patients frequently tell interviewers that they have never considered the possibility of the falsity of their beliefs.

(d) *Assessing the likelihood ratio*

This involves consideration of the information value of a datum with respect to H and $\bar{H}$. One possible error here, failure to consider $P(D/\bar{H})$, appears to be common in deluded patients. For example, one patient took the appearance of a police car in a busy thoroughfare as unequivocal evidence that the police were chasing him, neglecting the probability of this event occurring if the police had no interest in him. Similarly, the patient described above who erroneously estimated the probability of people smiling in the street had failed to consider alternative possible reasons for their behaviour.

It is also at this stage that the interaction of prior beliefs with new information occurs (Alloy & Tabachnik, 1984). Fischhoff & Beyth-Marom note that "the interpretation of new evidence may be affected by previous beliefs, thereby subverting the independence of the likelihood ratio and priors" (p. 247). Thus people can interpret and reinterpret new information so as to render it consistent with their beliefs. This type of error represents a less severe deviation from the normative model than does total failure to consider an alternative hypothesis: when such reinterpretation is encountered in deluded subjects, particularly if they acknowledge that the new (apparently inconsistent) data must be considered seriously, it seems that the abnormal belief system may be less resistant to change (Brett-Jones, 1984).

(e) *Aggregation errors*

Although Fischhoff & Beyth-Marom describe the possibility of an error at the aggregation stage of the model, i.e. in the computation of the product of prior odds and likelihood ratio, they present no common examples of such an error. Nor is it clear what role such an error might play in the maintenance of abnormal beliefs.

(f) Information search

Information search, the sixth stage, does appear relevant to our enquiry. Some deluded subjects report no information search relevant to their belief at all. For example, one patient had experienced a "delusional perception" at the stage of formation of his belief. A "delusional perception" is a primary disturbance of symbolic meaning, with no disturbance of perception, sensation or thinking. The basic feature is "the establishment of an unfounded reference" (Gruhle, 1915). The belief was that he was in direct contact with, and could influence, a famous athlete. He described how he had no need for any confirming or disconfirming evidence, so certain had the initial experience rendered him. In the authors' experience, many deluded subjects fail to search for information relevant to their hypotheses, since their conviction renders this, in their eyes, unnecessary. Other patients do search for information, but restrict the search to confirmatory evidence.

(g) Action

Action is the seventh and final stage of the Bayesian model. The output of the process of Bayesian inference, i.e. the posterior odds, summarises the subject's beliefs in a way that facilitates selecting the optimum course of action. However, it is clear that the relationship between belief and action is far from simple, even for normal subjects. Fischhoff and Beyth-Marom note that "believing H" is not equivalent to acting as though H is true, and suggest that we may in general be more cautious in our actions than in our expressions of belief. There is a considerable literature on the subject of an individual's willingness to stake money on his/her beliefs, and generally it is found that subjects are indeed more cautious in their actions than in their statements (Fischhoff *et al.*, 1977). With deluded subjects, one frequently striking feature is their remarkable lack of action congruent with apparently sincerely held beliefs.

Non-Bayesian Considerations

A number of other points, not covered by Bayesian analysis, are worthy of consideration here, before discussing experimental approaches suggested by the theoretical models. These concern the role of hallucinations in the formation and maintenance of delusions, and the question of generalised distortion *vs* content-specific or mood-specific distortion or processing.

The role of hallucinations

Hallucinations often appear to play a special role in the formation and/or maintenance of delusions. At the stage of hypothesis formation, hallucinations are data, often distressing and uncontrollable, for which an explanation must be found. Most individuals infer, understandably, that their experiences are the result of the activities of real objects or beings; and when, for example, the source of an auditory experience cannot be detected by the other senses, infer the existence of some other source for the sounds. Thus far, the processes appear "normal", although the resultant hypothesis may be fantastical. What appears to be abnormal is extent to which the belief is then maintained in the face of social rejection of its truth and the provision of an alternative hypothesis—that the individual is ill. Is it the special salience of the data—its ego-involving content and striking, often persistent, availability—which enables the formation and maintenance of this idiosyncratic explanation?

Content specificity

Is the distortion of processing in the deluded individual a generalised distortion of all judgemental processes, is it content-specific, or is it mood-specific? Cognitive deficits have been widely studied—e.g. in schizophrenia—but there has been relatively little attention paid to this particular question.

Winters & Neale (1983), in a review of theories of delusions, describe a number of "deficit" models, which invoke some dysfunction of cognitive processing (such as deficits in perceptual processing) to account for the formation of a delusional belief. However, the precise roles of specific cognitive deficits in the formation of delusions have not been experimentally identified or tested.

Arieti (1974) proposed that schizophrenic individuals form delusions through a failure of syllogistic reasoning, which is more generalised across content in some diagnostic sub-groups than in others. However, an experimental study of this hypothesis reported by Chapman & Chapman (1973) produced inconclusive results, and current reviewers such as Winters & Neale (1983) find little support for this view.

Robertson & Taylor (1985), in a study of 61 convicted schizophrenics and 41 "normal" prisoners, found that on tests of cognitive functioning the "deluded" sub-group of schizophrenics were closer to the "normals" than were the "mixed symptom" sub-group or the "simple schizophrenic" sub-group. However, the "deluded" group performed poorly

on two tasks of "what might be called immediate memory" (p. 95). The authors speculated that it may be that such people are liable to misinterpret external stimuli. This hypothesis is of the "deficit" type and implies a generalised distortion of judgemental processes.

Brennan & Hemsley (1984) on the other hand, in a study of illusory correlations (i.e. reported correlations of two events which in reality are not correlated) found a content-specific effect. On a task which involved stimuli particularly relevant to patients with paranoid delusions, a paranoid schizophrenic group reported particularly strong illusory correlations compared with a non-paranoid schizophrenic group and a "normal" group. A fuller investigation of the content-specificity of deluded subjects' distorted processing is overdue. Within experimental psychology, information-processing studies have recently been criticised for neglecting the role of content in tasks (Claxton, 1980) and for an over-reliance on neutral material.

Mood specificity

Although there is considerable literature concerning cognitive processing in depression, little if any experimental work has been published which involves *deluded* depressed patients. Do deluded depressed or manic patients exhibit generalised distortions of processing of material unrelated to their mood state? Can distortions of processing be detected in asymptomatic patients?

It is reasonable to speculate that these distortions are mood-specific: in depressed or euphoric states, the individual may be guided more by affective than by cognitive processing (Zajonc, 1980; Bower, 1981). Such is its salience that the experience of the mood state may take precedence over other types of data ("I feel (very strongly) like this, therefore I am this"), or may in some way influence inferences and information search: thus the manic patient, feeling excited, requires no further evidence to support his identification of his current feelings with an existential view, and may come to hold a grandiose self-perception regardless of other data. This hypothesis bears some similarity to Maher & Ross's (1984) view of delusions as explanations of anomalous experiences.

Experimental Approaches

This review of the literature suggests a number of experiments to clarify the processes of formation and maintenance of delusions. To test for generalised

or content-specific "distortion", it is possible to compare deluded and control subjects' performance on a "neutral" task of hypothesis formation. Volans (1976) reported an experiment involving the formation of hypotheses about the probability of a coloured ball being drawn from one of two hidden bags containing different proportions of two sets of coloured balls. This experiment allows for an examination of (i) the strength of the initial hypothesis, (ii) the number of trials taken to reach certainty, and (iii) the role of confirming and disconfirming evidence. Obsessional subjects were found by Volans to differ from controls on this task: it remains to be seen whether deluded subjects would differ in judgemental style, and thus show evidence of generalised distortion.

Given a Bayesian analysis, it is possible to devise experiments to test for deviations from the normative model; and a wealth of carefully designed experiments, used with normal subjects by Fischhoff and colleagues, is already available. In interpreting the results of any such experiment, however, "normal" deviation from the prescriptive model must not be forgotten: deluded subjects must be shown to differ quantitatively or qualitatively from "normal" controls, rather than simply deviating from the model itself, if claims of abnormality are to be justified.

Conclusions

The clinical relevance of the experimental approaches suggested resides in their potential for improving both the system of classification and the methods of modification of delusions. This systematic form of assessment of delusions (involving consideration of judgemental processes, content and mood) might aid the valid identification of subgroups predictive of such factors as change and aetiology. For those delusions untreated by current methods, a careful individual analysis of the reasoning process might identify the point in the process at which attempts at modification might be most effectively. (The patient described above, who was concerned about people smiling at him, was helped by treatment which successfully focused on a re-assessment of the frequency of people smiling in the street). Rachman (1983) describes a number of ways in which the findings of this branch of cognitive psychology may further our understanding of and therapy for abnormal beliefs.

In this paper, we have attempted to draw attention to current work in the field of normal psychology which has relevance to the study of delusions. This work has implications for certain basic questions

concerning the nature of the deluded individual's dysfunction, and points to new approaches in assessment of individual cases and the experimental analysis of delusions. Such approaches, it is hoped, will open the way to new possibilities in the psychological treatment of deluded individuals.

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Abnormal Illness Behaviour and Anxiety in Acute Non-Organic Abdominal Pain

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Of 105 patients admitted to a surgical ward with acute abdominal pain, 18 were considered to be without evidence of any organic aetiology for this symptom. These 'non-organic' patients were almost all female and differed from the 'organic' patients in state anxiety and in the illness behaviour questionnaire scales of psychological versus somatic perception, denial and affective disturbance. The two groups of patients could be differentiated on the basis of scores derived from the Illness Behaviour Questionnaire, and the State-Trait Anxiety Inventory.

In a considerable proportion of patients admitted acutely to a surgical ward with abdominal pain, no specific organic pathology is found. Some patients are observed and discharged while others undergo various medical or surgical procedures, most commonly appendicectomy. Various studies show that in 10-30% of patients undergoing this operation, no organic pathology is found (Jess *et al*, 1981; Boerema *et al*, 1981; Chang, 1981). Young women appear to be especially at risk of having a normal appendix removed (Lee, 1961). Comparison of life events and psychiatric disorder in patients complaining of abdominal pain, with and without histologically confirmed appendicitis, found an excess of both psychiatric symptoms (mainly depression) and of adverse life events in those whose appendix was not acutely inflamed. Creed (1981) suggested, as have others (Ingram *et al*, 1965; Meyer *et al*, 1964), that psychosocial factors are major contributors to the problem of appendicectomy in which the appendix is not acutely inflamed.

We hypothesised that depression, anxiety or some form of abnormal illness behaviour were the likely psychiatric reasons for admission to hospital with acute abdominal pain when no organic pathology was found. We thus set out to determine whether patients without an organic basis for their abdominal pain would differ from those with an organic basis for their pain on measures of depression, anxiety and abnormal illness behaviour.

Method

On the day following admission to a surgical ward at Christchurch Hospital, all patients with a major presenting complaint of abdominal pain between ages 15 and 60 years were approached and asked to participate in this study.

If patients agreed they were asked to complete the State-Trait Anxiety Inventory (STAI) (Spielberger *et al*, 1969), the Zung Self-rating Depression Scale (SDS) (Zung, 1965), and Pilowsky's Illness Behaviour Questionnaire (IBQ; Pilowsky *et al*, 1979).

After a minimum of six months (time to allow for any possible review of diagnosis) and without reference to the psychological test scores, the surgical notes were reviewed and the diagnosis recorded. If this was appendicitis, it was checked that it had been histologically confirmed. If non-specific abdominal pain was the diagnosis, then the notes were searched for any evidence of physical abnormalities such as an elevated temperature, white blood count or erythrocyte sedimentation rate, which suggested an organic aetiology.

Patients were grouped into four diagnostic categories: those with appendicitis, those with other organic diagnoses, those with non-specific abdominal pain with physical abnormalities, and those with non-specific abdominal pain without physical abnormalities. The mean (and standard deviation) for trait anxiety (range 20-80), state anxiety (range 20-80), Zung depression (range 20-80), and the seven scale scores of the IBQ were calculated for each diagnostic grouping. Categorical data were compared using a chi square test, while continuous variables were compared using Student's *t*-test. Using the data for female patients only, a stepwise discriminant analysis (SPSS-II) was performed on the 40 questions in the IBQ which contribute to its seven scales.

Results

During the course of this study 121 patients were admitted acutely to surgical wards with abdominal pain. Twelve were not approached (poor comprehension of English, staff members or too sick) and four declined to participate after having seen the questionnaires. The remaining 105 patients are the subjects of this study, and the most common surgical diagnoses were: appendicitis ($n=33$), non-specific abdominal pain (37), of whom 19 had and 18 did not have

TABLE I
Psychological measures in abdominal pain patients by diagnosis

	Organic cause			Non-organic	
	Appendicitis	Other organic cause	Non-specific (with abnormalities)	Total	Non-specific (without abnormalities)
Number	33	35	19	87	18
Age	29.5(±11.7)	37.5(±15.2)	26.9(±11.2)	32.2(±13.7)	30.6(±14.6)
M/F	19/14	17/18	7/12	43/44	1/17
Trait anxiety	37.7(±8.4)	37.1(±9.8)	35.9(±6.1)	36.6(±9.4)	39.1(±9.1)
State anxiety	37.6(±11.7)	40.2(±13.3)	37.7(±11.5)	38.1(±13.1)	48.1(±9.2)
Zung depression	35.4(±10.1)	38.1(±10.7)	36.2(±8.6)	36.1(±10.7)	39.0(±8.4)
<i>IBQ</i>					
General hypochondriasis	1.52(±1.73)	1.56(±1.48)	1.26(±1.48)	1.47(±1.57)	1.39(±1.04)
Disease conviction	1.88(±1.63)	2.46(±1.46)	2.26(±1.59)	2.20(±1.56)	2.61(±1.38)
Somatic vs Psychological	1.61(±0.61)	1.60(±0.96)	1.53(±0.70)	1.59(±0.78)	1.00(±0.69)
Affective inhibition	2.27(±1.35)	2.25(±1.64)	2.16(±1.07)	2.24(±1.41)	2.44(±1.69)
Affective disturbance	1.31(±1.47)	2.05(±1.75)	1.16(±1.38)	1.51(±1.61)	2.61(±1.38)
Denial	3.39(±0.97)	3.01(±1.58)	3.26(±1.76)	3.22(±1.42)	4.06(±1.39)
Irritability	2.06(±1.73)	1.95(±1.79)	1.89(±1.63)	1.97(±1.72)	2.11(±1.41)
<i>Duration of pain (days)</i>					
<7	31	25	16	72	15
7-30	2	6	2	10	1
>30	0	4	1	5	2
Median	2	2	1	2	3
					NS

Results expressed as mean (±s.d.)
Statistics compare 'organic' with 'non-organic' groups

TABLE II

Questions from the IBQ (plus answer to score) with unstandardised canonical discriminant function coefficients which produce the illness behaviour discriminant score

<i>Question from IBQ (Answer)</i>	<i>Coefficient</i>
1. Is your bad health the biggest difficulty of your life? (No)	-2.057118
2. Are you more irritable towards other people? (Yes)	-1.937602
3. Do you often think that you might suddenly fall ill? (Yes)	1.729845
4. Is it easy for you to let people know when you are cross with them? (No)	1.353237
5. Do you ever have silly thoughts about your health which you cannot get out of your mind, no matter how hard you try? (Yes)	-1.199663
6. Do you find that you get sad easily (Yes)	-1.156323
7. Do you think there is something seriously wrong with your body? (Yes)	0.976705
8. Except for your illness, do you have any problems? (No)	0.859750
9. Do you have personal worries which are not caused by physical illness? (No)	0.734986
10. Do you find that you lose patience with other people? (Yes)	0.612018
11. Do you ever think of your illness as a punishment for something you have done wrong in the past? (Yes)	0.367727
12. When you are angry, do you tend to bottle up your feelings? (Yes)	0.166017
Constant	-0.666633

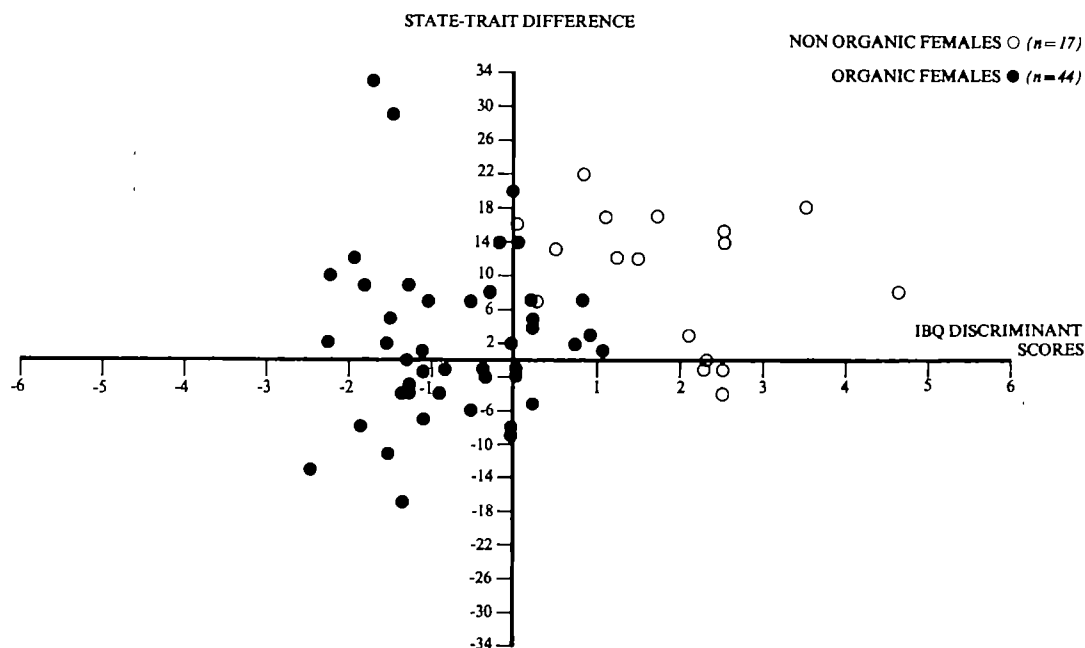


FIG. 1 A two dimensional plot of state minus trait anxiety and the illness behaviour discriminant function in females with an organic and a non-organic abdominal pain.

TABLE III
Psychological measures in non-organic female abdominal pain patients divided into those who have and those who have not had the same pain before

	Never had same pain before	Have had same pain before	
Number	8	9	
Age	25.9 (± 12.2)	35.8 (± 16.6)	NS
Trait anxiety	34.9 (± 6.3)	43.3 (± 10.1)	NS
State anxiety	51.0 (± 5.5)	47.9 (± 12.2)	NS
State-trait anxiety difference	16.1 (± 3.4)	4.6 (± 6.9)	$t = 2.24, P < 0.05$
Number with state-trait anxiety difference ≥ 12	8/8	2/9	$\chi^2 = 7.61, P < 0.01$
Zung depression	39.6 (± 7.9)	40.1 (± 8.0)	NS
<i>IBQ</i>			
General hypochondriasis	1.38 (± 1.30)	1.33 (± 0.87)	NS
Disease conviction	2.50 (± 1.41)	2.22 (± 1.48)	NS
Somatic vs psychological	1.25 (± 0.46)	0.78 (± 0.83)	NS
Affective inhibition	2.38 (± 1.85)	2.33 (± 1.50)	NS
Affective disturbance	2.13 (± 1.55)	2.89 (± 1.36)	NS
Denial	4.13 (± 1.36)	3.89 (± 1.54)	NS
Irritability	1.88 (± 1.25)	2.11 (± 1.69)	NS
IB discriminant score	1.618 (± 1.172)	2.139 (± 1.199)	NS
<i>Duration of pain (days)</i>			
<7	6	8	} NS
7-30	1	0	
>30	1	1	
Median	3	3	

abnormal physical findings, gastric-duodenal ulceration (5), gastroenteritis (4), pancreatitis (3), diverticular disease (3), cholelithiasis-cholecystitis (3), and 15 other diagnoses for either one or two patients. The duration of pain for these patients was skewedly distributed, with 82% having had their pain for less than seven days, although five reported pain for more than 99 days.

Table I presents the data (mean $\pm$ standard deviation) for all patients, by diagnosis. As the appendicitis, other organic cause and non-specific abdominal pain with physical abnormalities groups did not differ on any psychological variable, and to simplify statistical comparison, they are combined into one 'organic' group. This combined 'organic' group differs from the 'non-organic' group (i.e. non-specific abdominal pain without physical abnormalities) in terms of sex ratio, state anxiety, and on three scales of the IBQ (somatic versus psychological perception, affective disturbance and denial).

Although males and females with an 'organic' abdominal pain did not differ significantly on any of the seven scales of the IBQ, the illness behaviour discriminant analysis was performed on the data from females only, with the aim of examining differences between those with an 'organic' from those with a 'non-organic' abdominal pain. Table II lists the 12 items from the IBQ which were found to contribute to the illness behaviour discriminant score.

While the 'non-organic' patients had a significantly higher state anxiety, a better discrimination for individual patients was obtained using the state-trait anxiety difference. Using the maximally discriminating cut-off (state-trait difference ≥ 12), then 10/17 female 'non-organic' patients and 6/44 female 'organic' patients ($\chi^2 = 12.7, P < 0.001$) had this considerably higher state than trait anxiety. (Among the 'organic' males, 5/43 had a similar high state trait difference.) In Figure 1 the state-trait anxiety difference is plotted against the illness behaviour discriminant scores for 'organic' and 'non-organic' female patients.

In order to search for heterogeneity among patients who had 'non-organic' abdominal pain, the subjects were divided into those who confirmed and those who denied having had the same pain before (Table III). Those who had never had the same pain before had a higher state-trait anxiety difference but tended to have a less abnormal illness behaviour discriminant score (especially note the IBQ scale of psychological versus somatic perception).

Discussion

Within this group of 105 patients admitted acutely with abdominal pain, the most common surgical diagnosis was non-specific abdominal pain, closely

followed by appendicitis. However, those patients with non-specific abdominal pain could be divided into two groups. One group included both sexes and had abnormal physical findings; psychologically, these patients did not differ from other patients with an 'organic' cause for their abdominal pain. The second was predominantly female, did not have any abnormal physical findings, and these patients reported more state anxiety and achieved more abnormal scores on various measures of illness behaviour. Without taking into account the distinction between the presence, or absence of abnormal physical findings, the abnormal psychological findings would be partially masked. It seems reasonable to assume that all the patients with non-specific abdominal pain were regarded and treated similarly by the surgical teams, thus making it unlikely that the abnormal psychological findings arose in one group but not the other while the patients were in hospital.

Pilowsky (1978) developed the concept of abnormal illness behaviour and showed that pain clinic patients could be discriminated from general practice patients using a discriminant function which utilised the IBQ scales of psychological versus somatic perception, disease conviction, denial and irritability (Pilowsky *et al.*, 1979). Demjen & Bakal (1981) found that in patients with chronic headaches, those with continuous pain differed from those with episodic pain in their illness behaviour on the scales of psychological versus somatic perception and of denial. In the present study the non-organic patients differed on the scales of psychological versus somatic perception, denial and affective disturbance. In all three studies the IBQ scale of psychological versus somatic perception showed the greatest difference between groups.

Examination of the 12 questions which contribute to the illness behaviour discriminant score suggests a caricature of the female 'non-organic' abdominal pain patient as one whose health is the greatest difficulty of her life, who fears she may suddenly fall ill, who denies having silly thoughts about health yet thinks there is something seriously wrong with her body, who admits to no problems or personal worries other than her illness, and for whom illness may be construed as a punishment. These patients also deny feeling irritable towards other people and yet lose patience with them, while being unable to express angry feelings. These characteristics have been described by many earlier writers on pain-prone patients (e.g. Engel, 1959; DeVaul & Faillace, 1978; Blumer & Heilbronn, 1981).

Although the patients with 'non-organic' abdominal pain differed in terms of state anxiety from the

'organic' patients, they did not differ in terms of trait anxiety or depression. This latter finding is inconsistent with that of Creed (1981), who emphasised depression. Perhaps this discrepancy arises from methodological differences, in that we measured depressive symptoms (by self-report questionnaire), while Creed assessed for the presence of a depressive syndrome on the basis of a mental state examination.

When attempting to discriminate between the 'non-organic' and 'organic' patients on affective measures, the best separation of individuals is achieved not by state anxiety alone, but by the state-trait anxiety difference. We are not aware that this measure has previously been used, but it appears to have face validity in that it represents the difference (on very similar questions) between how a patient is at the present moment and how he or she generally is. This is possibly a measure of acute anxiety or exacerbation of anxiety.

The state-trait anxiety difference is not only of interest in discriminating between patients with 'organic' and 'non-organic' abdominal pain, but also when the 'non-organic' patients are divided into those who have and those who have not had a similar pain before. This division suggests the possibility of two subgroups: those subjects who have never had the same pain before tend to be younger, have a normal trait anxiety but a marked state-trait anxiety difference, and score less abnormally on the IBQ, especially on the somatic versus psychological perception scale. The other subgroup who have had the same pain before, tend to be older, have a less marked state-trait anxiety difference but a higher trait anxiety and score more abnormally on the IBQ, especially on the somatic versus psychological perception scale. Although the illness behaviour variables and anxiety variables are not totally independent, this division suggests a subgroup with acute pain related to acute anxiety, and one with recurrent pain associated with chronic anxiety and abnormal illness behaviour.

As the number of 'non-organic' patients is relatively small, and even this group may be heterogeneous, and because we have not repeated this derived discriminant function on a second sample, we are reluctant to explore issues such as sensitivity, specificity and hit rate on these data. However, the discrimination obtained between 'organic' and 'non-organic' patients (Figure 1) is remarkable, and supports the hypothesis that psychological factors are of relevance in those patients with acute abdominal pain in whom no 'organic' pathology is found.

Although there are limitations to dividing dis-

orders into those with and without an 'organic' aetiology, especially in view of the dependence of the emotions upon a physiological substrate, this dichotomy is likely to remain useful while surgeons and other medical staff have to make decisions about whether or not to treat. Our results emphasise the psychological differences between these groups of patients. Even when it is acknowledged that the pain of a patient without an organic aetiology is no less real, and the patient is in no less need of assistance for that distress, seeking medical attention and

treatment for pain may decrease the likelihood that an appropriate therapeutic response to the patient's psychological distress will occur. An inappropriate treatment response may then help to perpetuate a pattern of abnormal illness behaviour.

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The SOMA—A Questionnaire Measure of Sexual Anxiety*

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SOMA, a questionnaire designed by J. T. Quinn to measure sexual anxiety in patients presenting at a psychosexual clinic, has been used extensively as a clinical and research tool. Its construction and scoring are described, and data are presented for internal consistency, test-retest reliability, principal components analysis, and validity. Practical applications for diagnosis, prognosis, and research are discussed.

Anxiety and sexual dysfunction have long been associated with one another; psychoanalytical theory, learning theory, and the theories underlying sex therapy have all emphasised anxiety as an aetiological factor (Kaplan, 1982). Various methods of treatment of sexual dysfunction emphasise the relief of anxiety. Psychoanalysis holds that the resolution of deep-seated conflicts may relieve anxiety, while learning theories concentrate on specific circumstances that evoke and maintain disturbed behaviour in the current situation. Masters and Johnson (1970) described methods to alleviate 'performance anxiety', while Kaplan (1974), (1982) has discussed the use of psychodynamic techniques, sexual and communication tasks and tricyclic antidepressants to alleviate anxiety. Anxiety in the form of heterophobia is also considered to play a part in the genesis of homosexuality, and various treatment methods have been shown to be effective in changing sexual orientation; these include aversion relief (Thorpe *et al*, 1964) and desensitisation (Kraft, 1967).

Despite the emphasis given to sexual anxiety in the literature, few attempts have been made to devise instruments to measure it (see Norton & Jehu (1984) for a review of work in this field). Earlier research workers tended to use non-standardised methods of self-reporting of anxiety in certain sexual situations. Another approach has been the measurement of generalised neurotic anxiety in subjects with sexual dysfunction, but Cooper (1968) suggested that anxiety specifically related to sexual activities may be more important than general anxiety in the aetiology of sexual dysfunction. Hatch (1981) described psychophysiological measures of anxiety in patients with sexual dysfunction, which is an interesting

approach worth further research. Janda & O'Grady (1980) have described the development of the Sex Anxiety Inventory, consisting of 25 items with a forced-choice format; one of the responses to each item indicates sexual anxiety. The scoring system awards one point for each item on which the subject selects the response associated with anxiety. One disadvantage of such a system is that no allowance is made for the degree of anxiety experienced, so that a subject who feels only mildly anxious on a large number of items will be rated as highly anxious, whereas one who is extremely anxious about a small number of items will be rated as having only a mild degree of anxiety. Two further inventories are the Derogatis Sexual Functioning Inventory (Derogatis & Meyer, 1979) and the Sexual Arousal Inventory (Hoon *et al*, 1976) both of which purport to measure coital anxiety. Although inventories examine a comprehensive range of sexual skills and situations this unfortunately involves a loss of specificity.

It seemed valuable from a clinical standpoint to be able to measure the degree of sexual anxiety in patients presenting at a sexual dysfunction clinic. If such a measure were available, treatment could be more effectively planned for example, a high degree of heterosexual anxiety might indicate that particular attention should be paid to the desensitisation elements in the programme, or to counselling with a view to anxiety relief. SOMA (Sexual Orientation Method and Anxiety) is a questionnaire designed to measure sexual anxiety. It consists of items from the Sexual Orientation Method (Feldman *et al*, 1966), interspersed with questions relating to sexual anxiety, for which the scales were extracted from the semantic differential (Marks & Sartorius, 1968).

*The complete questionnaire and score sheet are available from The Secretary, Department of Mental Health, The Queen's University, 68 Fitzwilliam Street, Belfast BT9 7AB. Enclose the sum of £1 on application.

Construction of the Questionnaire

The Sexual Orientation Method (SOM) was described by

<i>No. of inconsistencies</i>	<i>No. of subjects</i>	
	<i>Heterosexual anxiety</i>	<i>Homosexual anxiety</i>
0	47	38
1	30	25
2	17	31
3	5	6
4	1	0
5+	0	0
Total	100	100

TABLE II
Mean pretreatment and post-treatment heterosexual anxiety scores

Clinical response	Pretreatment	Post-treatment	Pre-treatment inconsistencies
Improved (<i>n</i> = 17)	7.1 ¹	5.2	0.8
No change (<i>n</i> = 18)	7.2 ²	6.9	0.8
Worse (<i>n</i> = 11)	6.5 ²	6.7	1.8

¹*P* ≤ 0.001 (Wilcoxon Test)

²Standard deviation not significant.

sexual anxiety, 77% of subjects had 0 or 1 inconsistencies and 94% had two inconsistencies or less. For homosexual anxiety, 63% of subjects had 0 or 1 inconsistencies and 94% had less than three inconsistencies. This suggests that the technique gives a high degree of internal consistency of responses.

McCafferty (1984) studied a questionnaire of similar construction to SOMA in subjects with sexual dysfunction. He showed that subjects who had a high number of internal inconsistencies in their pretreatment testing had a poorer outcome after treatment than did those with a low number of inconsistencies. This phenomenon may reflect a personality trait or lack of commitment to treatment.

Forty-six subjects took part in an outcome study. They were assigned to one of three outcome groups: 'clinically improved', 'no change' and 'clinically worse'. All subjects completed SOMA prior to treatment; the means of the inconsistencies for the heterosexual anxiety scale for each of the outcome groups, is shown in Table II. The mean for the group who were 'clinically worse' after three months of treatment is higher than the means of the other two groups; this difference is statistically significant (*P* < 0.001, Mann-Whitney), suggesting that a high inconsistency score at initial testing may be a prognostic indicator of poor outcome.

Test-retest reliability was studied in patients who completed SOMA at their initial attendance but for some reason did not commence therapy immediately. If the delay was longer than eight weeks, the patient would normally be asked to complete the questionnaire again. These two administrations were considered as test and retest occasions: no treatment was received between test and retest. The patients had a variety of diagnoses.

The heterosexual anxiety and homosexual anxiety scores at the test and retest occasions were recorded for 35 subjects. Spearman rank correlation for the heterosexual anxiety scores was +0.95 (*P* < 0.001); for homosexual anxiety the correlation was lower (+0.75, *P* < 0.001) with a tendency for increased scores on retesting.

A principal component analysis was performed on the data obtained from 228 patients who attended a psychosexual clinic. A varimax rotation was then applied; the

matrix is shown in Table III. Three factors are extracted, which account for 93% of the total variance in the scores. The first may be seen as an 'orientation' factor involving the heterosexual and homosexual scores; the second is a 'dysfunctional' factor involving the heterosexual anxiety and homosexual scores, and the third factor is one of homosexual anxiety. All four scores contribute usefully to the information obtained. It would be possible to reduce the final amount of data presented to three items, i.e. the numerical scores on each factor. However, it has been kept in its original form in the interests of simplicity.

TABLE III
Varimax rotated factor matrix

Scores	Factor 1	Factor 2	Factor 3
Heterosexual	-0.70	-0.62	-0.02
Homosexual	0.89	0.13	0.35
Heterosexual anxiety	0.08	0.95	-0.17
Homosexual anxiety	-0.22	-0.14	0.95
Percentage of variance accounted for	55	22.4	15.7

Validity

Validation of SOMA was approached in two ways. The first involved the study of groups of subjects with assumed increased heterosexual anxiety. Two groups of normal subjects were drawn from hospital staff and medical student volunteers. These groups were unselected, except that they were of an age to be potentially sexually functioning, claimed to be heterosexual in orientation, and were satisfied with their orientation. There were 27 'normal male' subjects

TABLE IV
Means and standard deviations of scores (standard deviations are given in brackets)

	<i>n</i>	Heterosexual score	Homosexual score	Heterosexual anxiety	Homosexual anxiety
Normal males	27	45.70 (2.45)	12.63 (6.04)	3.93 (3.15)	9.96 (3.48)
Dysfunctional males	46	41.50 ¹ (6.38)	9.28 (5.20)	6.26 ¹ (4.48)	9.61 (3.34)
Homosexual males	62	25.53 ³ (10.53)	39.58 ³ (7.93)	8.23 ³ (3.90)	7.00 ³ (4.50)
Normal females	20	41.75 (4.20)	12.18 (2.35)	4.30 (3.07)	9.08 (4.98)
Dysfunctional females	52	30.90 ³ (12.41)	12.10 (8.21)	8.10 ² (3.94)	10.24 (3.55)

Levels of significance compared with normal subjects using the Mann-Whitney U-Test. ¹ $P \leq 0.05$; ² $P \leq 0.01$; ³ $P \leq 0.001$.

and 20 'normal female' subjects. Their scores are shown in Table IV.

Three groups of patients attending a psychosexual clinic were assumed to have increased heterosexual anxiety; these were dysfunctional males, dysfunctional females, and homosexual males. The dysfunctional groups included all patients presenting with any form of sexual dysfunction at the clinic in 1980 or 1981; in the case of dysfunctional males, this included erectile and ejaculatory dysfunction as well as disorders of arousal. The group of dysfunctional females similarly included disorders of arousal and orgasmic dysfunction. These scores were compared with those of normal subjects of the same sex. As predicted, these groups have higher scores on the heterosexual anxiety component of the SOMA than the non-clinical groups (Table IV).

The second approach to validation was to compare pre-treatment and post-treatment scores; it might be postulated that high pretreatment levels of sexual anxiety would tend to fall during treatment. Fifty patients who presented with sexual dysfunction were included in an outcome study; both sexes were represented and each patient completed the SOMA at the second pretreatment attendance at the clinic. At the same attendance, each patient was assessed clinically by one of the authors. Three months later each post-treatment subject was asked to complete the questionnaire again, and was again assessed clinically by the same author who was blind to the results obtained by the subjects on the SOMA on either occasion. Clinically, each patient was rated on a three-point scale as either improved, no change, or worse. Patients were also asked to class themselves subjectively as improved, the same, or worse. Those patients who both reported improvement and were rated as improved by the clinician were assigned to the 'improved' group. All were still undergoing therapy at the time of post-treatment assessment. Patients were graded as 'worse' if they either gave self-rating as worse or were rated by the clinician as worse. Five of these eleven patients had

failed to continue attendance at the clinic; they were rated by the clinician and completed SOMA at their homes. Four patients who could not be interviewed were dropped from the study; the remaining 18 (8 of whom had dropped out of treatment) were assigned to the 'no change' group.

The means and standard deviations for heterosexual anxiety for each of the three groups on the two test occasions are given in Table II, which shows a significant change in the expected direction for the improved group.

In order to further examine the nature of the homosexual anxiety and heterosexual anxiety scales, 52 subjects were asked to complete three additional questionnaires:

- The Hostility and Direction of Hostility Questionnaire (Caine *et al.*, 1967). This assesses a wide range of manifestations of aggression, hostility, and punitiveness. In this study, the questionnaire was used to assess intropunitive hostility and extra-punitive hostility, and to examine their relationship with sexual anxiety, as measured by SOMA.
- The Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975). The relationship between sexual anxiety and the neuroticism, extraversion and psychoticism scales of this questionnaire were examined.
- The Taylor Manifest Anxiety Scale (Taylor, 1953). The relationship of sexual anxiety to the measures of manifest anxiety and defensiveness were examined.

For the purpose of this comparison, all 52 subjects were chosen from the group of dysfunctional females; spurious correlations might otherwise emerge between different diagnostic groups. Spearman rank correlations were calculated for all these questionnaire scales and the heterosexual anxiety and homosexual anxiety scales of the SOMA. None of the correlations were significant. Thus sexual anxiety as measured by the SOMA is not merely a reflection of general anxiety.

Discussion

SOMA is a valid and reliable questionnaire which measures sexual anxiety in patients presenting with sexual problems. It is a useful diagnostic tool, when interpreted in the light of the normal clinical interview, but is not designed to assess sexual anxiety in subjects not presenting with sexual problems. As it indicates those patients with marked heterosexual anxiety, it should be of value in helping therapists to select those in whom treatment for anxiety will be of importance. It may also act as a prognostic indicator, in that those patients with high levels of sexual anxiety are likely to respond best to current forms of therapy. It is noteworthy that some patients present with

sexual dysfunction in the absence of marked sexual anxiety; this suggests that the current theoretical models concerning the origins of sexual dysfunction may be incomplete.

SOMA should be of value in clinical research into types of sexual anxiety and their distribution in the population. For this purpose the test requires a wider standardisation on non-clinical subjects, selected in terms of age, social status, and sexual experience. We have demonstrated that SOMA is sensitive to clinical improvement, and should thus be of value in research into the outcome of treatment. Serial testing on clinically improving subjects may give greater insights into the nature of clinical improvement.

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Psychiatric Disorders in Mildly and Severely Mentally Retarded Urban Children and Adolescents: Epidemiological Aspects

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A total of 149 children aged 13-17 years were examined. 83 were mildly and 66 severely mentally retarded. These children, especially the severely retarded ones, are representative of all mentally retarded children born in 1966-1970 and living in Göteborg, Sweden. 64% of the severely mentally retarded and 57% of the mildly mentally retarded children were suffering from a handicapping psychiatric condition. Autism-like 'psychotic behaviour' was common in the severely retarded. 0.2% of the total child population aged 13-17 years suffering from the combination of mental retardation and 'psychotic behaviour'. Epilepsy was associated with psychiatric abnormality, but Down's syndrome was generally not so associated.

Epidemiological studies of psychiatric disorders in mentally retarded children are scanty. Rutter *et al* (1970) performed a comprehensive study on the Isle of Wight in the 1960s and found the prevalence of behaviour problems—severe and mild—in children with mental retardation to be three or four times more common than in randomly selected children of a corresponding age-group. This is probably the only previous population-based study of psychiatric disorder in intellectually retarded children, and the findings are in need of corroboration. It is now almost 20 years old, was carried out in a typically rural area, and its total number of severely retarded children was small ($n=38$). Modern studies, especially from urban areas where psychiatric disorders are known to be more frequent than in rural settings, might well yield higher prevalence figures.

The present study, derived from a population-based series of 164 Swedish urban children with IQ < 70 and born in the period 1966-1970, had the following aims: to establish prevalence figures for psychiatric disorder in mentally retarded children and adolescents; to analyse the particular kind of psychiatric disorder in individual children; to analyse the effect of the degree of mental retardation on the occurrence and quality of psychiatric disorder; and to assess the association between psychiatric disorders and the additional impairments of epilepsy and Down's syndrome.

Method

Definition of terms

In accordance with the World Health Organisation recommendation of 1968, mild mental retardation (MMR)

was defined as an intellectual level in the tested IQ range of -3.3 to -2.0 standard deviation (SD) from the mean of 100 ($SD=15$), i.e. as an IQ of 50-70. The Wechsler Intelligence Scale for Children or the Terman-Merrill test were used. Severe mental retardation (SMR) was diagnosed in cases with IQ < 50.

All children showing a persistent (i.e. lasting longer than 1 month) psychiatric condition, handicapping to him or herself or to others, were diagnosed as suffering from a psychiatric disorder. Seven main psychiatric diagnostic categories were used:

- (a) *psychotic behaviour* (Gillberg, 1983), including cases with (i) infantile autism (DSM-III, 1980), (ii) schizophrenia (DSM-III, 1980), (iii) the triad of language and social impairment (Wing & Gould, 1979), excluding infantile autism and other classifiable childhood psychoses, (iv) severe impairments of social interactions, and (v) Asperger's syndrome (Wing, 1981)
- (b) *depressive syndrome*, as defined by Spitzer *et al* (1975)
- (c) *conduct disorder* in cases showing socially unacceptable behaviour, such as stealing, firesetting, or running away from home
- (d) *emotional disorder* in cases where anxiety and fear without loss of reality-sense were the most incapacitating symptoms
- (e) *psychosomatic disorder* in cases with abdominal pains, vomiting, diarrhoea, headache, and secondary enuresis or encopresis occurring in situations of psychic stress and without apparent physical cause
- (f) *hyperkinetic disorder* in cases with generalised hyperkinesia (i.e. hyperkinetic behaviour in several different settings)
- (g) *other psychiatric disorder* in cases which could not be included under any of the foregoing headings.

Some children were diagnosed according to more

TABLE I
Description of the mentally retarded series in the psychiatric follow-up study

	SMR	MMR
Number in original epidemiological study	73	91
Prevalence (%)	0.30	0.37
Dead at follow-up	4	0
Moved to another region	1	4
Refused to participate	2	4
Total attrition rate at psychiatric follow-up (dead excluded) (%)	3	8
Number in psychiatric follow-up study	66	83
Boy:girl ratio	38:28 (1.4:1)	54:29 (1.9:1)
Age range	13-17	13-17
Epilepsy: <i>n</i> (% in brackets)	18 (27)	8 (10)
Down's syndrome: <i>n</i> (% in brackets)	18 (27)	2 (2)

than one psychiatric diagnostic category. In such cases, a hierarchy was used in accordance with the order of listing given above. For example, if the child had been diagnosed as suffering from both an emotional disorder and a depressive syndrome, the latter was considered the main diagnosis.

All children on anti-epileptic medication because of 'epilepsy' or who had had at least one seizure in the 12 months preceding the psychiatric study were diagnosed as suffering from epilepsy.

Population investigated

The original population studied consisted of all children born in 1966-1970 living in Göteborg on the 31 December 1978—a total of 24 498 children. Gothenburg is an industrial city on the West Coast of Sweden with 450 000 inhabitants. Through screening and register searches (as described by Hagberg *et al.*; 1981a, 1981b) 91 children with MMR and 73 children with SMR were found, corresponding to population frequencies of 0.37% and 0.30% respectively. The prevalence figure for SMR is considered a true one, whereas that obtained for MMR is a minimum one. The MMR category did not comprise only cases known to services for handicapped children, since 20% were ascertained by screening procedures in schools. We believe that the majority of clearly mentally retarded children with IQ < 70 in the studied age-group have been detected.

The children were examined during the spring of 1984, when they were 13-17 years-old. Details of the series examined are given in Table I.

TABLE II
Psychiatric disorders in cases with severe mental retardation

Disorder	<i>n</i> (%)	% with epilepsy	% with Down's syndrome	Boy:girl ratio
So severely retarded that rating of psychiatric state was not possible	7 11	57	29	1.3:1
No psychiatric disorder	17 26	0	53	0.9:1
Depressive syndrome	1 1.5	100	0	0.1
Emotional disorder	3 4.5	0	67	0.5:1
Conduct disorder	3 4.5	0	0	2:1
Psychosomatic disorder	2 3	0	50	2:0
Psychotic behaviour	33 50	39	12	2:1
Schizophrenia	1 1.5	0	0	1:0
Infantile autism	5 8	60	20	4:1
Triad of language and social impairment	18 27	33	11	2.6:1
Severe social impairment	9 14	44	11	1.3:1
Total with psychiatric disorders	42 64	33	17	1.6:1

TABLE III
Psychiatric disorders in cases with mild mental retardation

<i>Disorder</i>	<i>n(%)</i>	<i>% with epilepsy</i>	<i>% with Down's syndrome</i>	<i>Boy:girl ratio</i>
No psychiatric disorder	36 43	8	6	1.1:1
Depressive syndrome	3 4	0	0	3:0
Emotional disorder	8 10	13	0	1.7:1
Conduct disorder	10 12	0	0	1.5:4
Psychosomatic disorder	3 4	0	0	0.5:1
Hyperactive disorder	9 11	22	0	3.5:1
Psychotic behaviour	12 14	17	0	5:1
Schizophrenia	1 1	0	0	1:0
Infantile autism	3 4	0		2:1
Triad of language and social impairment	5 6	40	0	4:1
Severe social impairment	2 2	0	0	2:0
Asperger's syndrome	1 1	0	0	1:0
Other disorders	2 2	0	0	2:0
Total with psychiatric disorder	47 57	11	0	2.9:1

Psychiatric diagnostic assessment

All children were seen individually, and assessed by at least one of three doctors who all had a minimum of six months' training in child psychiatry. Each doctor examined approximately 50 of the 149 children. The first author is a child psychiatrist with special training in the field of mental retardation, childhood psychoses, and child neurology; he first examined between three and six children, each in collaboration with the second or third author, until agreement regarding symptoms and ratings of psychiatric disorders was reached. One of the parents (usually the mother) was interviewed with a highly structured manual covering psychiatric symptomatology, social and family factors, developmental milestones, siblings' and parents' health, hereditary factors, important somatic disorders, and treatment given by other doctors. The child was observed (and interviewed whenever appropriate) for at least 40 minutes. All manuals, medical records, and interview questionnaires were reviewed by the first author, and comprehensive diagnoses formulated by him; the complete diagnostic evaluation battery is available on request.

For statistical comparison, chi square tests with Yates' correction were used.

Results

Children with severe mental retardation

The results for this group are summarised in Table II. About one in ten of the SMR children were so severely mentally

handicapped, and hence their behavioural repertoire so limited, that it was impossible to make a psychiatric classification. Only about one quarter of the SMR children were considered clearly psychiatrically normal. Altogether, 64% of the group showed major psychiatric disorders. The disturbances grouped in the 'psychotic behaviour' category were by far the most common, affecting exactly half of all the SMR children, but only 15% of these children showed infantile autism; the remaining 85% were all severely psychiatrically disturbed, had problems in the fields of social relationships and language, and demonstrated deviant behaviour such as ritualism, resistance to change, self-destructiveness, and hyperactivity. More than half ($n=9$) this group with 'psychotic behaviour' were pervasively hyperactive, but because they all showed concomitant severe disturbances of social relatedness and language, they were not classified separately as suffering from 'hyperactive disorder'.

The calculated frequency for the combination of SMR plus 'psychotic behaviour' in the general population was 0.15% in the 13-17 year age-group.

All other psychiatric disorders were much less frequent in the SMR group, but it was possible to definitely diagnose disorders such as 'depressive syndrome' and 'conduct disorder' in a few children.

Children with mild mental retardation

In the MMR group (Table III) there were no psychiatrically unclassifiable cases, even though two boys (with

disturbances of sexual behaviour) were referred to the 'other disorder' category. Slightly less than half the children were judged 'psychiatrically normal'.

Psychotic behaviour, conduct disorder, hyperactive disorder (without 'psychotic behaviour') and emotional disorder were all common, each category accounting for 10–14% of the whole MMR group. The boy with Asperger's syndrome also had periodic mania and depression, but showed no sign of such disorders at the time of the psychiatric study.

The frequency of 'psychotic behaviour' plus MMR in the general population was estimated at 0.05% in the 13–17 year age-group.

Epilepsy

In the SMR group 27% suffered from epilepsy. There were significant differences between the psychiatrically non-disturbed and disturbed groups with regard to epilepsy. In the former group none of the children had epilepsy, whereas in the latter fully one third, had a seizure disorder ($P < 0.01$). Epilepsy was particularly common among children with psychotic behaviour.

In the MMR group 10% suffered from epilepsy. There were no clear-cut differences between psychiatrically normal and abnormal children in respect of epilepsy in this group. However, hyperactive disorders and psychotic behaviour tended to be associated with epilepsy slightly more than the other conditions (22% and 17% respectively in the two former categories, compared with 4% in other groups of psychiatrically disturbed children).

Down's syndrome

In the SMR group, Down's syndrome correlated significantly more strongly with 'psychiatric normality' than with 'psychiatric abnormality' ($P < 0.02$). However, it is worth noting that one of the Down's syndrome children suffered from classic Kanner-type infantile autism. In the MMR group there were only two children with Down's syndrome, both of whom were judged to be 'psychiatrically normal'.

Sex ratios

There was a marked preponderance of boys in the MMR group (1.9:1), but in the SMR group the difference was less marked (1.4:1). Boy:girl ratios were close to population ratios in the groups of psychiatrically normal mentally retarded children (both SMR and MMR included). In marked contrast, the boy:girl ratios were raised in the psychiatrically deviant groups (MMR: $P < 0.05$; SMR: not statistically significant), and especially in those showing either psychotic behaviour or hyperactive syndrome.

Discussion

This is possibly the first-ever systematic study of psychiatric disorders in a population-based series of

both severely and mildly mentally retarded children. The results, therefore, must be considered to be of particular interest. In the Isle of Wight study (Rutter *et al*, 1970) 50% of the severely mentally retarded children were said to suffer from a 'psychiatric disorder', on the basis of interviews with parents and observations of the children, while a further 13% were too severely retarded to qualify for a rating of psychiatric state. The only recent epidemiological study of psychiatric disorders in severely mentally retarded urban children (0–15 years) is that of Corbett *et al* (1975), who suggested that a little less than half of the severely mentally retarded children in South-East London had 'behaviour disturbance', according to results obtained at psychiatric examination of the children.

Reid (1980) tried to apply the multiaxial classification scheme for psychiatric disorders in childhood and adolescence (Rutter *et al*, 1975) to a group of 41 mildly and 19 severely mentally retarded Scottish children who attended a special child psychiatric out-patient clinic for the mentally retarded. She concluded that it was quite feasible to diagnose psychiatric disorder in mentally retarded children using the same criteria as for children of normal intelligence.

The main finding of the present study—i.e. that a high proportion (more than half) of mentally retarded children have additional psychiatric handicap conditions—taken with the evidence from the British studies, has considerable clinical implications. Child psychiatric services of a wide variety must be provided for the affected children. In Sweden there are not at present enough services to take care of the multitude of problems of individuals and families that constitute the reality behind the crude figures of this report.

However, before drawing firm conclusions several issues need to be discussed. Firstly, is the population of children studied really representative of mentally retarded children, in Sweden or elsewhere? Hagberg *et al*. (1981a, 1981b), who carried out the earlier study of mental retardation, argued that the sample is indeed representative. Certainly the reported prevalence of 0.3% SMR in our study is similar to rates found by other researchers in Sweden (Gustavson *et al*, 1977; Gillberg *et al*, 1983) and the UK (Lewis, 1929; Goodman & Tizard, 1962; Rutter *et al*, 1970) and elsewhere (Tizard, 1966). Also, the rates of epilepsy and Down's syndrome in this group are typical of groups of severely mentally retarded children (Corbett, 1981). It is thus our contention that the SMR group is a representative one. The MMR group is more problematic; we are aware that our low frequency figure (0.37%) stands out as atypical

(compared with the expected value of 2.3%), and corresponds to a mean IQ of 110–112 in the general population. Terman-Merrill (1974) attribute this secular trend of increasing IQ to cultural factors (early stimulation). Furthermore, we know that a number of children with IQs close to the cut-off point of 70 have not been included (Gillberg *et al.*, 1983). A child with an IQ of 72 at any particular time might well test below 70 at any other time. Prevailing attitudes of psychologists are of major importance in this respect; we know that in some ways the modes of application of the test methods have changed over the years. For instance, some psychologists interpret the test results in a more positive way than intended by the test manual, so that established IQ would tend to increase in the child population. Therefore, we consider our MMR group to be representative of all clearly mildly mentally children, but acknowledge the possibility that some children with IQs very close to 70 might have been missed. However, our frequencies for epilepsy and Down's syndrome are not different from those found in other studies of mentally retarded children (Rutter *et al.*, 1970; Peckham, 1974).

Secondly, how reliable and valid were the psychiatric diagnoses? The first author alone made all the final diagnoses on the basis of the information elicited at the time of the psychiatric assessment. There was more than 95% agreement between the assessors after 3–6 cases of simultaneous assessment, indicating that this information was reliable.

The same kind of psychiatric classification was recently used in a study of six and seven year-old children with perceptual, motor, and attentional deficits (Gillberg, 1983). It was concluded that the psychiatric diagnoses were both reliable and valid; the total population rates for psychiatric disorders obtained were very similar to those found in other studies of urban young children. It is thus our contention that other psychiatrists would agree that 64% of SMR and 57% of MMR children in our study had truly deviant psychiatric states.

Thirdly, how far are the findings in respect of psychiatric disorders applicable to other groups of mentally retarded children? This is heavily dependent on whether or not the SMR and MMR groups are considered representative. Since an urban child population was studied, differences might appear when compared with results from non-urban populations. Rutter *et al.* (1970) found 50% of SMR children on the Isle of Wight to have clear psychiatric abnormalities, and Corbett *et al.* (1975) found 43% of South-East London SMR children to be mentally ill. These findings, taken with ours, indicate that the frequency of psychiatric disorder in the SMR group

is not likely to be heavily influenced by differences in the rural or urban nature of the environment. Also, the type of psychiatric problem—especially those grouped under the heading of 'psychotic behaviour'—and the frequent association with epilepsy indicate that brain dysfunction (malfunction rather than loss of function (Corbett, 1985)) is responsible for much of the high rate of psychiatric disorder. All the SMR children had signs of brain damage or dysfunction (Hagberg *et al.*, 1981b).

The findings in respect of psycho-social problems have not yet been compiled, and conclusions must be drawn tentatively in this regard; this should not be taken to mean that psycho-social factors are of no importance. In the MMR group there are no sound epidemiological data with which comparisons can be made. In this group too, signs of brain damage or dysfunction occurred in more than half the cases (Hagberg *et al.*, 1981b), although the kinds of psychiatric disorder are more akin to those seen in normally intelligent children, which might infer the importance of psycho-social factor in at least some of the cases. Since psycho-social diversity is the main reason for urban children having more psychiatric disturbance than rural children (Rutter, 1975), one might expect that in rural areas one would find a lower prevalence of child psychiatric disorder than our urban 57%.

Fourthly, what do the diagnostic sub-categories imply? One half of the severely retarded and one seventh of the mildly retarded children showed 'psychotic behaviour', indicating the combination of disturbances of social relatedness, language, and behaviour seen typically in infantile autism. These proportions indicate that 0.2% of the total child population in the 13–17 age group show the combination of mental retardation and the triad of language and social impairment (Wing & Gould, 1979). This figure is the same as that found by Wing in her studies of handicapped children under 15 years old in Camberwell, South-East London. It indicates that 'autistic-like conditions' are much more common than autism, and will continue to cause problems of differential diagnosis. 'Pure' infantile autism occurs only in 10–20% of these conditions in this study and in a recent epidemiological study specifically aimed at finding autism cases in the same region (Gillberg, 1984).

All the children in the group showing 'psychotic behaviour' are in need of intense and continuous psychiatric care—some as long-term in-patients, others in the form of regular out-patient treatment or access to a specialised child psychiatric team. Many of the parents and siblings too are severely handicapped by the child's disorder—far more so

than parents and siblings of other mentally handicapped children. Special education, neuropharmacological expertise, social training programmes, family treatment (ranging from family therapy to regular contact and information), and sometimes supportive individual psychotherapy are essential features in the treatment repertoires that should be provided.

Among the children with other psychiatric disorders the degree of handicap tended to be more variable, but on the whole the psychiatric conditions were of a chronic or relapsing nature. According to medical records and other data about the children, they would have been diagnosed in the same or a similar category a year or more before the present study. This needs emphasising in view of the fact that many children in this study had reached puberty and it might be argued that the high rate of psychiatric disorder was due to this 'transient' phenomenon. It appeared that puberty sometimes tended to aggravate the problems, especially in the group with psychotic behaviour, but only rarely caused or precipitated them. However, there were three children (one with conduct disorder and two with psychotic behaviour) in the SMR group whose psychiatric problems had obviously begun at the onset of physical puberty.

A majority of the children with psychiatric disturbance had been seen for out- or in-patient psychiatric assessment during development. However, this also held true of children who were not suffering psychiatric disturbance or Down's syndrome with an IQ of around 20 or higher. Many mentally retarded children in Sweden, regardless of concomitant psychiatric disorder, are seen by child psychiatrists at an early age for assessment of their mental handicap. Child psychiatric consultation *per se*, as measured by registration at child and youth psychiatric services, is therefore not an indication of psychiatric disturbance in these children.

The degree to which epilepsy correlated with psychiatric disorder, especially in the SMR group, was striking. In this group all the epilepsy patients either were so severely retarded as to be considered

unrateable in respect of psychiatric state or were suffering from some kind of psychiatric disorder. However, almost the opposite applied in the Down's syndrome group, where psychiatric disturbance was much rarer, though the stereotype that all such children are good-natured and without psychiatric problems was contradicted by the finding that four out of the 18 severely mentally retarded children with the syndrome in this study showed 'psychotic behaviour' (including one case of infantile autism).

Boys were more often affected by psychiatric disturbance than girls; this is in keeping with research on psychopathology among normal younger children (e.g. Rutter *et al.*, 1970), but inconsistent with data from teenagers (e.g. Gillberg & Höök, 1977). The reason for the discrepancy is possibly the lower mental age in mental retardation, yielding groups which, from the developmental point of view, are comparable to pre-school and primary school age children.

This comprehensive epidemiological study of 13-17 year-old mentally retarded children in Göteborg has shown that between one half and two thirds of all such children have severe psychiatric disturbances. Differences in the frequency of psychiatric disorder between severe and mild mental retardation are small, even though there appear to be considerable differences in the nature of these disorders. In most cases the psychiatric conditions can readily be classified in terms similar to those used with children of normal intelligence, but in the severe mentally retarded group there is a sub-group in which classification is impossible. Long-term experience with mentally handicapped people is probably a prerequisite for arriving at an optimal psychiatric diagnosis. Much more effort and energy needs to go into the task of providing psychiatric services for mentally handicapped children and their parents.

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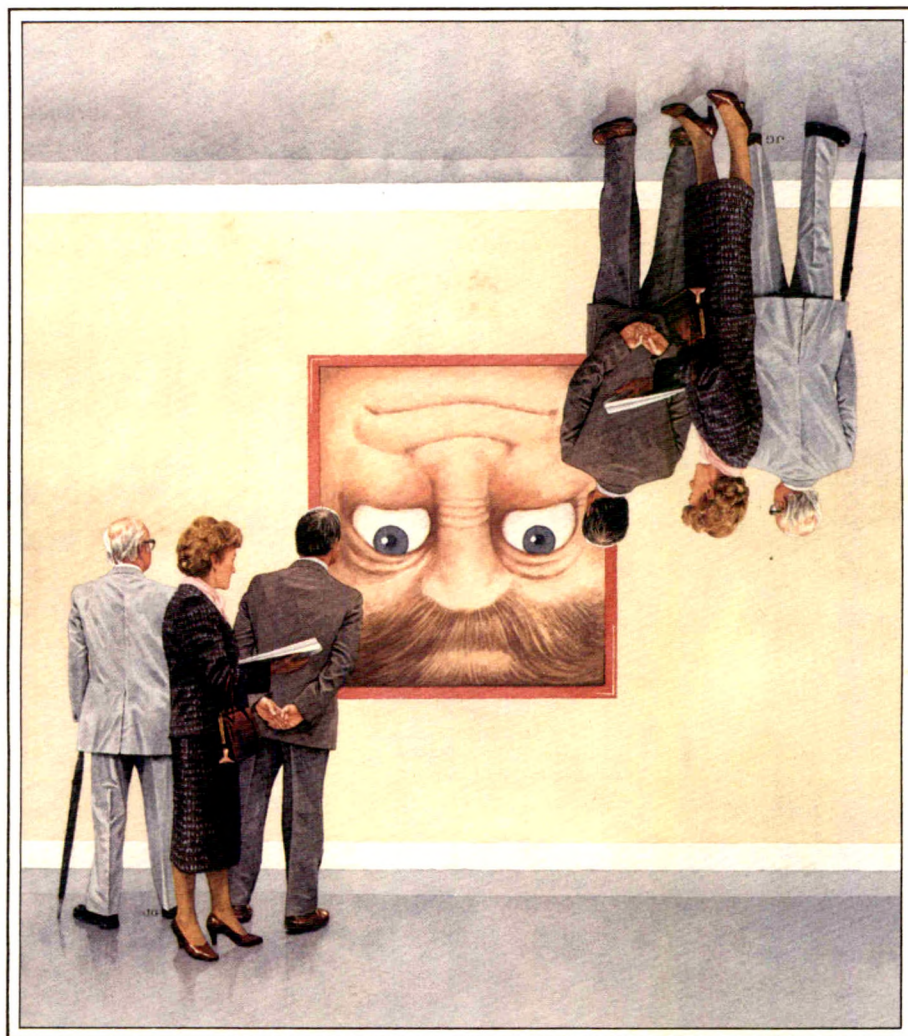
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¹ Johnson DAW Br J Psychiat 1975; 126: 457-461.

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Psychiatric Morbidity in First-Year Undergraduates and the Effect of Brief Psychotherapeutic Intervention—A Pilot Study

T. K. SZULECKA, N. R. SPRINGETT and K. W. DE PAUW

Out of 1279 first-year undergraduates, two matched groups of students potentially vulnerable to psychological disturbance were identified. One was left to its own resources; the other was offered psychotherapeutic intervention, the effects of which were measured by the number of consultations with general practitioners, type of treatment and rate of withdrawal from university. Although the students in the intervention group had fewer consultations, lower General Health Questionnaire scores at follow-up, and fewer withdrew from university, due to the small numbers involved none of these differences achieved statistical significance.

First-year university students are especially prone to develop psychological and personal problems. Kidd (1963) reviewed the relevant literature and emphasised that during this unknown life situation, the individual is newly confronted with the need for independence in thinking, decision-making, and actions. A new approach has to be taken in studies and in living in a student community. A balance has to be struck between the demands of the university and a freedom never before experienced. Rook (1959) pointed out that this intellectual freedom may, in the weaker members, lead to a sense of loss of direction and a feeling of inadequacy. Students who present for treatment within the first two months at university are those with more severe disturbances (Malleon, 1961; Payne, 1967; Entwistle *et al*, 1977). Davidson & Hutt (1964) analysed case records of 500 Oxford undergraduate patients and found that those who had a poor relationship with their parents, underwent previous psychological investigation, had a positive family history of psychiatric illness, and who studied English, law, music or classics, were more likely to be referred to the psychiatric services.

This paper examines the usefulness of the 28-item General Health Questionnaire (GHQ; Goldberg & Hillier, 1979) in screening a student population for psychological disturbance, and the results of offering psychological help to those first-year undergraduates who, on the basis of high GHQ scores, appear to be at risk of developing psychiatric illness. The benefit of short-term psychotherapy for such students has been discussed by Dorosin *et al* (1976), Haggerty *et al* (1980) and Pinkerton & Rockwell (1982).

The following hypothesis was proposed, that the provision of psychotherapeutic intervention has

an effect on the number of students withdrawing from the university, the consultation rate with their general practitioners, and the amount of prescriptions issued for minor tranquillisers.

Location

In 1980, 1963 students entered Nottingham University and registered in the Faculties of Arts (342), Law and Social Science (485), Pure Science (663), Applied Science (343), and Medicine (130). Most first-year students live in Halls of Residence, which provide the main focus of social life. Support for those with emotional problems may be obtained from other students, hall wardens and hall tutors, a part-time psychologist, a full-time students counsellor, three chaplains and academic tutors. The University Health Centre has four GPs who are experienced in dealing with psychologically disturbed students. They have in-patient facilities at their disposal, where minor psychological problems are treated without the necessity for formal involvement of the psychiatrist who visits the centre once a week, and to whom there is easy access at other times. A friendly and informal atmosphere at the Health Centre helps in the rapid exchange of information and expertise. Student-to-student services include a 14-hour overnight telephone counselling service called "Niteline" and for students with a homosexual orientation, weekly telephone support is available through "Gayline".

Method

The 28-item General Health Questionnaire (GHQ1) was administered to incoming students at the time of their registration with the Nottingham University Student Health Centre. All students who scored six or more on the GHQ1 were divided into two groups. The groups were matched for sex, age, subjects studied, GHQ1 scores, and the four GHQ subscores, type of relationship with parents, previous psychological investigation or help and family history of psychiatric disorder. The last three items were

determined by means of an additional questionnaire attached to the GHQ1 (Appendix), derived from Davidson & Hutt's (1964) observations, and are referred to here as vulnerability factors. The matching was carried out by a statistician who had no personal contact with any of the students. Another independent person arbitrarily designated one group as the Intervention Group (IG) and the other as the Control Group (CG). A third matched group of non-cases (MG), with the closest possible match to the IG but without vulnerability factors and with zero scores on the GHQ1, was included in the follow-up procedure to minimise the chances of the cases being identified by GPs and friends, and to provide additional comparison to the other two groups. Within the first month the IG members were invited by post for a 10–15 min individual interview which consisted of an inquiry into their feelings about the administration of the GHQ, their adjustment to university life and stresses related to separation from their families. They were also informed about the availability of support for any emotional problems, from one of the authors (TKS) doctors of the University Health Centre, or the Student Counsellor (NRS). Students who did not keep the first appointment were offered another one.

Students from the IG who asked for help had their initial interview extended, or were offered brief psychotherapy sessions with TKS, or were referred for more appropriate help. The students from the remaining two groups and those from IG who did not keep appointments were left to their own resources.

In the last month of the academic year all three groups

were individually invited to the Health Centre to repeat the General Health Questionnaire (GHQ2). Those who did not respond, despite the offer of another appointment, were visited in their Halls of Residence by TKS or KdeP. Those who had left the university had the questionnaire sent to their home.

At the end of the academic year the rate and character of consultations with the University Health Centre were studied. Each withdrawal was examined in terms of date and reason for dropping out.

Results

The GHQ1 was completed satisfactorily by 1279 students; 104 of them (8%) scored six or more (Table I).

On follow-up in the third term, three students from the IG refused contact and three others did not complete the questionnaire. From the remaining 46, 13 scored six or above (28%). In the CG, one student did not complete the GHQ; of the remaining 51, 22 scored six or above (43%). The improvement in GHQ scores was greater in the IG group, but failed to reach statistical significance.

Initial interview

Thirty-five out of 52 students (67.5%) from the IG kept their appointments with TKS. Five of the students seen (14%) expressed a need for further support, and were subsequently seen for longer interviews: two were satisfied

TABLE I
Demographic characteristics and GHQ scores in intervention, control and matched groups (n = 52 in all groups)

	<i>Intervention Group (IG)</i>		<i>Control Group (CG)</i>		<i>Matched Group (MG)</i>	
Sex	Male	Female	Male	Female	Male	Female
Age						
Mean	18.5	18.3	18.5	18.4	18.5	18.3
Range	18–22	17–19	18–23	17–21	18–22	17–19
GHQ scores						
Mean	7.2	9.9	8.4	9.0	0	0
s.d.	1.6	4.4	2.4	2.7	0	0
Number of students with vulnerability factors*:						
None	18	15	17	16	26	26
I	3	1	2	2	0	0
II	1	2	0	1	0	0
III	0	1	0	1	0	0
IV	1	1	1	2	0	0
V	1	1	2	1	0	0
More than one vulnerability factor	2	4	4	3	0	0
Refused to complete		1				

*See Appendix.

TABLE II
Withdrawals

Student number	GHQ scores		Sex	Vulnerability factors	Age	Faculty	Term left
	First term	Third term					
Intervention group (IG)							
1072	14	15	F	I,II	18	Pure Science	I
0035	6	7	M	None	19	Arts	III
0854*	22	0	F	I	18	Arts	III
0866*	22	3	F	I,III	18	Arts	I
Control group (CG)							
2178	13	1	M	V	18	Pure Science	II
0517	9	0	F	II,III	18	Arts	II
0337	6	†	M	None	18	Social Science	I
0509	10	0	F	III	18	Arts	II
1020	13	0	F	I, II & III	18	Social Science	II
0498	8	9	M	I	18	Applied Science	Failed
Matched group (MG)							
0742	0	†	F	None	18	Maths	Failed
0102	0	0	M	None	18	Physics	Failed
2190	0	1	M	None	17	Maths	Transferred
0216	0	0	M	None	18	Maths	Lost one year Failed

*Did not keep appointment with TKS.

†Did not complete it.

with a single hour-long session each; one was seen twice more and wished to have further contact, which was not possible due to her timetable; one was referred for group psychotherapy after five one-hour consultations. The fifth student (later referred to as Student No. 1072) had already been admitted to the Health Centre and was seen on five occasions. Three students (5.7%) extended the initial contact to 40–90 min. These three were weepy, complained of sleep and concentration difficulties, and had 10 months' amenorrhoea in one case. Their distress was due to anxieties related to separation from home and pressure to develop new loyalties and friends. All but one declined immediate follow-up, but felt reassured by the knowledge of the available supportive facilities. A further two students readily acknowledged the emotional nature of their problems, e.g. difficulties in sharing a room and making friends, and an association between migraine attacks and a facial tic with psychological pressure. At first they did not desire further intervention, but consulted their GP at a later date.

Consultations with University Health Centre

The Students' Health Centre had 9883 NHS registrations

in 1980 and 9017 in 1981. In the period from 1 August 1980 to 31 July 1981, 16 115 consultations with a GP were made: 62% of these consultations were made by undergraduate students. The consultation rate for those who saw a doctor was 4.1 per person per year.

Twenty-eight IG students made 125 consultations (mean 2.3); 12 were classified by the GP as psychiatric (three individuals), 13 were due to trauma, 21 students never consulted their doctors, and three case notes were not available for examination. Twenty-seven CG students made 148 consultations (mean 2.9), 15 were psychiatric (11 individuals), five were due to trauma and four case notes were not available. Thirty-one students from MG made 104 consultations; of them, one had two psychiatric consultations, 15 were due to trauma (eight students), 19 never consulted their doctor, and two case notes were not available for examination. Missing case notes were unavailable as they had already passed to the home GP after the student's withdrawal from university. None of the students received prescriptions for minor tranquillisers. The mean number of consultations per person per year for those who consulted a doctor was 4.3, 4.0 and 3.3 for IG, CG and MG respectively. None of the differences were statistically significant.

TABLE III
Effects of intervention

	IG	CG	MG
Reduction in GHQ scores (GHQ1 - GHQ2)			
Mean	4.7	3.5	-1.7
s.d.	5.5	6.1	2.9
Number withdrawing from university	4	5	0
Failed	0	1	3 (6%)
Number who consulted GP	28 (58%)	37 (77%)	31 (62%)
Number of consultations per student who saw GP: mean	4.3	4.0	3.3
Consultation rate for all students			
Mean	2.4*	2.8	2.1
s.d.	3.3	2.4	2.9
<i>t</i> -test (IG vs CG)		NS	
Prescription of minor tranquillisers	0	0	0
Number of individuals with psychiatric consultation (number of all consultations)	3 (12)	11 (15)	1 (2)

*One student in IG consulted 19 times.

Withdrawals

Table II shows withdrawals from IG, CG and MG.

Of the four students who withdrew from the IG, two were seen by TKS. One student (No. 1072) was already an in-patient at the Health Centre at the time of the initial interview with a diagnosis of paranoid psychosis and she was later transferred to a local psychiatric hospital. At the 10-month follow-up her GHQ score remained high, and 13 months later she was still incapable of resuming her academic career. Another student (No. 0035) complained of suicidal thoughts and of dissatisfaction with various aspects of life on entering university, but turned down an offer of further help. He saw his GP on four occasions, complaining of headaches. In June his father suggested psychiatric help, but the student withdrew in July without taking any exams or seeing a specialist. The two remaining students (Nos 0854 and 0866) did not keep their appointments with TKS; they both scored 22—the highest score of those tested.

Table III summarises the effects of intervention.

Discussion

The main effect of the psychotherapeutic intervention offered was reflected by the number of definite psychiatric consultations with GPs. The CG group consulted more frequently but group members were

not persistent in seeking help. This could have been due to the fact that they were sufficiently helped by a single consultation, or more likely were satisfied with a single reassurance. In the IG group, this role was taken by TKS, and only those in need of more intensive help consulted their GP's at a later stage. If early intervention did nothing else but reach those in need of reassurance, in a group identified by high scores on the GHQ1 one in six students needed it. If both the GHQ and vulnerability factors were combined in an effort to identify the group needing help, the results could have been more dramatic. Unfortunately, in this pilot study, the consultation rate of students with various vulnerability factors was not examined. However, on the basis of their increased rate of psychiatric morbidity (Davidson & Hutt, 1964) and increased withdrawal rate, we can draw the conclusion that both the GHQ and vulnerability factors are useful tools in identifying students in need of psychotherapeutic help.

Our findings indicate that first-year students use the University Health Centre to the same extent as the rest of the population, and that students with increased GHQ scores do not consult their doctors more often, despite being in need. This was confirmed by our own observations of their reluctance

to become involved with helping agencies, and supports the view that students with academic problems also tend to avoid seeing advisers (Nisbet & Welsh, 1976) and are more reticent about disclosing personal information (Hall *et al.*, 1982). Therefore, our initiative to get in touch with students in need increased the chances of reaching those who would otherwise have been reluctant to seek help.

Students who were seen in the third term approved of the use of the questionnaire—but later in the first term, when newcomers might have overcome their first anxieties about settling into university life. They saw it as evidence of care, and it strengthened their confidence in the Health Centre, reflected in the relatively high consultation rate for the first-year students. Cobb (1976) has defined support as 'information leading the subject to believe that he is cared for and loved, esteemed, valued, and accepted'. For the majority of our students, the screening procedure implied that "somebody bothered", but extreme reactions in both directions were present, ranging from a resentment at intrusion into private areas to a feeling of gratitude.

GHQ scores or suspected psychological vulnerability were not disclosed to students during the interview: only when students themselves expressed a wish for help were their problems discussed. It remains an open question whether such a disclosure would have been of any help or would only have added another psychological burden. As Lucas *et al.* (1966) have pointed out, it is difficult to find the right level of intervention. Determining valid measures of improvement in any psychotherapeutic intervention remains problematic (Candy *et al.*, 1972; Bloch *et al.*, 1977; Adams, 1978; Prioleau *et al.*, 1983; Shepherd, 1984) and is even more difficult in brief contacts such as in this study.

The withdrawal rate for the intervention group (9.7%) was lower than that of the control group (11.5%), but this failed to reach statistical significance.

The reduction of the withdrawal rate from university through psychological intervention was possibly not more striking not only because of the small numbers involved, but also because in 67.5% of those who responded, preselection of less vulnerable cases took place, and only those who expressed a need for help were offered it. Confidentiality prevented disclosure of the results to GPs and jeopardised involvement of those who were more readily available to students at the later stage. Student No. 0035 seems to be a good example of this. His withdrawal could perhaps have been prevented by alerting the GP to his psychological problems. This demonstrates the potential usefulness of

prior knowledge of GHQ scores and 'vulnerability factors' as a means of alerting GPs to the somatic presentation of primary psychiatric disturbance which is so commonly found in a general practice population (Goldberg & Huxley, 1980). The two students who never kept their appointment with TKS were further examples of those who resist any offer of help; they either demonstrate the limits of such an approach in this group or emphasise the need for a more determined follow-up. Student No. 1072 had a major psychiatric disorder and was in need of more intensive psychiatric help than short intervention.

Students themselves stressed the advantage of discussing problems with their peers. All psychotherapeutic factors in a spontaneously organised group would then seem to come into play (Yalom, 1975) and many of them were relieved to discover that they were not isolated in their distress. Facilitation of such naturally occurring self-help groups in halls of residence could be an appropriate way of helping vulnerable students. Its therapeutic value could be increased by involving tutors normally responsible for a defined group of students who can be brought together without fear of being labelled as psychiatrically disturbed. This would also increase involvement in the student community itself, would aid integration into university life, and possibly reduce the drop-out rate from university (Tinto, 1975). It is striking that three students (6%) from the MG failed their examinations and that none left the university of their own accord. Ryle (1969) indicated that academic underachievement in emotionally stable students could be due to the anxiety levels being too low for efficient work. The lack of change in the GHQ2 scores in our sample, despite academic problems, supports this theory further. The MG performance and rate of failure might have been influenced by additional academic pressures.

Acknowledgements

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Appendix

Student Vulnerability Inventory

Please circle the correct answer

I Would you describe the relationship with your father as:

	Very good	Satisfactory	Hostile	Yes	No
	Resentful	Ambivalent	Other (specify)		
II Would you describe the relationship with your mother as:				IV Is there anybody of your close family (father, mother, siblings) who has suffered from a psychiatric illness?	
	Very good	Satisfactory	Hostile	Yes	No
	Resentful	Ambivalent	Other (specify)	V Is there anybody in your distant family who has suffered from a psychiatric illness?	
III Have you had previous psychological investigations or help?				Yes	No

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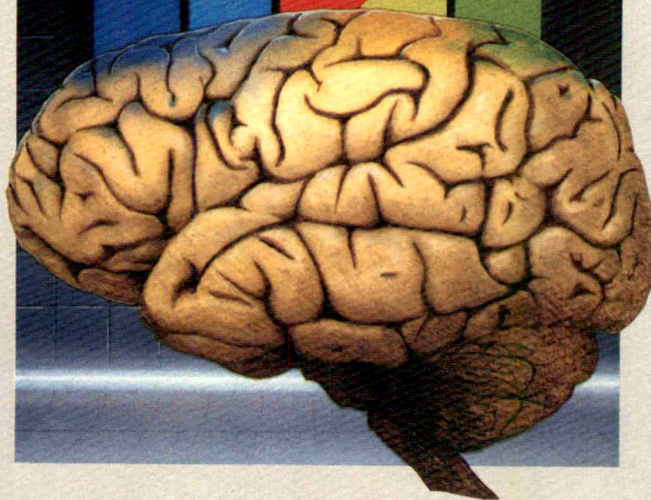
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A SCHIZOPHRENIC.

Adolf Wölfli spent 35 years in the Waldau Mental Hospital, Berne, suffering from schizophrenia. During that time he produced many drawings that have since gained widespread acclaim.

The picture below is one of a number that, we believe, give an insight into the schizophrenic mind and is reproduced with the kind permission of the Museum of Fine Art, Berne.



A SOLUTION.

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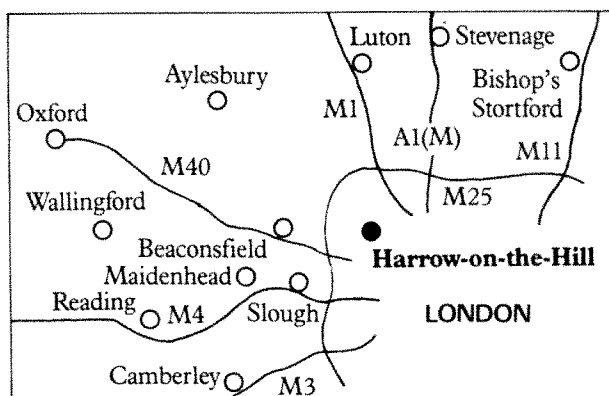
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Autism and Autistic-like Conditions in Swedish Rural and Urban Areas: A Population Study

SUZANNE STEFFENBURG and CHRISTOPHER GILLBERG

The total population of children under 10 years in one Swedish urban area and one rural area was screened for infantile autism (IA) and autistic-like conditions (AC). A total prevalence of 6.6 per 10 000 was found, which is somewhat higher than in previous similar studies of the same region. Infantile autism accounted for two-thirds of the cases. Boys far outnumbered girls, but this was entirely accounted for by the IA group. The preponderance of autistic boys was less pronounced among the severely mentally retarded children. Mental retardation was seen in almost 90% of cases and only one child had an IQ exceeding 100; clinical and laboratory signs of brain dysfunction were also found in a majority of cases. Distribution by social class was no different in either patient group from the general population.

To our knowledge there exist four published population-based studies of infantile autism and similar conditions (Lotter, 1966; Wing & Gould, 1979; Gillberg, 1984; Bohman *et al.*, 1983). These have all yielded fairly consonant results, with frequency figures ranging from 4 to 6 per 10 000 children, 'nuclear autism' accounting for about half of these. The frequency figures are almost stunningly similar in view of the well known fact that the diagnosis of infantile autism continues to cause controversy. Wing & Gould (1979) made claims for a new diagnostic category, viz. 'the triad of language and social impairments', in which they included cases of 'nuclear autism'. They found frequency figures for this triad of 21 per 10 000 in a south-east London borough. Gillberg, working in Gothenburg, Sweden, obtained exactly the same figure for the estimated frequency of the triad in the general teenage population (21 per 10 000) by extrapolating from studies of the mentally retarded (Gillberg *et al.*, 1986); and an even higher estimate for the general population when children with minor neurodevelopmental disorders were screened (Gillberg, 1983a). The studies by Wing and Gillberg show that there exist a number of children with syndromes similar to infantile autism in terms of both symptomatology and needs for services, and both these authors have argued for 'a spectrum view' of autism. However, both authors agree with Rutter (1978) in acknowledging the existence of a core group of classically autistic children within this spectrum. These are the kinds of children originally described by Kanner (1943) and said to fulfil the crucial diagnostic criteria of autistic aloneness and

stereotyped behaviour (elaborate routines) suggested by Kanner & Eisenberg (1956).

Two of the population-based studies originated in Sweden. These might be considered to be of special interest, as they represent investigations performed in areas with quite different social structures and were performed by researchers who discussed diagnostic criteria in some detail. The study by Bohman *et al.* (1983) was performed in the late 1970s in an almost exclusively rural area in northern Sweden, whereas the one by Gillberg was done in the same period in a large industrial urban area in the south-west of Sweden. The rural study came up with a frequency for 'childhood psychosis' of 6 per 10 000, 'classical autism' accounting for half of the cases. The urban study, on the other hand, yielded a corresponding figure of 4 per 10 000, 'infantile autism' cases again constituting about 50%. The difference between the results of these studies might prove to be due to chance; or it might turn out to be important, considering the well known facts that child psychiatric problems in general are very much more common in urban than in rural areas (Rutter *et al.*, 1970; Rutter, 1975) whereas mental handicap tends to be distributed the other way around.

Gothenburg and its neighbouring rural area (the county of Bohuslän) now provide unique possibilities for population-based studies of infantile autism and similar conditions. We performed population screenings in 1978 and 1980 and these paved the way for intensified detection and diagnostic services in the area. We therefore considered it of special value to perform new population screenings in these areas, in order (a) to shed some further light on the issue

of diagnosis, and (b) to analyse the importance of certain demographic factors in autism and autistic-like conditions.

Definitions

Infantile autism (IA) was defined in accordance with the criteria of the DSM-III (A.P.A., 1980), which requires:

- (a) Onset before 30 months of age
- (b) Pervasive lack of responsiveness to other people (autism)
- (c) Gross deficits in language development
- (d) If speech is present, peculiar speech patterns such as immediate and delayed echolalia, metaphorical language, pronominal reversal
- (e) Bizarre responses to various aspects of the environment, e.g. resistance to change, peculiar interest in or attachments to animate or inanimate objects
- (f) Absence of delusions, hallucinations, loosening of associations, or incoherence, such as are found in schizophrenia.

All children with IA also fulfilled the criteria of both Rutter (1978) and Coleman & Gillberg (1985) for infantile autism. In the following text this diagnostic entity is also referred to as 'autism' or 'autistic syndrome'.

Autistic-like conditions (AC) were defined as conditions similar to IA in respect of symptomatology, but either showing some highly atypical traits (e.g. extremely clinging behaviour or confusion) or commencing after the age of 30 months (according to medical/psychological record data). What we refer to as 'autistic-like conditions' have been referred to as 'other childhood psychoses' in previous publications.

Method

Populations studied

Urban: All children born during the years 1975–1984 and living in the city of Gothenburg on 31 December 1984 constituted the urban target population. The total number of children in this population was 42 886 (21 949 boys and 20 937 girls).

Rural: All children born during 1975–1984 and living in the county of Bohuslän on 31 December 1984 constituted the rural target population. The total number in this population was 35 527 (18 268 boys and 17 259 girls).

Organisation of services for autistic children in Gothenburg and Bohuslän

During the last ten years diagnostic and treatment facilities

for 'psychotic' children have been rapidly developed in Gothenburg and Bohuslän. The second author (C.G.) has performed two major screening studies, one in 1978 and the other in 1980. These have led to markedly increased interest in autism and similar conditions among paediatricians, child psychiatrists, child neurologists, audiologists, well-baby clinic personnel, school health officers (doctors, nurses and psychologists), teachers, and officials concerned with mental retardation. Both authors have given lectures on autism to all these categories of professionals, and the symptoms of autism and autistic-like conditions as they appear from infancy through childhood have been described in some detail.

At the present time, most children in Gothenburg and Bohuslän who are suspected of suffering from autism and autistic-like conditions are referred to the second author for a diagnostic evaluation. Six or seven years ago, many autistic and autistic-like children were referred for the first time only after the age of seven years. Nowadays, most children with autism have their condition diagnosed at a much earlier age, often at 1–2 years, very rarely after age 5 years.

In Sweden the child health services encompass 99% of all children, who have regular medical check-ups almost every year before age 10 years. Before starting school they are seen at age 6 years by a clinical psychologist. All children known to the Board for Provisions and Services to the Mentally Retarded (BPSMR) have medical check-ups yearly.

It is improbable that any large fraction of children with autism or autistic-like conditions could escape recognition in a region where coverage is so nearly complete and knowledge in this particular field is at a fairly high level.

Questionnaire screening

In November 1984, a letter of information and a questionnaire were distributed to all paediatricians, child psychiatrists and doctors working in the institutions for the mentally retarded, phoniatric/audiology departments, school health authorities and well-baby clinics in the city of Gothenburg and the county of Bohuslän, with the request that the questionnaire be completed and returned within two months. All schools (including special schools for autistic, mentally retarded, maladjusted, blind and deaf children) are regularly surveyed by paediatricians, child psychiatrists, ophthalmologists or audiologists. This meant that the screening by questionnaire was not limited to children referred to clinics. Furthermore the second author (C.G.) is in charge of the medical section of the BPSMR (covering Gothenburg and Bohuslän) and of services and schools for autistic children within the Child Psychiatric University Clinic in Gothenburg. This means that he has himself examined all possibly autistic or definitely autistic children attending schools for mentally retarded and autistic children in Gothenburg and Bohuslän.

Each doctor receiving a questionnaire was asked if he/she (or any of his/her collaborators) knew of any child born in 1975 or later with 'infantile autism, childhood schizophrenia, childhood psychosis, mental retardation

with autistic traits, severe disturbance of social relationships plus speech-language disorder, or severe compulsive disorder'. The doctor was requested to state the child's diagnosis, give a short description of the symptoms, report age of onset, summarise tests and other investigations, describe treatment given, and also describe the type of dwelling and the school attended by the child. Through the questionnaire we also reached all clinical child psychologists working in the region, as they all work in close collaboration with the doctors we contacted. All persons (doctors, psychologists, social workers, teachers, physiotherapists, etc.) known to be involved with services for psychotic children were contacted personally by the second author and interviewed in accordance with the questionnaire.

Register search

The registers of the child psychiatric/child guidance clinics, paediatric clinics and the BPSMR were searched in

January 1985 in order to identify cases of 'infantile autism', 'childhood psychosis', 'childhood schizophrenia' and 'mental retardation with autistic traits'.

Diagnostic procedure

All children reported were either dismissed as 'non-suspected' cases after we had conducted a thorough interview with the reporting doctor, psychologist or teacher, or were personally assessed by the second author (C.G.). more than 90% of the reported cases were known to the second author already before the questionnaire screening.

Clinical assessment

All children known by the second author personally to suffer from infantile autism and similar conditions had previously been thoroughly examined by him. All the case records (medical and school reports) of these children were re-analysed.

TABLE I
Number of cases of infantile autism (IA) or autistic-like conditions (AC) in Gothenburg (urban) and Bohuslän (rural) at the end of 1984

Year of birth	IA		AC		Total (IA + AC)		
	Gothenburg	Bohuslän	Gothenburg	Bohuslän	Boys	Girls	All
1975-1977	7	12	6	2	22	5	27
1978-1980	10	2	6	1	13	6	19
1981-1984	3	1	0	2	4	2	6
Total	20	15	12	5	39	13	52

TABLE II
Prevalence figures for infantile autism (IA) and autistic-like conditions (AC) in Gothenburg (urban and Bohuslän (rural) at the end of 1984 (among children born 1975-1984)

Diagnostic group	Gothenburg	Bohuslän	Total
Infantile autism			
prevalence	4.7/10 ⁴	4.2/10 ⁴	4.5/10 ⁴
boy:girl ratio ¹	5.4:1	6.1:1	5.7:1
Autistic-like conditions			
prevalence	2.8/10 ⁴	1.4/10 ⁴	2.2/10 ⁴
boy:girl ratio ¹	1.0:1	1.4:1	1.2:1
IA + AC combined			
prevalence	7.5/10 ⁴	5.6/10 ⁴	6.6/10 ⁴
boy:girl ratio ¹	2.4:1	3.8:1	2.9:1

¹Population-based ratio, adjusted to allow for boy girl ratio in the general population

TABLE III

Intellectual levels (IQs) of boys and girls with infantile autism (IA) and autistic-like conditions (AC): urban and rural areas combined

Intellectual level	IA		AC		IA + AC	
	n(%)	boy:girl	n(%)	boy:girl	n(%)	boy:girl
IQ < 50	21(60%)	16B:5G	9(53%)	4B:5G	30(58%)	20B:10G
IQ 50-70	10(29%)	10B:0G	6(35%)	5B:1G	16(31%)	15B:1G
IQ > 70	4(11%)	4B:0G	2(12%)	0B:2G	6(12%)	4B:2G
Total	35(100%)	30B:5G	17(100%)	9B:8G	52(100%)	39B:13G

Children suspected of suffering from psychosis on the basis of the questionnaire reports and not known by the author were assessed using routine clinical child psychiatric techniques. Relevant case records were collected and scrutinised.

A majority of the children in the study had already been tested with the Griffiths Developmental Scale for Children (Griffiths 1970) and the Vineland Social Maturity Scale (Magne & Wahlberg, 1961). The remaining children had been considered untestable and were judged clinically to be severely mentally retarded (IQ less than 50). The Autism Behavior Checklist (Krug *et al.*, 1980) and a newly constructed interview schedule pertinent to the study of autism were used to elicit relevant information in all cases.

A diagnosis of infantile autism (IA), autistic-like condition (AC), or 'non-autistic/non-autistic-like' was assigned in each case.

conditions (Table II). Infantile autism was considerably more common than autistic-like conditions (4.7 versus 2.8 per 10 000).

Bohuslän (rural). The prevalence for IA and AC combined in the rural county was 5.6 per 10 000. IA was more frequent than AC, accounting for 4.2 per 10 000 children.

Intellectual level

Slightly more than half of the children were severely mentally retarded (Table III). One-third of this group consisted of children with tested IQs in the 40-50 range. A large majority of the remainder had developmental or social quotients below 30. About 15% of the children were of normal or near-normal intelligence.

Statistical methods used

Chi-square tests were used in the statistical analyses of the results.

Results

Numbers of children with IA and AC Gothenburg (urban): There were 20 children (17 boys and 3 girls) with infantile autism in Gothenburg (Table I). Of these, only three were aged 4 years or under at the time of the study. Twelve more children (6 boys and 6 girls) with autistic-like conditions were identified: all of these were aged 5 years or older.

Bohuslän (rural) There were 15 children (13 boys and 2 girls) with IA in Bohuslän. One of these was under 4 years old. A further five children (three boys and two girls) were found to have AC: two of these were under 4 years old.

Population prevalence

Gothenburg (urban): Altogether 7.5 per 10 000 of all children in Gothenburg suffered from autism or autistic-like

TABLE IV

Obvious clinical organic signs of handicap associated with infantile autism (IA) and autistic-like conditions (AC): urban and rural areas combined

Clinical sign	IA (n=35)	AC (n=17)
Epilepsy	5(14%)	7(41%)
Moebius syndrome	3(9%)	
Martin-Bell syndrome ¹	3(9%)	
Tuberous sclerosis	1(3%)	
Laurence-Moon-Biedl syndrome	1(3%)	
Hydrocephalus	1(3%)	
Neurofibromatosis	1(3%)	
Hearing deficits		1(6%)
Cerebral palsy		1(6%)
Total number at Children	14(40%)	8(47%)

¹Several more children had other chromosomal abnormalities

Boy:girl ratios

In the autistic group the boy:girl ratio was 5.7:1 after adjustment for the boy:girl ratio in the general population. In the group with autistic-like conditions the corresponding ratio was 1.2:1. Among the severely mentally retarded children (IQ < 50) the boy:girl ratios were much lower than among those with milder degrees of intellectual retardation: this trend was particularly pronounced in the autistic group.

Associated handicaps

Two-fifths of the autistic group and almost half of the group with autistic-like conditions had 'obvious clinical organic signs' (other than mental retardation) indicative of CNS-dysfunction (Table IV). A majority of the children in both groups had radiological, physiological or chemical signs of nervous-system damage or dysfunction.

Almost a quarter of the children had developed epilepsy (Table IV). This was much more common in the group with AC. Two-thirds of the children with seizures were girls. Ten of the 12 children with epilepsy had had an onset of seizures before 12 months.

Three autistic boys suffered from congenital bilateral palsy of the facial nerves (Moebius syndrome) and another three had the Martin-Bell syndrome with some or all the physical characteristics of that diagnostic entity and the fragile (X) (q273) chromosomal abnormality.

TABLE V

Analysis by social class of children with infantile autism or autistic-like conditions, compared with a random sample of 7-year-olds from the general population. There were no statistically significant differences between the three groups

Social class	IA	AC	General population
I	1(3%)	1(6%)	4(7%)
II	10(29%)	6(35%)	24(41%)
III	24(68%)	10(59%)	31(53%)

Social class

There was no obvious or significant social-class bias (class assessed according to father's occupation: Swedish Central Bureau of Statistics, 1968) in either the IA or the AC group when contrasted with a randomly selected group of normal seven-year-olds (this latter group is described in detail in Gillberg & Schaumann, 1982). If anything, there was a slight trend towards lower social class among the autistic and autistic-like children (Table V).

Discussion

This population-based study of autism and autistic-like conditions yielded higher prevalence figures

than an almost identical study performed a few years earlier by the same team in the same region (Gillberg, 1984). The previous study found a total of 4.0 such children per 10 000, infantile autism accounting for half the cases: the new study showed a total prevalence of 6.6 per 10 000, infantile autism accounting for two-thirds of the cases. The higher prevalence is probably due to better detection of cases in the new study. It is highly unlikely that autism has become relatively much more common in only five years. The prevalence figure obtained now is very close to that found by Bohman *et al* (1983) in northern Sweden, in another area with good coverage and relatively well-developed assessment facilities.

In our region, 'autism detection sensibility' has been raised most particularly among those working with the mentally retarded from 1980 through to 1984: almost all of the 'extra' prevalence of autism and autistic-like conditions found in this study compared with our previous ones is accounted for by severely mentally retarded children. The prevalence of autism and similar conditions among mildly retarded and normally intelligent children is almost exactly the same as in the previous studies.

There were slight (though statistically insignificant) differences in prevalence between urban and rural areas, with urban prevalence being higher. Whether these findings reflect real differences or dissimilarities with regard to coverage and assessment facilities is not yet known. This difference in prevalence does not accord with previous suggestions (Gillberg, 1984) that autism might be more common in the countryside than in cities.

The tendency for 'autism' to be a more common diagnosis than 'autistic-like conditions' (or 'other childhood psychoses') is probably a result of increasing awareness of the early symptoms, leading to early referral. As in our previous studies, our diagnoses of infantile autism did not rely on medical records except to confirm the presence of central symptoms before age 2½ years.

We would argue for the abolition of the term 'other childhood psychoses' when referring to autistic-like conditions: the major clinical signs and symptoms in these groups of children really do qualify them for the description of 'autistic-like'. True, there exist symptoms not typically seen in autism (such as confusion and extremely clinging behaviour) and the onset is usually somewhat later than in infantile autism, but the similarities are really overwhelming.

The boy:girl ratio is considerably higher in this study than in our previous ones. An overall ratio of 2.7:1 was found this time, compared with 1.8:1 four

years ago. Infantile autism was found by this study to be more than five times as common in boys as in girls, whereas autistic-like conditions tended to be only marginally more common among boys. As in Lorna Wing's study (Wing, 1981) and our own previous studies, there was a clear trend towards decreasing boy:girl ratios with decreasing IQ. In spite of our efforts to reach all possibly autistic children in the areas studied, we are aware that we might have missed some very young autistic children and a few with very high IQ levels. We do not believe, however, that any considerable fraction of mentally retarded children with autism or autistic-like conditions could have been missed.

A third of the IA and half of the AC children had obvious clinical organic signs such as epilepsy, tuberous sclerosis, neurofibromatosis and the fragile (X) syndrome. Almost all the autistic children showed clinical, radiological, neurophysiological or neurochemical signs indicative of central nervous system damage/dysfunction (Gillberg, 1985). These results are in sharp contrast with those presented in the early days of infantile autism, when the disorder was supposed to be psychogenic.

Social class did not differentiate either of the index groups from a comparison group of primary-school-age children without major handicaps. If anything, there was a trend (statistically insignificant) towards infantile autism being relatively more common in low social classes. This result accords

well with modern studies of social factors in infantile autism (Wing, 1980; Gillberg & Schaumann, 1982; Bohman *et al.*, 1983). There is now a convincing body of evidence from population-based studies that infantile autism is *not* associated with high social class, and we hope that the fruitless arguments about this issue will finally come to an end.

Finally, we would like to draw attention to the fact that there exist other children, far outnumbering those in the present study, with 'autism spectrum' problems or 'autistic traits'. These have been referred to as children with 'psychotic behaviour' (Gillberg, 1983a, 1983b) or 'the triad of language and social impairment' (Wing & Gould, 1979). Among severely mentally retarded children these traits occur fairly often (Gillberg *et al.*, 1986) and they are also seen frequently in children with moderate degrees of motor-perceptual disturbance. No attempt was made in this study to detect all such cases.

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Two Stage Design in a Community Survey

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By comparison with the PSE-ID system, we showed that the GHQ-60 could be used with good results as a screening instrument in the first of a two stage community survey. Unlike the sensitivity and negative predictive values, the specificity and positive predictive rates reach low figures in this study. The global efficiency and positive and negative predictive values of the test are significantly improved by raising its cut-off score, but at the expense of great reduction in sensitivity. The revised scoring system failed to provide better prediction of 'caseness' than conventional scoring. The GHQ does not distinguish fully between persons in the community afflicted by 'transient states of distress' and those whose symptoms would classify them as 'cases' by the PSE.

There is a widespread consensus that statistics derived from patients under treatment give a very incomplete estimate of the total psychiatric morbidity of the community, because of the many factors affecting the recognition and declaration of illness. To overcome this bias, a measurement of untreated morbidity is required, and a very appropriate research strategy for such measurement is the two stage screening survey (Eastwood, 1971; Vázquez-Barquero, 1978; Duncan-Jones & Henderson, 1978; Deming, 1977). In the first stage, the population under study is classified, usually by means of a questionnaire, in terms of the probable presence or absence of psychiatric pathology. In the second, a variable proportion of 'probable cases' and at times also of 'non cases' has to be examined by a psychiatrist, in order to make a definitive diagnostic evaluation.

The decision to integrate psychiatric screening into the community surveys has the implication of having to select for the analysis of morbidity two different instruments, (screening questionnaire and psychiatric interview), whose efficiency and reliability should be estimated by testing them jointly. Widely recommended instruments are the General Health Questionnaire, GHQ (Goldberg, 1972; Goldberg, 1978) and the Present State Examination, PSE (Wing *et al.*, 1974).

The GHQ was originally designed for identifying mental illness in general practice (GP). It has been shown to possess high levels of validity in different medical settings (Goldberg, 1972, 1978; Maguire *et al.*, 1974; Ballinger, 1975; De Paulo *et al.*, 1980; Nott & Cutts, 1982; Hobbs *et al.*, 1983). These high validity figures have also been confirmed by us in two

Spanish medical samples (Muñoz *et al.*, 1978; Vázquez-Barquero *et al.*, 1985). In all these, however, the diagnosis of 'case' was established through the Clinical Interview Schedule, CIS (Goldberg *et al.*, 1970). This interview, although capable of achieving high reliability, is reported to produce a lower threshold for case identification than the one used with the PSE-ID system. This appears to be due to the fact that these two instruments were developed in completely different ways. While the concept of 'case' incorporated in the CIS was derived from primary care patients the PSE was based on psychiatric in-patient populations; therefore, if one wishes to obtain comparable data, it appears necessary to use a higher cut-off point for the GHQ, when used alongside the PSE-ID (Finlay-Jones & Murphy, 1978; Goldberg, 1981).

Different studies have tested the GHQ using the PSE-ID as external criterion of validity. Newson-Smith & Hirsch (1979) reported a highly significant correlation (0.80) between the two instruments in interviews with self-poisoning patients. Finlay-Jones & Murphy (1979) validated the GHQ-30 in two samples of women in a GP setting. They found that compared with the shortened version of the PSE, the GHQ misclassified 26% of respondents. False positives were suffering from physical illness, loneliness, and recent life events, while false negatives tended to be women with chronic psychiatric disorders mainly anxiety. Rabins & Brooks (1981), relying on a subjective evaluation of information gathered with the PSE, found that the GHQ-28 was a valid instrument (correlation coefficient: $r=0.83$; sensitivity: 92%; specificity: 92%) for detecting emotional morbidity in patients attending a multi-

ple sclerosis clinic. Robinson & Price (1982), used a modified version of the PSE, in a sub-sample ($n=30$) of 103 patients attending a stroke clinic. They found that the total score of GHQ-28 correlated highly with a depression scale derived from the PSE ($r=0.88$). In a recent study by Byrne (1984) 211 women referred to a gynaecological out-patient clinic were screened for psychiatric disorders on the GHQ-60. A random sub-sample ($n=35$) of these women were interviewed, at a second stage, using the PSE. In this group, the specificity of the GHQ was 86%, the sensitivity 67%, false positives 20%, and the overall misclassification rate 26%.

Two major validity studies tested the screening capacity of the GHQ in general population samples. Banks (1983) sampled 200 17 year olds; he confirmed that different versions of the GHQ correlated highly with the PSE-ID. Duncan-Jones & Henderson (1978) incorporated both instruments into a two-stage strategy of analysis of a general community sample in urban Canberra. They found a high correlation between PSE and GHQ scores (0.76); however, a certain number of high scorers on the first stage were not confirmed as 'cases' by the PSE on the second stage. This was thought to be partly related to applying a more stringent criterion of caseness than the one used in the original Australian validity study (Tennant, 1977), and partly due to the fact of some respondents changed their illness status from ill to well during the two-stage interval. It was concluded that the combined use of these two instruments provides a rigorous description of morbidity but an upward revision of the GHQ cut-off score was recommended when used alongside the PSE-ID system.

We describe here an attempt to test the performance of the GHQ-60 as a screening instrument against the PSE-ID in a two-stage community survey; this is part of a mental health survey, which we are conducting in a sea-side rural community of Santander (Cantabria), Spain.

Method

Design

The design was based on a two stage (screening questionnaire and psychiatric interview) cross-sectional analysis of a representative sample of the population studied.

Description of the area

The area under study consists of three adjoining counties, on the eastern side of the bay of Santander. This area extends for approximately 105 km² and has a current population of approximately 9,250. This community was

chosen to represent the non-urban coastal part of the province; it has a mixture of dairy farming, fishing, light industry, and tourist activities.

Subjects

(a) First stage

A random sample of 1250 names was drawn from an electoral register of the three counties, and thus exclusively of persons over 17 years of age. Special care was taken to make sure that each name had equal chance of being selected.

At this stage, all the subjects were visited by a team of interviewers, who were specially trained final year medical students. During a single session, each person was asked to complete the GHQ-60 items, as well as a range of medical and social questionnaires.

The GHQ score was calculated by using the non-weighted GHQ method: the cut-off score of 11 was used, above which the patient is considered to be a 'probable psychiatric case' (Goldberg, 1978). Below-threshold schedules were allocated into two different batches in terms of GHQ score, giving a breakdown of the population into 0-5 and 6-11 scorers.

(b) Second stage

The criterion for the selection of subjects to be included at this stage was: (i) All subjects who scored 12 or more, on the GHQ ('probable positives') in the first stage. (ii) An approximately equal number who scored below 12 ('probable negatives'); this second group was selected at random from each batch of below-threshold schedules returned from the first stage. The description in numerical terms of this sampling method is shown in Table I.

The subjects selected at this stage were interviewed in their homes within the following month (between 23 and 45 days after; $\bar{X}=34.2$, S.D.=5.37) by a team of psychiatrists, using the ninth edition of the PSE (Wing *et al*, 1974). They had been trained in the use of the PSE by medical staff from the MRC Social Psychiatry Unit at the Institute of Psychiatry, London. At the time of the interview the psychiatrists were unaware of each persons' answers to the GHQ and to other psychopathological instruments.

The PSE data were analysed using the CATEGO program (Wing *et al*, 1974) and the Index of Definition, ID (Wing *et al*, 1978). The ID allows eight degrees of definition of symptomatology to be established; at the threshold and definite levels (5-8), disorders are sufficiently well defined as to be considered pathological. Subjects could also receive a CATEGO diagnosis.

Because of the stratified sampling in the second stage, the prevalence figures have to be weighted to reconstitute the original sample (Table I). This was done by applying the following mathematical formula:

$$\text{Prevalence} = \frac{\sum \frac{e_i n_i}{m_i}}{N}$$

e_i = number of cases detected in each of the three batches of GHQ score (0-5; 6-11; ≥ 12) for the group under analysis.

n_i = number of persons in the first stage sample in each batch of GHQ score for the group under analysis.

m_i = number of persons in the second stage sample in each batch of GHQ score for the group under analysis.

N = total number of persons in the first stage sample for the group under analysis.

Chi-square analysis was used to compare rates, while Student's *t*-test was performed to compare groups mean values.

Results

Table I shows the distribution of subjects in the first and second stages, according to their sex and GHQ score. Six females scoring 12+ on the GHQ, as well as some others 'low scorers' (three female, seven male) refused to collaborate in the second stage, and had to be substituted. It was also necessary to replace three more 'low scorers' (two male, one female) because of incomplete data. All

the substitutes were selected randomly from the group of persons scoring below threshold at the first stage. In spite of the substitutions, a proportional representation of the two levels of low scorers detected in the first stage was maintained in the second.

Psychiatric morbidity

The measurement of morbidity by the PSE-ID system found in the second stage for the different GHQ scores is shown in Table II: 149 (32.7%) persons had no symptoms (ID level = 1), 181 (40.3%) presented symptoms between ID levels 2 and 4, and 122 (27.0%) could be classified as 'psychiatric cases' (ID levels 5-8). For this latter group, it is appropriate to use the CATEGO programme to make clinical diagnosis (Wing *et al*, 1974). Table III shows the diagnoses found for this sample in each level of GHQ score. Because the population investigated in the second stage was not a true probability sample, the prevalence frequencies had to be adjusted according to the probability of selection. The total weighted prevalence found in this sample was 14.7%, in which, female rates are substantially above the male rates (male=8.11%; female=20.58%).

TABLE I
Distribution of GHQ scores in the two-stage sample

GHQ Score	First stage			Second stage		
	Male	Female	Total	Male	Female	Total
12+	68	159	227	68	151	219
6-11	85	136	221	16	31	47
0-5	428	347	775	93	93	186
Total	581	642	1223	177	275	452

TABLE II
Results of PSE-ID levels in relation to GHQ scores

PSE Index of definition	0-5		GHQ scores 6-11		12+	
	Male	Female	Male	Female	Male	Female
1 No symptoms	64	49	6	5	12	13
2 Non-Specific symptoms	24	30	5	19	20	33
3						
4 Specific symptoms	3	7	2	3	14	21
5 Borderline	1	4	2	2	16	47
6						
7 Cases	1	3	1	2	6	37
8						

TABLE III
Results of PSE-CATEGO diagnosis in relation to GHQ scores

PSE-CATEGO Diagnosis	CIE Diagnosis		Number of Cases			% Weighted prevalence	
			GHQ-scores 0-5	6-11	12+		
A+, B+,	Anxiety N. Phobic N. Obsessive N. (300.0; 300.2; 300.3)	Male	1	1	5	2.56	7.82*
		Female	5	3	45	12.34	
R+, N+,	Depression (300.4; 296.2)	Male	1	1	15	4.29	6.11*
		Female	2	1	36	7.75	
M+, \	Manic psychosis (296.1)	Male	0	0	1	0.17	0.08*
		Female	0	0	0	—	
S+, P+, O? X,	Schizophrenic Psychosis paranoid state (295.3; 297.9; 299)	Male	0	1	1	1.09	0.73*
		Female	0	0	3	0.49	
	Total Morbidity	Male	2	3	22	8.11	14.74*
		Female	7	4	84	20.58	

*Total prevalence.

These morbidity figures are mainly accounted for by depressive and anxiety symptomatology. It was apparent that in this community, women tended to develop more anxiety and men more depression.

Validation analysis of the GHQ

In view of the fact that the PSE-ID system appears to produce a higher threshold for case identification than the Clinical Interview Schedule, which was the one used in the Spanish original validity study of the GHQ (Muñoz *et al.*, 1978), it seemed pertinent to test the performance of the GHQ on different cut-off points. Table IV shows the number of subjects according to whether they were defined as 'normal' or 'psychiatric case' on the 4/5 ID threshold score, and on different cut-off points for the GHQ. The statistics derived for males and females using these scores are in Table V; with a cut-off value of 11/12, the sensitivity for both sexes is superior to 80%, while the specificity drops, especially in women, below 70%. Accordingly, while the negative predictive value reaches very high

figures in both sexes the positive predictive rates are, especially in males, quite low. By raising the cut-off point in the GHQ to 19/20, it is possible to obtain a progressive reduction of the overall misclassification rate, together with significant rises of the positive predictive values. At this maximum cut-off point, the negative predictive value is still agreeably high. This improvement of the efficiency of the GHQ when the cut-off point is raised to 19/20 is achieved at the expense of decreased sensitivity and increased false negative rates. The differences which appear in these rates between the two sexes are not statistically significant.

There is a widespread opinion that the criteria used for case identification on the PSE are far more stringent than those applied with the CIS. This implies that we would classify as 'normal' those whose symptoms would have been sufficient to be included as cases in the original validity studies. If this were so, one would expect that by lowering the threshold of normality on the PSE-ID system to 3/4 (ID level: 1-3 = 'non case'; 4-8 = 'case'), we would improve the validity of the GHQ. Table VI shows that by

TABLE IV
GHQ scores for different cut-off points related to psychiatric assessment by the PSE-ID system

GHQ Score		Male (n = 178)		Female (n = 274)		Total Sample (n = 452)	
		Psychiatric assessment (PSE-ID)		Psychiatric assessment (PSE-ID)		Psychiatric assessment (PSE-ID)	
		Normal (ID = 1-4) n = 152	Psychiatric case (ID = 5-8) n = 26	Normal (ID = 1-4) n = 178	Psychiatric case (ID = 5-8) n = 96	Normal (ID = 1-4) n = 330	Psychiatric case (ID = 5-8) n = 122
Cut-off score 11/12	'High score' n = 220	46	21	68	85	114	106
	'Low score' n = 232	106	5	110	11	216	16
Cut-off score 13/14	'High score' n = 177	34	17	55	71	89	88
	'Low score' n = 275	118	9	123	25	241	34
Cut-off score 15/16	'High score' n = 142	22	16	39	65	61	81
	'Low score' n = 310	130	10	139	31	269	41
Cut-off score 19/20	'High score' n = 91	11	11	18	51	29	62
	'Low score' n = 361	141	15	160	45	301	60
Cut-off score 20/21	'High score' n = 80	9	11	16	44	25	55
	'Low score' n = 372	143	15	162	52	305	67

TABLE V
Validity statistics of the GHQ for different cut-off scores

	11/12			13/14			GHQ cut-off scores 15/16			19/20			20/21		
	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T
Sensitivity	80.8	88.5	86.9	65.4	73.9	72.1	61.5	67.7	66.4	42.3	53.1	50.8	42.3	45.8	45.1
Specificity	69.7	61.8	65.5	77.6	69.1	73.0	85.5	78.1	81.5	92.8	89.9	91.2	94.1	91.0	92.4
Positive predictive value	31.4	55.5	48.2	33.4	56.3	49.7	42.1	62.5	57.0	50.0	73.9	68.1	55.0	73.3	68.8
Negative predictive value	95.4	90.9	93.1	92.9	83.1	87.6	92.8	81.8	86.7	90.4	78.1	83.4	90.5	75.7	82.0
Misclassification Rate	28.7	28.8	28.8	24.2	29.2	27.2	18.0	25.5	22.6	14.6	23.0	19.7	13.5	24.8	20.4

1. M = male, F = female, T = total

TABLE VI
Validity figures for the GHQ after lowering the threshold score of 'caseness' on the PSE-ID system ('normality': ID=1-3; 'psychiatric case': ID=4-8)

GHQ Cut-off score 11/12	Psychiatric Assessment PSE-ID					
	Male n = 178		Female n = 274		Total Sample n = 452	
	'Normal' (ID=1-3) n = 133	'Case' (ID=4-8) n = 45	'Normal' (ID=1-3) n = 146	'Case' (ID=4-8) n = 128	'Normal' (ID=1-3) n = 279	'Case' (ID=4-8) n = 173
'High score' (GHQ = 12-60)	32	35	46	107	78	142
'Low score' (GHQ = 0-11)	101	10	100	21	201	31
Sensitivity		77.8		83.6		82.1
Specificity		75.9		68.5		72.0
Positive predictive value		52.3		69.9		64.6
Negative predictive value		90.9		82.6		86.6
Misclassification rate		23.6		24.5		24.1

doing this, it is possible to increase the specificity to 72.0% and the positive predictive value to 64.6%, without making the sensitivity and negative predictive rates much worse. These variations of the validity statistics are apparent in both sexes.

The correlation between GHQ scores and PSE-ID ratings was 0.61 using Spearman's rank correlation. Males exhibited a slightly lower correlation coefficient than females (male: 0.56; female: 0.61). Other socio-demographic factors such as age, marital status, social

TABLE VII
Revised Scoring System for the GHQ*

Response categories	Conventional scoring	CGHQ ₁ Scoring		CGHQ ₂ Scoring	
		N-Items	P-Items	N-Items	P-Items
1. Not at all	0	0	0	0	0
2. No more than usual	0	1	0	2	0
3. Rather more than usual	1	1	1	3	3
4. Much more than usual	1	1	1	3	3

*From Goodchild & Duncan-Jones, 1985.

TABLE VIII
Validity statistics of two new scoring systems (CGHQ₁; CGHQ₂) on different cut-off points

Scoring system	Cut-off point	Sensitivity	Specificity	Mis-classification Rate
CGHQ ₁	27/28	79.5	68.5	28.5
	29/30	74.6	73.0	26.5
CGHQ ₂	68/69	82.8	71.2	25.7
	73/74	77.9	74.2	24.8

class, educational level, or unemployment did not significantly influence the validity statistics of the GHQ.

Analysis of new scoring systems for the GHQ

In an attempt to overcome the reported inadequacy of the GHQ to detect chronic neurotic illness Goodchild & Duncan-Jones (1985) proposed two new scoring systems (CGHQ₁ and CGHQ₂). These revised systems are based on the assumption that the response 'no more than usual' to items describing a symptom might indicate chronic illness, although conventionally it would be scored as healthy. They differentiated a set of items describing pathology ('N-Items') from others describing positive or healthy states ('P-Items'). On the GHQ-60, these two sets of items are composed by: 'P-Items': 1, 7, 15, 16, 21, 26, 27, 28, 30, 31, 32, 33, 35, 37, 42, 46, 53, 54; 'N-Items' the 42 remaining items.

On the different scoring systems, the four response categories for each item of the 'N-Items' and 'P-Items' sub-sets are scored according to the rules describe in Table VII.

Table VIII shows the validity statistics of the new scoring on different cut-off points. The cut-off scores where the validity indices are most nearly equal are, for the CGHQ₁ 29/30, and for the CGHQ₂ 73/74. The validity indices derived in this way are almost equivalent to the ones obtained with the conventional GHQ scoring at the level 13/14. In view of the relevance of reaching a higher sensitivity ratio, for epidemiological purposes, the cut-off points for these revised systems could alternatively be placed at the levels 27/28 (CGHQ₁) and 68/69 (CGHQ₂) for a two-stage community survey. The validity statistics derived from these cut-off scores are comparable to those obtained from the conventional GHQ scoring at the level 11/12. The similarity of the screening efficiency of the GHQ on these scoring systems is confirmed by Spearman's rank correlation of the GHQ score on each of the three systems with the PSE-ID score (PSE-GHQ=0.61; PSE-CGHQ₁=0.52; PSE-CGHQ₂=0.59). It appears, therefore, that the proposed modifications of the GHQ scoring systems do not significantly improve the detection of psychiatric morbidity in our data base.

False positives (GHQ cut-off score 11/12, PSE-ID threshold score 4/5)

In this study, 114 persons (46 male and 68 female) were classified as 'false positive' and it is relevant to try to identify the factors that contribute to this status by comparing 'false positive' with 'true negative'. This applies to comparisons of age distribution, sexual differences, marital status, work situation, and presence of physical complaints. We found more false positives in the widowed ($X^2=11.3$; $df=1$; $P<0.001$), in older ages (false positive: mean age=52.0; $SD=18.3$; true negative: mean age=46.9; $SD=18.5$; $t=2.39$; $P<0.05$) in persons out of work ($X^2=7.4$; $df=1$; $P<0.001$) and with higher mean levels of physical complaints ($t=11.70$; $P<0.001$).

To test whether the time interval between the GHQ and the PSE could contribute to these high figures of 'false positives', by allowing some transient symptomatology to be spontaneously resolved before the second stage interview, we compared the 'false positive' groups with the remaining validity groups in terms of their time intervals. The mean time interval between GHQ and PSE assessment for the 'true positive' group was 34.1 days ($SD=5.0$), which was significantly lower than that of the 'false positive' group ($\bar{X}=35.7$ days, $SD=5.44$; $t=2.26$; $P<0.01$). The mean time interval in the 'true negative' group was also significantly lower ($\bar{X}=33.24$ days, $SD=5.60$) than that of the 'false positive' ($t=3.87$; $P<0.001$), but this did not happen in the case of the 'false negative' group (time interval: $\bar{X}=34.2$ days, $SD=5.46$; $t=1.00$; NS). The significance of time interval differences held also good when 'false positives' were compared with the sum of the remaining groups 'remaining groups' time interval: $\bar{X}=33.52$ days, $SD=5.41$; $t=3.38$; $P<0.001$).

False negatives (GHQ cut-off score 11/12, PSE-ID threshold score 4/5)

Sixteen persons (five male, eleven female) were classified as 'false negatives'. As Table III shows, ten of these belonged to the diagnostic category of 'anxiety states', five to that of depression, and one to schizophrenia. Among the 'anxiety states' there was higher tendency, not statistically significant, to produce 'false negatives'. Earlier studies suggest that defensiveness and chronicity of psychiatric symptomatology are the most important factors in determining 'false negative' status. We found only three of our 'false negative' patients were afflicted by long-standing psychiatric symptomatology, while six patients were suffering from long standing physical complaints. We did not find any significant association between 'false negative' and social or demographic factors.

Discussion

The psychiatric morbidity found in this community was much lower than that previously found with a similar design in the Baztan valley (Vázquez-Barquero *et al*, 1981; 1982). Although there were evident sociological differences between these two rural Spanish communities, one could postulate that

these differences were mainly due to having used two different psychiatric interviews: the Clinical Interview Schedule in the Baztan valley and the Present State Examination in this community. This is also supported by the fact that our present figures are similar to the ones detected by other authors applying the PSE to quite different communities (Henderson *et al*, 1979; Bebbington *et al*, 1981).

We demonstrated in this study that contrary to what has been previously stated (Tarnopolsky *et al*, 1979; Benjamin *et al*, 1982), the GHQ is a valid instrument for screening possible cases in the initial step of a two-stage community survey. The good performance of the GHQ in this situation is demonstrated by the high sensitivity rate reached in this study (above 0.80 in all cases), which guarantees the ability of the GHQ not to miss possible psychiatric cases in the second stage.

However, unlike the sensitivity and false negative values, the specificity and false positive values are considerably poorer in this study. We applied the PSE as the external criterion of caseness, which is reputed to use a more stringent criterion for case identification than that used by the CIS. One should therefore expect that raising the cut-off point on the GHQ or lowering the threshold of 'caseness' on the PSE-ID, would have significantly reduced the proportion of 'high scorers' in the GHQ which were evaluated as 'non cases' on the PSE, and, to a certain extent, this happened. However, in spite of these manipulations of the thresholds, we are still left with high figures of 'undetected high scorers', suggesting that the relevant point here may not be the severity, but rather the quality, of the symptomatology. It therefore appears that the GHQ fails to distinguish between clinical states such as the ones classified as 'cases' or even 'specific psychiatric symptoms' by the PSE, and other types of psychiatric complaints. These psychiatric manifestations may be equivalent to what other authors have defined as 'transient distress symptoms' (Finlay-Jones & Murphy, 1979; Corser & Philips, 1978).

An additional methodological explanation for our high figures of 'undetected high scorers' is that the time which elapsed between the completion of the GHQ and the psychiatric interview could have allowed some transient psychiatric morbidity to resolve spontaneously and not be reported at the interview. There is support for this (Finlay-Jones & Murphy, 1979; Corser & Philips, 1978) and it is possible to suggest that the GHQ detects as 'cases' some individuals whose symptoms are very transient. The relevance of transient symptomatology in an untreated population has also been substantiated by Tennant *et al* (1981) in the second Camberwell

survey, in which it was shown that 49% of cases identified at a first interview were no longer cases a month later while we demonstrated that the 'false positive' rate is increased significantly by the time interval between GHQ and PSE assessment. This finding strengthens the conviction that transient disturbances are detected by the GHQ, and also throws some light on the impact of the time interval on the application of a two-stage design in a community survey.

Shrout & Fleiss (1981) stated that analysis of the performance of a screening test should not stop with its sensitivity and specificity since, we do not know who is truly a case and who is not. The rates that indicate the correctness of classification are the positive and negative predictive values, which depend not only on the validity coefficients of the test, but also on the prevalence of the disease in the population. The cut-off score where the GHQ reaches its maximum efficiency, as measured by these values, is the 19/20; at this level, we obtained a overall mis-classification rate of 19.7%, together with a reasonably balanced positive and negative predictive rates (68.1% and 83.4% respectively). Raising the threshold of caseness from 11/12 to 19/20, when using the GHQ-60 alongside the PSE-ID system, is convenient (Goldberg, 1981), but the gain we reported in the efficiency of the test was achieved at the expense of a considerable drop in the sensitivity rate. In order not to miss a high proportion of 'cases' in the second stage of a two stage community survey, we have to choose a lower cut-off score, and thus be prepared to tolerate a much worse positive predictive value.

Our findings show that contrary to previous reports (Tarnopolsky *et al.*, 1979; Hobbs *et al.*, 1984), sex differences do not introduce a significant degree of variation in the correlation coefficient between the GHQ and the PSE, and the same is true of the remaining validity figures. Since the performance of the GHQ is not significantly affected by the social background of the respondents, socio-demographic factors do not significantly influence the screening capacity of the GHQ in the community.

False positive status was characterised by a predominance of females, widowed marital state, old age, unemployment, and higher levels of physi-

cal complaints. This tendency of false positives to congregate among distressed persons with adverse life events or physical illness is in agreement with the findings of Finlay-Jones & Murphy (1979) and of Corser & Philips (1978); it raises again these authors' reservation about the ability of the GHQ to distinguish psychiatric disorders from transient states of distress.

When the GHQ is used to screen for possible cases in the initial step of a two-stage design, the most relevant quality is the ability to minimize false negatives, because they represent cases that are lost for the survey. Contrary to those of Benjamin *et al.* (1982), our results demonstrate that this is not an important source of bias in the community. It has been said repeatedly that the most important factor in determining false negative status is, at least in consulting settings, the chronicity of psychiatric symptoms, but our results, although inconclusive, support the opinion of Goldberg (1978) that this is not an important source of misclassification in the community.

Greater weight is provided to chronicity in the new GHQ (CGHQ₁; CGHQ₂) scoring systems (Goodchild & Duncan-Jones, 1985). This revised scoring provides better prediction of morbidity than the conventional GHQ scoring, and advocates suggest that it be adopted for epidemiological research, but its use did not improve the screening capacity of the GHQ in our data. This may be because the aim of the new system is to give a more accurate reflection of chronic symptomatology and not of 'transient states of distress', which were the sort of conditions which appeared to provoke the higher percentage of mis-classification in our study, whether these new scoring systems improve screening in clinical settings should be tested in an appropriate study.

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Body Size Estimation in Bulimia

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Body size estimation was studied in 22 patients with bulimia and 20 normal controls. Two methods of body size estimation were used, a distorting television image method (DTIM) and the image marking method (IMM). The subjects estimated body size of a dummy as well as their own body on the DTIM. When estimating body size on the DTIM the bulimics overestimated and the controls underestimated, there being a significant difference between the two groups. There was no difference between the groups when estimating the size of the dummy. On the IMM a significant difference was found between the bulimic and control groups, the bulimics overestimating body size and the controls being more accurate. Marked directional effects were found with the DTIM. The two methods of body size estimation are compared.

Bulimia is an eating disorder which has been operationally defined in DSM-III (APA 1980). The clinical features described in the DSM-III diagnostic criteria include recurrent episodes of binge eating, severely restrictive diets, self-induced vomiting and abuse of laxatives or diuretics. The patient experiences depressed mood following eating binges and is aware that the eating pattern is abnormal. If the bulimic episodes are due to anorexia nervosa the patient would be excluded from a DSM-III diagnosis of bulimia. Bulimia nervosa was described by Russell (1979) as a later sequel of anorexia nervosa. More recently he has stated "in 'true' bulimia nervosa there is a history of a previous episode of anorexia nervosa, possibly of minor severity" (Russell, 1985). Bulimia nervosa is generally considered a disorder of greater severity than DSM-III bulimia.

There have been numerous studies of body size estimation in anorexia nervosa prompted by Bruch's (1962) proposal of a disturbance of body image in the condition. Methods used to examine body size estimation can be divided into those that measure 'body part' estimation such as Slade and Russell's (1973) visual size-estimation apparatus and 'whole body' methods such as the distorting photograph method (Garner *et al.* 1976). Studies measuring 'body part' estimation have found an overestimation of body size in anorexia nervosa (Slade & Russell 1973, Button *et al.* 1977, Pierloot & Houben 1978, Crisp & Kalucy 1974, Garner *et al.* 1976, Casper *et al.* 1979, Fries 1977). However, most studies have found a similar degree of overestimation amongst controls. Using a 'whole body' method Garner *et al.* (1976) found that controls

underestimated body size, whereas anorexics produced a mean score near the non-distortion position, the difference being significant. More recently Touyz *et al.* (1984a) using a similar method also found anorexics to be on average accurate in estimating body size, there being no difference from controls.

Bulimics have abnormal attitudes towards food, eating and body weight in common with patients with anorexia nervosa. Body image disturbance has also been described clinically in bulimia nervosa with virtually all patients in one series reporting feeling persistently fat (Fairburn & Cooper 1982). Body size estimation in bulimia has been examined by Freeman *et al.* (1984) using a modified video camera technique. Significant differences were found between bulimic and control groups, with patients with bulimia nervosa overestimating body size. This study examines body size perception of patients with bulimia using both 'whole body' and 'body part' methods as the two methods have produced different results in body size estimation in anorexia nervosa. The 'whole body' method used was a distorting television image method (DTIM), similar to that described by Allebeck *et al.* (1976). It is essentially the same as the technique used by Freeman *et al.* (1984), the only difference being the equipment used and the degree of distortion of body size achieved. It has been shown to be a reliable procedure with an internal consistency coefficient of 0.62 (comparing frontal and profile body image scores) and a high test, retest reliability ($r=0.90$). The 'body part' method used was the image marking method (IMM) introduced by Askevold (1975), which has an internal consistency coefficient of 0.60 (Garner & Garfinkel 1983).

Method

Subjects

The experimental group consisted of 22 patients with bulimia, all fulfilling DSM-III criteria. Of these, 20 also fulfilled Russell's criteria for bulimia nervosa. Twenty-one were out-patients consecutively referred to the Edinburgh Eating Disorders Clinic at the beginning of their treatment and one was an in-patient. Nine patients were referred by their general practitioner, four by a psychiatrist or psychologist and one by a self-help group; Eight were self-referred. The control group comprised of 20 subjects who were trainee medical and paramedical staff. It was decided that control subjects who were 25% underweight or overweight would be excluded for these reasons.

All subjects completed the Eating Disorder Inventory (EDI) produced by Garner *et al* (1983). The subjects were required to wear a medium blue leotard for both methods of body size estimation. While it was inevitable that all subjects were aware that body size estimation was being measured the subjects were not aware of the aims of the study or the findings up to that point in time.

The Distorting Television Image Method (DTIM)

The apparatus is illustrated schematically in Figure 1. The subject stood 5 metres from a National WV.341N video camera (focal length of lens=75 cm) against a neutral background, and illuminated with a video light.

They were able to view an image of themselves on a black-and-white television monitor to their left. The conductor (AW or AA) could narrow or widen the television image electronically, using control apparatus which varies the voltage across the horizontal and vertical axis of the television monitor. The vertical control was set so as to achieve a 50% degree of distortion in a positive or negative direction on the horizontal axis. The conductor could view the distorted image on a second monitor to ensure a smooth change of distortion, and could read the voltage on the horizontal axis from a voltmeter with a digital read out. The system had previously been calibrated producing a graph of voltage against degree of distortion. The degree of distortion of the image could therefore be obtained from this graph using the voltage results.

The conductor adjusted the television picture to its narrowest and gradually widened it. The subject stopped the conductor verbally when they felt that the image was a correct reproduction of themselves. The television picture was then set to its widest and gradually narrowed, the subject again indicating when the image was accurate. This was necessary as the pilot study had shown marked directional effects, that is when the image was initially narrow the subject tended to reproduce a narrow image and when initially wide the subject tended to reproduce a wide image. The two trials were repeated and the scores averaged to give a composite score. The video camera was next focused on the subject's face and exactly the same procedure was carried out. A white shop window dummy was then placed in front of the video camera, on the spot the subject had

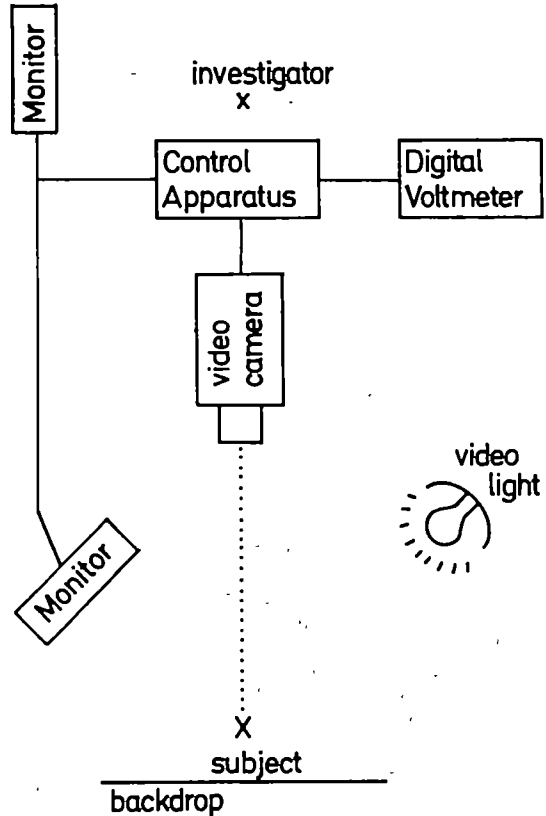


Fig. 1 Illustration of distorting television image apparatus.

been standing. The subject was then required to examine the dummy for 20 seconds and was asked to indicate the correct reproduction of the dummy on the monitor, again over four trials.

The Image Marking Method (IMM)

This method is described in detail by Askevold (1975). The subject stands at arm's length from a white board. They are asked to imagine standing before a mirror, looking at themselves. The investigator stood behind the subject and touched body points on the subject's shoulders, waist and hips. The subject marked on the board where they felt these points would be in the imaginary mirror. The actual widths were then recorded.

Results

The mean age of the patients with bulimia was 24.9 years (s.d.=6.2); their mean weight on assessment was 61.3 kg (S.D.=13.5) or 105.4% (s.d.=19.1) of average weight for height (Documenta Geigy 1962). The mean lowest past adult weight for the bulimics was 52.9 kg (s.d.=9.2)

TABLE I
Clinical features and their frequency in the bulimics (n = 22)

<i>Clinical features</i>	<i>%</i>
Bulimia	100
More than 3/day	5
2-3/day	18
daily	32
More than 1/week	36
1/week	9
Self-induced vomiting	73
2-3/day	27
daily	23
2-3/week	18
monthly	5
Laxative abuse	50

and the mean highest past adult weight was 69.3 kg (s.d. = 14.2). The clinical features of the 22 bulimic patients are shown in Table I. The mean age of the control group was 25.4 years (s.d. = 5.1); their mean weight was 56.5 kg (s.d. = 5.8) or 99.4% (s.d. = 8.7) of average weight for height and age. The bulimics had a significantly higher score than controls on the following EDI subscales; Drive for Thinness, Bulimia, Body Dissatisfaction, Ineffectiveness, Perfectionism, Interpersonal Distrust and Interoceptive Awareness. There was no significant difference between the groups on the Maturity Fears subscale. Body size estimation was calculated according to the formula of Slade and Russell (1973):

$$\text{Body Perception Index (BPI)} = \frac{\text{average perceived size}}{\text{actual size}} \times 100$$

The results of the DTIM are shown in Table II. The BPI is presented separately for measurements taken when the image was initially narrow and initially wide, because there were such marked directional effects. These two scores are combined to give a composite score. If the composite scores are examined first it can be seen that when estimating the size of the dummy, the groups are equally inaccurate. However, when estimating the size of their own body, the bulimics overestimate body size, with the controls showing an underestimation. When estimating the size of their face the bulimics again show an overestimation with the controls being fairly accurate. The raw data of the whole body composite scores gives a more comprehensive picture and is shown in Figure 2. There is only one control with a BPI score of 105 or greater, whereas this is the case for 10 of the bulimics (45%), a significant difference ($\chi^2 = 6.90$ $P < 0.01$). There is a large overlap of scores between the bulimics and the controls with 27% of the bulimics underestimating body size.

When the BPI results presented separately in Table II are examined, marked directional effects are seen in both groups when estimating the size of the whole body, face and dummy. For example the whole body BPI results for the bulimics for the two directions are 90.4 and 115.7 which are significantly different ($t = 7.57$ $P < 0.001$). The whole BPI results of the controls for the two directions are 86.3 and 105.9 which are again significantly different ($t = 7.75$ $P < 0.001$). It should be noted that the difference in whole body BPI results between the bulimics and the controls is only significant when the image was initially wide. The same situation is encountered when looking at the BPI scores for the face. The difference between the groups is again only significant when the initial image presented was wide.

The results of the IMM are shown in Table III. Both groups underestimated the size of their shoulders. The bulimics overestimated waist size with the controls being accurate. Both groups overestimated the size of their hips,

TABLE II
Body Perception Index of the bulimics (n = 22) and normal control (n = 20) with the Distorting Television Image Method

	<i>Bulimics</i>	<i>s.d.</i>	<i>Controls</i>	<i>s.d.</i>	<i>t test values</i>		
<i>Whole body</i>							
Narrow image initially	90.4	7.8	86.3	7.7	1.70	NS	
Wide image initially	115.7	13.6	105.9	8.3	2.85	<i>P</i>	0.01
Composite score	103.1	8.9	96.1	7.3	2.76	<i>P</i>	0.01
<i>Face</i>							
Narrow image initially	93.7	12.6	87.4	8.8	1.89	NS	
Wide image initially	120.0	12.5	111.1	10.2	2.54	<i>P</i>	0.05
Composite score	106.8	10.7	99.2	6.6	2.80	<i>P</i>	0.01
<i>Dummy</i>							
Narrow image initially	79.0	9.0	80.0	7.4	0.39	NS	
Wide image initially	104.3	8.7	102.6	8.7	0.62	NS	
Composite score	91.6	7.8	91.3	6.7	0.13	NS	

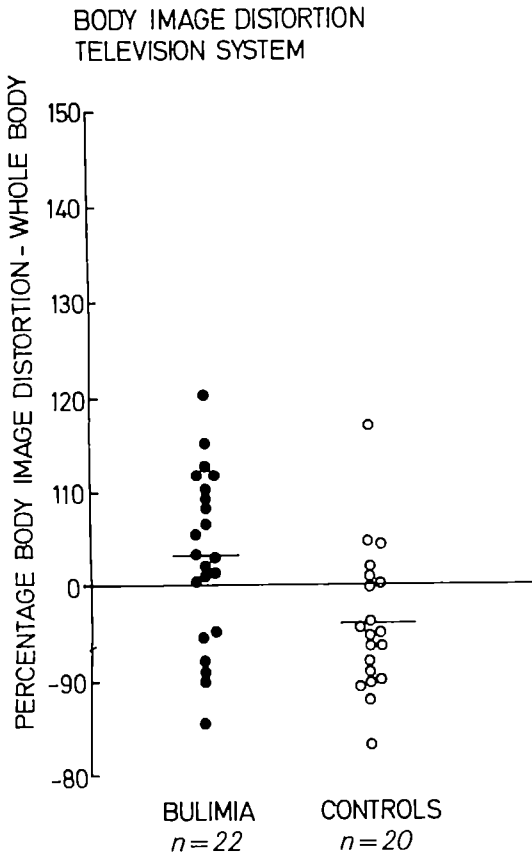


Fig 2 Body perception indices (whole body) with the distorting television image method.

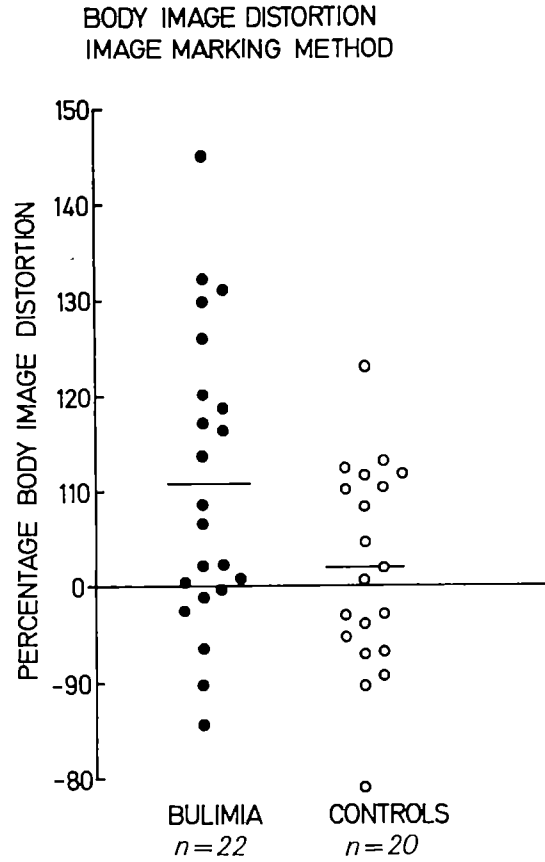


Fig 3 Body perception indices (composite score) with the image marking method.

TABLE III
Body Perception Index of the bulimics (n=22) and normal controls (n=20) with the Image Marking Method

	<i>Bulimics</i>	<i>SD</i>	<i>Controls</i>	<i>SD</i>	<i>t test values</i>	
Shoulders	97.7	12.3	94.3	11.6	0.9	NS
Waist	111.2	17.5	99.0	14.6	2.07	P 0.05
Hips	123.4	23.2	111.2	12.6	2.16	P 0.05
Composite score	110.8	15.5	102.2	10.9	2.10	P 0.05

with the degree of overestimation being significantly greater in the bulimic group. When the shoulder, waist and hip size estimation are averaged a composite score is obtained for the whole body, with the bulimics overestimating body size and the controls being more accurate. The individual composite scores are presented in Figure 3. Only one control had a BPI score of 115 or more, whereas

this was the case for nine (41%) of the bulimics ($\chi^2 = 5.60$ $P < 0.02$). There is a large overlap of scores between the bulimics and controls with 27% of the bulimics underestimating body size.

If we compare the whole body BPI scores on the DTIM with the composite score of the IMM we find that for the groups combined they score 6.9 percentage points higher

on the BPI when tested on the IMM ($t=2.71$ $P<0.01$) There is no significant correlation between the BPI results for the two methods amongst controls ($r=+0.07$, $t=0.29$ NS), but there is a significant positive correlation amongst bulimics ($r=+0.44$, $t=2.19$ $P<0.05$) Rank-order correlations between severity of bulimia and body size estimation were examined. Severity of bulimia was based on frequency of binge eating. Where this was equal amongst individuals, frequency of self-induced vomiting was examined Kendall's rank correlation coefficient was used. There was no significant correlation between severity and BPI on the DTIM ($\tau=-0.024$ NS) or between severity and BPI on the IMM ($\tau=+0.03$ NS) There was a significant correlation between Body Dissatisfaction subscale score on the EDI and the BPI on the DTIM for the bulimics ($r=+0.64$, $t=3.72$ $P<0.001$) but not for the controls ($r=+0.39$, $t=1.79$ NS). No significant correlation was found between the Body Dissatisfaction subscale score and the BPI on the IMM for bulimics ($r=+0.03$, $t=0.13$ NS) or controls ($r=+0.01$, $t=0.04$ NS).

Discussion

The results of this study confirm those of Freeman *et al* (1983) in finding a significant difference between bulimic and control groups in estimating body size with the DTIM. This was not a general perceptual disorder of the bulimic patients as they did not differ from controls in estimating the size of a dummy. A significant difference was also found between the bulimic and control groups in estimating body size using the IMM. Bulimics had therefore been shown to overestimate body size with a 'whole body' method (the DTIM) and a 'body part' method (the IMM).

The very large directional effects that were found with the DTIM are difficult to explain. Subjects are clearly strongly influenced by the initial image they are shown. Previous studies have not presented data separately in this way. Our study suggests that such detail may be extremely important. The study by Freeman *et al* (1983) found a BPI for bulimics of 107.4 compared to 103.1 in this study. Furthermore, the controls in that study had a BPI score of 103.4 compared to 96.1 in our study. In view of the marked directional effects shown, it would seem that the major reason to account for the difference between the studies is the degree of distortion achieved with each method. Freeman *et al* achieved distortions of up to 20% 'thinner' and 40% 'fatter' whereas this study was able to achieve distortions of 50% in both directions. The initial images presented to subjects in the Freeman study were biased towards the 'fatter' direction and consequently BPI scores were higher. These directional effects are

also likely to be operating in other methods of estimating body size such as the distorting photograph method (Garner *et al* 1976) and the visual size estimation apparatus (Slade & Russell 1973). In future, research workers using these methods should present data separately so that directional effects can be examined.

With separate presentation of the data it can also be seen that the difference in whole body BPI scores between the bulimic and control groups is only significant when the initial image was wide. This is also true for the difference in face BPI scores between the groups. When the bulimics are presented a 'fatter' image of their body or face they are more likely to confirm that image than are controls. This effect would not be noted if only a composite score was given and again this is likely to be of relevance in other studies.

When the DTIM a 'whole body' method is compared with the IMM a 'body part' method, it is found that the combined groups have a significantly higher BPI on the IMM. This is in keeping with studies of body size estimation in anorexia nervosa, where 'body part' methods have found anorexics to overestimate body size, whereas 'whole body' methods have not found this. This means that estimation of body size is very much influenced by the method by which it is measured which should not be surprising in view of the directional effects found within the DTIM.

The BPI scores on the DTIM showed no correlation with those of the IMM for the controls though there was a significant positive correlation amongst the bulimics. This is a similar result to that of Garner *et al* (1976) who found that the visual size-estimation method (a 'body part' method) did not correlate with the distorting photograph method (a 'whole body' method) for controls but amongst anorexics and the obese there was a significant positive correlation. Clearly subjects with eating disorders who overestimate body size with a 'whole body' method do so with 'body part' methods as well. However, it becomes difficult to argue that such overestimation represents a distorted body image because if both these methods are measuring a subject's 'body image' then you would expect to find a significant positive correlation between the two methods amongst the controls.

Hsu (1982) has argued that overestimation of body size in anorexia nervosa does not necessarily indicate a disturbed body image but may reflect some other aspect of psychopathology associated with eating disorders such as fatness phobia. It could therefore be argued that the bulimic subjects in this study expressed the way they felt about their

body by overestimating body size, without having to invoke the term 'body image'. The fact that the BPI scores on the DTIM correlate significantly with the Body Dissatisfaction subscale of the EDI would lend support to this view.

An alternative explanation for the lack of correlation of BPI scores for the two methods amongst controls could be that each method is measuring a different aspect of body image. Slade (1984) has proposed that 'whole body' methods may measure a relatively fixed cognitive attitude to body size, and 'body part' methods measure a more fluid state of body size sensitivity strongly influenced by affective and emotional factors. Longitudinal studies utilising both types of body size estimation methods would be needed to confirm this.

It would seem that the concept of 'body image' is not at present a clear one. Light may be shed on this area by Touyz *et al* (1984b) who differentiate between how a subject feels they look and how they actually see themselves when estimating body size. This distinction may prove to be of help in our understanding of the term 'body image'. At the moment, it would seem preferable to use the more easily defined term of 'body size estimation' than 'measurement of body image' when describing methods which simply measure a subjects estimation of their own body size.

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Control of Auditory Hallucinations through Occlusion of Monaural Auditory Input

MAX BIRCHWOOD

A schizophrenic patient whose severe level of auditory hallucinations had proved refractory to neuroleptic medication was given two treatment techniques derived from Green's theory that hallucinations represent verbal activity in the non-dominant hemisphere. Voice activity was markedly reduced in frequency and severity over a six-month period, and led to general improvements in interpersonal functioning. There was evidence for independent and additive effects of the two techniques

In spite of the therapeutic advances from the neuroleptic drugs, the persistence of auditory hallucinations remains a significant problem for both acute and chronic schizophrenic patients (Falloon & Talbot, 1981). Apart from the distressing nature of the experience itself, there is evidence that such symptoms may depress social functioning and in some cases underpin the formation of delusions (Wallace, 1984). There have been a number of reports of attempts to control hallucinations by behavioural strategies; but the results have been variable, and not firmly based on theory (Marzillier & Birchwood 1981).

Green *et al* (1983) suggested that auditory hallucinations represent verbal activity originating in the non-dominant cerebral hemisphere, and that the dominant hemisphere perceives this verbal activity as alien, due to a primary disassociation in the functioning of the cerebral hemispheres in schizophrenia. Green (1978, Green *et al*, 1983) reported evidence suggestive of poor inter-hemispheric transfer of complex manual and auditory information, which has been independently replicated (Carr, 1980; Hatta *et al*, 1984): this may not be a secondary (e.g. drug-induced) phenomenon (Myslobodsky *et al*, 1983), as similar results have been found in high-risk samples (Hallett & Green, 1983). Green's theory is consistent with previous observations of subvocal speech and movements of vocal musculature, concomitant with reports of "voices" (Erickson & Gustafson, 1968; Gould, 1948; Inouye & Shimizo 1970; McGugan, 1966). Green & Preston (1980) report that the content of (amplified) subvocal speech in one schizophrenic corresponded to the patient's self-reports of his voices, which were Schneiderian in character.

One prediction which stems from this theory is that if auditory input to the non-dominant hemisphere is reduced, and incompatible (dominant

hemisphere) verbalisations encouraged at the onset of the hallucinations, the voices may be disrupted. A further report (Green *et al*, 1980) records the success of this technique where auditory input is reduced using an ear-plug and the patient is taught to name things in his immediate environment at the onset of the voices; the choice of side for the ear-plug is determined by the results of an auditory comprehension (transfer) test. James (1983) has reported two cases where these techniques were used with apparent success; however, reversing the side of the ear-plug had no effect on voice reduction, which he regarded as evidence against the efficacy of the plug and as inconsistent with Green's theory. However, both techniques were employed simultaneously, so that while the side of the ear-plug was reversed, the subjects continued to use the naming technique; this confounding does not enable any effect of the ear-plug to be distinguished from the other voice control technique. More importantly, James employed an ear-plug when there was no indication from the auditory comprehension test to do so.

This paper reports a case where the effect of occluding auditory input to one ear was examined directly, in the case of a severely hallucinating schizophrenic patient.

Method

The subject

The subject was a 37 year-old male, with a two-year history of schizophrenia. At the height of the acute episode, his voices appeared to him to control everything he did or said, and to be capable of predicting his future; they instructed him to do irrational and bizarre things, which he felt he had to comply with. Following in-patient treatment, his overall presentation became more rational, and he reported that he had regained control over his thoughts

and behaviour. Following discharge he was maintained on neuroleptics (clopixol, 400 mg, fortnightly; orphenadrine, 50 mg t.d.s. and pimozide, 4 mg nightly). He lived in lodgings, and attended a work unit as a day patient, but still had continuous and severe hallucinations. His voices occurred at a very high frequency, were brief but highly repetitive in nature, and were perceived in a high-pitched male voice; they addressed him in the third person. His insight was fragmentary, and he sometimes believed the voices came from a previous life; he found the voices very distressing, and frequently shouted back at them. They occurred independently of any event or activity, but he volunteered the information that talking aloud seemed to suppress them.

Assessment

Green's Auditory Comprehension Test (Green, 1984) consists of a number of short stories of varying length and complexity, delivered under monaural or binaural listening conditions; the measure of comprehension is the number of items correctly recalled. The present subject's results were: right ear—60 items correct; left ear—35 items correct; both ears—63 items correct.

This indicates a significant left ear deficit in language comprehension, consistent with previous research results, and suggests a deficit in hemispheric integration (Green *et al.*, 1983). However, a binaural deficit did not emerge. Pure-tone audiometry indicated no hearing loss in either ear.

Intervention and Results

The subject was provided with a hand-held counter, which he was instructed to depress at the onset of each discrete episode of voice activity. Baseline recordings were taken for 28 days, and indicated a mean of approximately 310 episodes of voice activity per day (equivalent to one episode per three minutes). A sponge-type sound occluding ear-plug was then placed into the left ear, with no expectation given to the subject about its purpose. The results are shown in the figure.

There was a 46% drop in voice activity in the first week, compared with the last week of the baseline period; 12 days following this intervention, the second technique derived from Green's theory was employed. The patient was instructed to say 'Stop', and to name things in his immediate environment, with the emphasis being on loudness and speed of naming. During the 12 days in which the subject wore the ear-plug, there was a slow rise from 140 to 210 episodes of voice activity per day. On the introduction of the second technique, this rise was apparently 'checked', although the possibility that this simply represented spontaneous fluctuations in the record cannot be ruled out. There was a slow improvement over the following 84 days. The recording was suspended and the patient followed up 44 days later, and 24 further days of recordings were taken, showing evidence of a further moderate decline. To test the specific effect of the ear-plug, the subject was now requested to remove it, whilst continuing with the 'stop

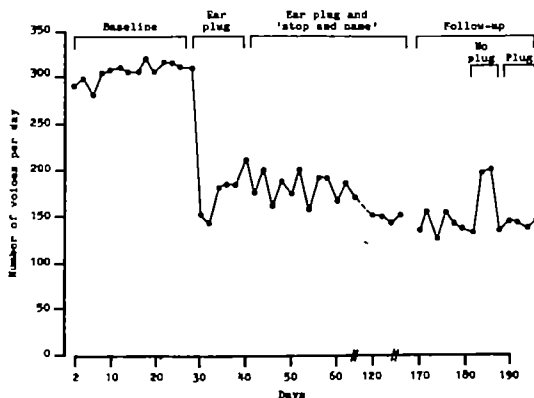


FIG. The effect of the treatment techniques on hallucinatory activity

and name' technique, but after four days of daily monitoring, it was decided to re-introduce the plug, as the patient reported feeling disturbed by the voices. (Further experimental manipulations of the techniques were not attempted for this reason). The removal of the plug had given rise to a 25% increase in voice activity (see Figure). Concomitant with the decline in self-reported voice activity, the patient also commented on a reduction in their audibility, and further validation of the patient's improvement came from ward staff, who commented that he appeared much more animated and talkative.

Discussion

The results show clearly that the effect of occluding auditory input to the (presumably) non-dominant hemisphere was markedly to reduce the frequency and severity of voices in this severely hallucinated schizophrenic patient. The efficacy of incompatible dominant hemisphere verbalisations (the 'stop and name' technique) was less certain, as experimental manipulations of this technique could not be attempted, but two observations suggest that this technique did have an independent effect. Firstly, a rise of 70 episodes of voice activity per day, over the 12 days during which the ear-plug was employed, was discontinued on introduction of (the 'stop and name' technique) though this could be accounted for by 'spontaneous' fluctuations in the record. Secondly, during the experimental withdrawal of the ear-plug over four days the rise in voice activity did not return to baseline, and this was possibly accounted for by the continued use of this technique. However, it is possible that the techniques had actually led to a reduction in the level of the baseline, in which case this conclusion would have to be tempered. Only complete withdrawal of both techniques would establish this.

The results are consistent with an independent effect of 'stop and name' and an additive one with the ear-plug, but the data are inconclusive on this point. Variants of these two techniques have in fact been independently reported. Falloon & Talbot's (1981) study of schizophrenics' self-reported coping strategies revealed that reduced attention to voices, particularly where this involves the evocation of alternative thoughts or verbalisations, appeared to be a helpful tactic. Incompatible verbalisations were employed in a successful single case study (Erickson & Gustafson, 1968)—a feature common to many other successful single-cases (Marzillier & Birchwood, 1981). McGuffin (1979) reports the case of a young disturbed schizophrenic patient who of his own accord, wore ear-plugs "to hear things clear": this also led to an improvement in his general sense of well being and interpersonal functioning (similar in fact to the present case). He also mentions two further cases of hallucinated patients who showed improvement with ear-plugs.

Many of the subjects of Green *et al* (1983) showed a binaural deficit in comprehension, compared with the "best" ear—a phenomenon not observed in the present subject. One is tempted to speculate that in such a severely hallucinated patient there may be somewhat more profound hemisphere disassociation, thereby reducing interhemispheric inter-

ference, which Green suggests accounts for the binaural deficit. Gazzaniga *et al* (1979) reported the case of a split-brain patient, where the non-dominant hemisphere seemingly developed expressive and intrusive speech. There is, however, another theory of auditory hallucinations within the cerebral laterality literature, which is consistent with the treatment findings. This idea, espoused by Flor-Henry (1976), argues that this and other psychotic phenomena occur as a result of disorganisation in the dominant orbital temporal-frontal regions. Flor-Henry draws parallels between the hallucinations sometimes observed in temporal lobe epilepsy and those of schizophrenia. However, if the present subject had such an impairment, one might have anticipated no ear differences in language comprehension if the functions of the primary language areas were disturbed.

The present results, together with those reported by Green and James, suggest the need for further research to establish the generality and specificity of the theory and of the relevant voice-control techniques.

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Obsessional Symptomatology and Adverse Mood States*

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Patients suffering from obsessional neurosis without other psychiatric diagnosis were examined with reference to the association of the mood disorders of depression, anxiety, and outwardly and inwardly directed irritability. All mood disorders were common, and many occurred together in individual patients, but anxiety was the most prevalent. Inwardly directed irritability was significantly related to the overall severity of obsessions, and to the degree of handicap or interference in everyday life experienced by the patient.

The relationship of obsessional neurosis to disorders of mood has been the subject of intensive enquiries over many years. Sometimes, reports of associations have appeared to be objective, but at other times objectivity has been submerged by the need to provide support for a preconceived theoretical standpoint. The literature has been reviewed by Snaith (1981a) and by Farid (1983); only its salient features will be presented here. Although the mood disorders of depression, anxiety, and irritability are usually inextricably intermingled, the present report deals with them as separate states.

Depression: The observation of the association between depressive illness and obsessional symptoms commences with the study of Lewis (1934), in which 21% of severe melancholics had obsessional symptoms; Stengel (1945) considered that cyclical depressive illness could present with such prominent obsessional symptoms that the underlying nature of the disorder was obscured. Kringlen (1965) noted the association between obsessional neurosis and depressive symptoms; Kendell & Discipio (1970) found a greater than chance association between obsessions and depression, and Coryell (1981) confirmed an association between the two disorders. The relationship between obsessional symptoms and an underlying disorder of mood has been examined in a series of papers by Beech (e.g. 1971); an underlying mood disorder appeared to be a primary event in a chain culminating in obsessional behaviour, and the mood disorder was characterised by depression and irritability rather than anxiety. Miller (1980) found that obsessional patients showed a marked negative cognitive set rather than true depression, and speculated that obsessional symptoms are coping devices which defend against depression.

Anxiety: The relationship between obsessional symptoms and anxiety is a central theme for those who examine the concepts from a behavioural standpoint. Lorr & Rubinstein (1956) found a significant association between obsessional symptoms and an anxiety-tension factor. Rachman (1971) considered that obsessional ruminations arise in the context of altered mood states and that attempts to escape the ruminations by neutralising thoughts reduce anxiety only temporarily, the relief leading to further positive reinforcement of the disorder. Beech (1978) postulated that there is a defect of the arousal system, so that minor alterations to the incoming stimulation precipitate major defensive reactions, leading to anxiety.

Irritability: This term is widely used but rarely defined. Until recently, the *Index Medicus* had no Medical Subject Heading for irritability, and all works relating to the concept had to be classified under the heading either of aggression or hostility, but from 1985, Irritable Mood is to be included as a Medical Subject Heading (Snaith & Taylor, 1985). The literature on the relationship of this mood disorder to obsession is not very extensive, and it is not always possible to be sure that the terms have been used in the same sense. Fernando (1977) found a relationship between obsessional symptoms and aspects of hostility, while Mellett (1974) detected an unusual inclination among obsessional patients to suppress anger. In another study (Manchanda *et al.*, 1979), there was a significant difference between obsessionals and a matched control group of neurotic depressives in relation to hostility, the obsessionals showing greater acting-out hostility and extra-punitive tendencies; Miller (1983) has confirmed a relationship between hostility and obsessional neurosis.

*Based on a dissertation for the degree of M.Med.Sc. University of Leeds and which was awarded the Max Hamilton Prize.

The present study aimed to examine the relationship between aspects of obsessions and disorders of mood, in a group of patients suffering from severe disabling obsessional neurosis, who accord to a strict definition of obsessional neurosis (Feighner *et al.*, 1972) and who were not currently suffering from other major psychiatric disorder which might be considered to be the primary disorder or which would at least lead to a collateral classification in the International Classification of Diseases. Aspects of mood disorder (depression, anxiety, and irritability) were defined and assessed both at interview and by self-report. This report is a survey of associations, and no attempt has been made at a theoretical interpretation.

Method

The sample consisted of consecutive referrals to the author, who had notified his colleagues in two hospitals of his interest to study mood disorder in relation to obsessional neurosis. The criteria for the diagnosis of obsessional neurosis were those of Feighner *et al.* (1972); no patient was included whose primary diagnosis was considered not to be that of obsessional neurosis, but who manifested obsessional symptoms in the setting of some other psychiatric disorder. The purpose of the research was explained to the patients, and their consent obtained. The final sample consisted of 20 patients (17 females, three males), with a mean age of 36 years (range: 23–49). Many patients had suffered from the disorder for several years, and the mean age of onset was 21 years (range: 16–26). Patients and referring psychiatrists considered the neurosis severe enough to require psychiatric treatment. At the time of the interview, seven subjects were in hospital and the rest outpatients.

The following assessments were undertaken during the course of an interview lasting between one and two hours:

(i) *Leyton Obsessional Inventory* (LOI) (Cooper *et al.*, 1970). This produces an overall index of the number of obsessional traits and obsessional symptoms present; it also provides scores representing the degree to which the patient attempts to resist the obsessional symptoms (Resistance), and the degree to which the symptoms interfere with the daily life of the patients (Interference).

(ii) *The Clinical Interview* (CI), based on the Standardised Psychiatric Interview (Goldberg *et al.*, 1970). The measures relating to depression, anxiety, and irritability were extracted; each mood disorder is rated on a five-point (0–4) scale, and for the purpose of the present research, scores of 0 and 1 were taken to indicate an absence of the mood disorder, and of 2, 3 or 4 to indicate the presence of a moderate to very severe degree of the disorder. The definitions provided by the instrument were adhered to, except in the case of irritability. The manual states that irritability should be assessed not only according to the degree of irritability and impatience in relationship with others, but also according to the degree of personal sensitivity; this latter aspect was omitted in the present study, and the

ratings reflected the degree of the patients' own statements about poor control, or feelings of poor control over temper, leading to expressions of aggression or violence.

No rating as high as 4 was made in any patient, but ratings of 3 reflected severe arguments or actual, albeit mild, physical violence.

(iii) The Irritability-Depression-Anxiety (IDA) Scale (Snaith *et al.*, 1978). This self-completion scale provides separate sub-scales for: depression, anxiety, and two aspects of irritability: expressed towards others (outward irritability), and directed towards the self (inward irritability).

The patients' scores on the IDA scale were unknown to the author at the time of the clinical interview. There is a broad agreement between the first three sub-scales of the IDA and the appropriate Scales of the CI, but the inward-irritability sub-scale had no counterpart in the CI.

Results

The scores on the CI and the IDA Scale are given in Table I. Only scores representing abnormality of mood, i.e. 2 or 3 on the CI or above the borderline range for the sub-scales of the IDA, are recorded; abnormality appears on one or other dimension in 19 patients, although four patients had no abnormality of any score on the CI.

Strict criteria were used for the definition of the presence of a mood disorder—that an abnormality of mood should be present on both the CI and the self-assessment scale before being recorded as such; this is in accord with Snaith's (1981b) view that in investigations of this type, a combination of clinical and self-assessment has advantage over either method alone. However, since the inward irritability self-assessment scale had no counterpart in the CI, reliance had to be placed on that scale alone. The criteria for inclusion of an abnormality of mood were therefore raised from the score above the top of the borderline range to one in the upper third of the score range, i.e. 9 or higher. By these criteria, anxiety was present in 15 patients, depression in nine patients, outwardly directed irritability in five and inwardly directed irritability in eight. No disorder of mood was present in four patients, but disorder of all four mood states in four.

The next stage of analysis was to examine the presence or absence of mood disorder in relation to the variables of the LOI (Table II).

The only significant associations were between the two aspects of irritability and the total LOI Score, and between Inward Irritability and the Interference of obsessions in daily life. It was considered that this method was a meaningful way of presenting the findings, but rank correlations (Spearman) were also calculated, and these support the analysis.

The correlations which were statistically significant were: CI—Anxiety/LOI—Total, $r+0.46$, $P<0.05$; CI—Anxiety/LOI—Interference, $r+0.51$, $P<0.02$; IDA—Outward Irritability/LOI—Total, $r+0.52$, $P<0.02$, IDA—Inward Irritability/LOI—Total, $r+0.74$, $P<0.01$ and IDA—Inward Irritability/LOI—Interference, $r+0.61$, $P<0.02$. Thus, correlations over the total range of scores for each

TABLE I
Mood and obsessional scores

Mood disorders										Leyton obsessional inventory			
No	Sex	Age	Depression		Anxiety		Irritability		Total		Interference	Resistance	
			CI	IDA	Case	CI	IDA	Case	CI	IDA (In)	score		
1	F	41	—	—	*	—	—	—	—	—	34	16	20
2	F	34	2	8	*	2	11	*	—	—	28	28	26
3	F	49	2	—	*	2	11	*	2	—	33	22	41
4	F	43	—	8	*	—	—	—	—	—	36	36	33
5	F	31	2	11	*	3	15	*	2	11	53	57	80
6	F	35	2	7	*	3	13	*	2	11	54	88	55
7	F	41	—	10	*	2	15	*	—	—	42	37	29
8	F	29	2	12	*	3	12	*	3	10	50	58	23
9	F	28	—	9	*	2	12	*	—	—	26	27	29
10	F	38	2	12	*	2	13	*	2	10	37	23	33
11	M	46	2	8	*	2	11	*	2	—	58	62	87
12	F	33	2	12	*	3	12	*	3	10	50	58	23
13	F	49	—	—	*	—	9	*	—	—	38	38	58
14	F	35	3	9	*	2	11	*	—	—	34	36	28
15	F	51	2	14	*	3	15	*	—	9	39	56	49
16	M	23	—	—	—	—	—	8	—	9	45	55	41
17	F	27	—	—	—	2	—	—	—	—	37	60	38
18	M	52	—	—	—	2	10	*	—	—	45	30	29
19	F	29	—	—	—	2	14	*	—	10	48	56	56
20	F	27	—	—	—	2	11	*	2	—	33	22	41

*Mood disorder present, see text.

TABLE II
Mood states and LOI variables

Mood disorders		n	Leyton obsessional inventory		
			Total score	Interference	Resistance
Anxiety	Present	15	42.1 (2.6)	44.1 (5.0)	41.9 (5.2)
	Absent	5	38.0 (1.9)	41.0 (7.8)	36.4 (6.4)
Depression	Present	9	44.8 (3.5)	52.8 (5.5)	44.9 (8.2)
	Absent	11	38.1 (1.9)	36.3 (5.0)	37.0 (3.6)
Outward irritability	Present	5	49.8 (3.5)*	59.6 (7.2)	44.2 (8.4)
	Absent	15	38.2 (1.9)	38.4 (4.1)	39.9 (4.9)
Inward irritability	Present	8	47.0 (2.2)**	56.4 (6.1)*	45.0 (6.8)
	Absent	12	37.2 (2.4)	37.6 (4.0)	45.0 (6.8)

Mean, (Standard errors), significance of difference (t-test) only significance values of $P < 0.02$ or higher are considered. ** $P < 0.01$, * $P < 0.02$

mood disorder of the CI and sub-scales of the IDA, with each LOI variable showed a weak significant relationship between Clinical Anxiety and LOI Total and LOI Interference. However, the clinical assessment of irritability did not have significant correlations with any of the LOI variables, although the irritability sub-scales of the IDA did.

Discussion

This study confirms previous reports (*inter alia*, Orme, 1965; Walker & Beech, 1969; Vaughan, 1976) that depression, anxiety, and irritability are prevalent and inter-related in patients suffering from obsessional neurosis. The central finding is that irritability, as defined above, has a clearer relationship than other mood disorders with both the overall severity of the obsessional neurosis and the degree to which the patient considers that his symptoms interfere with daily life. A weakness of the study arises from the definition of overall severity and other aspects of the obsessional disorder, inherent in the use of the LOI. Its scores of severity are from the number of obsessional symptoms present, which would allow only low scores to be derived by a patient who suffers from a single obsessional symptom and yet is severely handicapped by it. Further studies should pay attention to this point, and also to the fact that at the time of assessment, patients in hospital may be less severely handicapped by obsessional symptoms than those assessed at home.

The finding that anxiety is the commonest associated mood disorder should be taken in context with the fact that patients suffering from primary depressive or anxiety states were excluded. Moreover, this finding should not be related to consideration of the relationship between anxiety provoked by, or associated with the individual obsessional symptom, since the concept used was that of pervasive disturbance of mood, rather than anxiety directly engendered by the obsessions. It is in the latter sense that Walker & Beech (1969) made their observations of the relationship between obsessions and mood. The finding that outwardly directed irritability is less commonly experienced than that inwardly directed may be a useful observation on which to base both further studies and clinical management. Inwardly directed irritability is found to be both common and related to the overall severity of the obsessional neurosis and to the degree of interference or handicap produced by the symptoms. However, this concept should not be too readily equated with that of suicidal propensity; two out of the four items of the IDA Scale, on which the concept is based, relate to thought of hurt or harm to the self (not necessarily with fatal result), and the other two items are less strongly expressed, referring to annoyance and angry feelings of the patient about his own state. It is in this sense of the second of these concepts that the finding of a strong association between inward irritability and overall severity and handicap of the neurosis confirms the expected. This

annoyance with their behaviour probably underlies the observation of Pollitt (1969) that patients suffering from obsessional neurosis are embarrassed about their seemingly incomprehensible behaviour, and so delay in seeking help.

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‘Demand Characteristics’ in Body-Size Estimation in Anorexia Nervosa

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We asked 24 patients with anorexia nervosa and 30 normal controls to estimate their body-size several times, each time using different instructions. The degree of over-estimation was found to vary predictably with the wording of the instructions. Informing the subject that she had made an error without specifying the direction of the error resulted in reduced over-estimation on a subsequent trial, for both anorexics and controls. ‘Internally directed’ instructions were associated with a greater degree of over-estimation than ‘external’ instructions in both groups, but particularly in anorexic subjects. Our results indicate the necessity of controlling the ‘demand characteristics’ of such experiments.

The hypothesis that anorexics misperceive their body-size has received considerable experimental attention and the results have given it partial support (Garner & Garfinkel, 1981). One feature of the published data is a greater variation in the mean body-size estimates of the anorexics than in the estimates of controls. The source of this variation is unknown but there are reports that some anorexics under-estimate their body size although most over-estimate it (Touyz *et al*, 1984). Garner & Garfinkel (1981) have noted the relatively heterogeneous nature of many of the samples used in the experiments (e.g. variations in the history of the disorder; the presence/absence of vomiting; different attitudes towards eating and weight gain; presence/absence of depression and other indices of psychopathology; and variations in testing conditions). All these might contribute to the mis-estimation of body size.

One area which remains unexplored is the effect of the ‘demand characteristics’ inherent in experimental settings: it is possible that the variation in body-size estimations by anorexics is partly a consequence of idiosyncratic ways in which subjects interpret instructions and attempt to guess the experimenter’s hypothesis (Orne, 1969). Several reports exist of the effects of manipulating the instructions. Crisp & Kalucy (1974) and Pierloot & Houben (1978) instructed subjects to re-estimate their body-sizes after initial exposure to the task. Crisp & Kalucy (1974) observed a reduction in estimated body-size and claimed that the effect could only be obtained ‘within an overall psychotherapeutic relationship’ which would allow the patient to drop her defences. Although it is probable that this condition was not met in Pierloot & Houben’s

(1978) experiment, the second estimates they obtained tended likewise to be lower than the first, although the effect was not statistically significant. However, their second estimates were obtained under changed stimulus conditions: the previously darkened room was illuminated. The effects of instruction and setting were thus confounded. The same problem arises in the interpretation of Norris’s (1984) data. He requested subjects to re-estimate their body-size after they had had an opportunity to look in a mirror. They were explicitly instructed to use the ‘visual and tactile impressions gained at the mirror’ but were not told if their previous estimates were accurate or inaccurate. The anorexics significantly reduced their over-estimates of body-size on the second occasion. The problem is that it is not known whether they would have given more accurate estimates of body-size had they had access to the mirror before the first estimation. Norris also reports that the relationship between experimenter and subject in his study was not particularly close: it would therefore seem that a therapeutic relationship is not a necessary condition for the reduction of body-size estimates by anorexics. To return to the demand characteristics of the experimental setting; while it may be difficult to hide the general nature of the experimental hypothesis from intelligent and well-informed subjects (Szmukler, 1984), it is possible that the exact wording of the instructions to the subjects may have a predictable impact as their estimations of body-size. Some investigators have indicated that the exact form of the wording may vary from subject to subject (Button, 1979; Houben, 1979; both personal communications), which would probably magnify any variation due to the demand

characteristics inherent in the situation. It follows that increased precision in the instructions should result in reduced variability in the estimates.

The following paper reports on the effects of a systematic and controlled variation in the wording of instructions to subjects. We hypothesised that:

1. 'Ambiguous' instructions, which gave the subject no point of reference from which to make her judgement, would result in an over-estimation by anorexic subjects compared with controls. (As far as we can determine, our 'ambiguous' instructions were most like those used in other body-size estimation experiments).
2. Instructions which 'confronted' the subject with the fact that she had made an error in judging her body-size (without indicating the direction of the error) would lead her to reappraise her estimation, one way or the other.
3. 'Internal' instructions, which asked the subject to base her estimates on how she *felt* her body to be, would lead to over-estimation compared with 'external' instructions. ('External' instructions required the subject to estimate her body-size as if she were another person looking at her).
4. Both 'internal' and 'external' instructions would produce less variability in the subjects' estimations of body-size than 'ambiguous' instructions.

In addition to these comparisons we planned to investigate the uncontrolled factors—vomiting, in-patient/out-patient status, and time since last menstruation—which might contribute to variation in the sample.

Method

A total of 30 controls and 24 anorexic females were each given four different sets of instructions for estimating their body-sizes.

Subjects

The control subjects (all female) were nurses ($n=13$), secretaries ($n=11$), research assistants ($n=5$) or shop assistants ($n=1$). They were all healthy volunteers, recruited primarily from Addenbrooke's Hospital, Cambridge.

The anorexic subjects consisted of 12 in-patients and 12 out-patients: 9 were vomiters and 15 non-vomiters. All but two of the anorexics met criteria A, C, D, E and F for anorexia nervosa as described by Feighner *et al* (1972). The two exceptions had ages at onset of 26 and 34 years. Criterion B of Feighner *et al*—anorexia with accompany-

ing weight loss of at least 25% of original body weight—could not be used as information about original body weights was not available. Certainly all patients had lost a considerable amount of weight. Of the 24, five had had a menstrual period within the last month, seven more had had one within the last year; seven had last had a period 1–5 years ago; four had last menstruated more than five years ago; and one subject said that she had never menstruated. Two of the patients had children.

This sample of anorexics is more heterogeneous than those used in previous studies, primarily because out-patients were used as well as in-patients. However, if the effects of instructions are observable in such a varied sample, it would suggest that the differences found are quite robust.

Apparatus

We used a visual-size estimator built to resemble the machine used by Slade & Russell (1973). Body calipers were used to obtain actual body measurements.

Procedure

Subjects were tested individually in a totally dark photographic room at Addenbrooke's Hospital. The first author served as the experimenter for all subjects. Each session took about one hour.

The subject's height and weight (in normal indoor clothing) were measured. She was then told:

'I am going to ask you to make some estimations about various parts of your body. You will do this four times, with a short rest period between each session. The most important thing for you to do is to listen very carefully to my instructions at the beginning of each session, because they change each time. Are you ready to begin?'

The subject then took her place behind a line 155 cm away from the visual-size estimator, as in Slade & Russell's (1973) experiment, and the lights were extinguished. She was then given four sets of instructions (see Appendix), interspersed with short breaks. The order in which the instructions were given was determined in advance by means of a Latin Square, except that the 'confrontation' instructions were always administered immediately after the 'ambiguous' instructions. With each set of instructions the subject was asked to estimate the width of her face across the zygomas, bust (just below the armpits), waist (at narrowest point), and hips (at the widest point), as in Slade & Russell's (1973) study. She was also asked to estimate the depth of her stomach (the distance between the iliac crest and a point directly forward of this on the front side of her body) as in Button *et al*'s (1977) experiment. During the first trial only, the experimenter touched the subject's face, bust, waist, hips and stomach at appropriate levels to ensure that she understood what was to be estimated. Two estimates were required for each body part, one with the lights moving slowly outwards and one with them moving slowly inwards, as in Slade & Russell's study. The data were averaged for each trial (c.f. Button *et al*, 1977).

TABLE I
Means and standard deviations of age, height, weight and actual body widths of control and anorexic samples

	Controls (n=30) Mean (s.d.)	Anorexics (n=24) Mean (s.d.)	t
Age	21.8 (2.87)	24.5 (6.11)	—
Height (cm)	164.0 (5.70)	159.9 (4.37)	2.1*
Weight (kg)	58.9 (7.10)	46.7 (6.72)	3.296*
Face	11.4 (0.54)	11.2 (0.76)	—
Bust	26.1 (1.61)	24.3 (1.79)	3.79**
Waist	23.0 (1.57)	20.9 (1.99)	4.19**
Hips	32.0 (1.70)	28.3 (2.25)	6.88**
Stomach	19.2 (1.73)	17.4 (1.88)	3.75**

* $P < 0.05$ ** $P < 0.01$

Immediately after each set of estimates the subject was given some magazines to read outside the testing room for 3–5 minutes so that the experimenter could 'do some calculations', except that after the 'ambiguous' instructions the lights were switched on and the subject's actual body widths were ascertained using body calipers before she was excused from the testing room (the subject was not shown these or any other figures). She was then given the instructions for the 'confrontation' condition.

After completing the four sets of estimations, the subject was asked a series of questions about her vomiting, bingeing and general health. Finally, the rationale for the experiment was explained to the subject and her questions were answered.

Results

The basic descriptive statistics for the anorexic and control groups are shown in Table I. There was no significant difference between the groups with respect to age or the real width of the face. The anorexics in our study were slightly older than the average age of the samples reported by Garfinkel *et al* (1978), Pierloot & Houben (1978) and Slade & Russell (1973), but no older than the sample obtained by Button *et al* (1977). Our anorexics were slightly taller but slightly less heavy than the control group and were also, as expected, significantly thinner across the bust, waist, hips and stomach.

Body perception indices (BPIs) were calculated by averaging the values obtained on the two trials for each condition and expressing this value as a percentage of the real size (Slade & Russell, 1973). The average BPIs for each body part in each experimental condition, for anorexics and controls, are shown in Table II. These data were submitted to analyses of variance using the Multivariate programme of Finn (Finn, 1974). An initial ANOVA indi-

cated that there was no effect due to the order in which the instructions were given. The data were then collapsed over order. Under the 'ambiguous' conditions, the anorexics over-estimated all body parts compared with the controls, thus confirming the first hypothesis. 't' tests for each body site were all significant ($P < 0.001$, d.f. = 52 in all cases; face, $t = 3.63$; bust, $t = 4.47$; waist, $t = 5.47$; hips, $t = 6.06$; stomach, $t = 4.87$).

Confronting the subject with the information that she had made an error in her judgement produced the predicted change in the estimate of body-size, namely a significant reduction ($F = 63.59$; d.f. = 1,42; $P < 0.001$). However, there was no difference between anorexics and controls in this respect: inspection of the raw data showed that every subject reduced her estimate of body-size following the 'confrontation' instruction.

'Internal' and 'external' instructions resulted in different estimates of body-size, as predicted: 'internal' instructions produced larger estimates than 'external' ones ($F = 7.36$, d.f. = 1,42, $P < 0.01$). There was a significant difference between anorexics and controls in the way in which they responded to these instructions ($F = 6.85$, d.f. = 1,42, $P < 0.05$), with the larger effect being seen in the anorexic sample.

Post hoc analyses

Inspection of the means of variances of the anorexic group showed that despite the effects of the instruction on the means there was still considerable variation in the scores. *Post hoc* analyses were conducted to investigate the possibility that the sample might be stratified according to three criteria which might indicate the severity of the disorder: admitted vomiting as a way of controlling food intake; the in-patient/out-patient status of the patient; and the time since last menstruation.

TABLE II
Average Body Perception Indices (percentage of real size) for controls and anorexics under four different experimental conditions. Data in brackets are standard deviations

	Face	Bust	Waist	Hips	Stomach	Overall average
Controls						
Ambiguous	132.8 (24.0)	104.2 (14.8)	111.9 (15.9)	97.6 (10.4)	107.5 (17.6)	110.8 (12.06)
Confrontation	128.8 (20.5)	96.2 (13.3)	108.1 (11.6)	92.5 (10.9)	103.0 (12.7)	105.72 (14.23)
Internal	132.6 (20.9)	103.9 (11.7)	115.3 (11.8)	98.4 (8.3)	108.8 (17.4)	111.8 (14.73)
External	132.0 (20.4)	103.9 (13.6)	113.9 (14.8)	97.5 (10.8)	107.9 (16.8)	111.04 (15.61)
Overall	131.55 (21.5)	102.05 (13.40)	112.3 (13.65)	96.5 (10.16)	110.8 (16.25)	
Anorexics						
Ambiguous	188.2 (36.8)	121.2 (18.5)	146.4 (28.9)	122.5 (20.3)	151.1 (39.0)	142.08 (29.88)
Confrontation	148.7 (35.4)	109.2 (17.6)	131.3 (22.8)	109.8 (16.4)	136.3 (33.5)	127.06 (26.36)
Internal	161.1 (35.3)	120.9 (16.2)	145.3 (27.0)	121.1 (18.7)	147.3 (41.1)	139.14 (29.25)
External	153.6 (31.18)	118.0 (22.4)	140.1 (27.4)	117.4 (22.2)	142.5 (37.8)	134.32 (28.93)
Overall	157.9 (34.87)	117.33 (18.82)	140.77 (26.62)	117.95 (19.52)	144.3 (37.95)	

The small sample-size precluded any orthogonal comparisons of these indices and they were probably confounded. Three independent analyses of variance failed to reveal any systematic differences between groups. Moreover, grouping the data in this way had no effect on reducing the variances in the sample.

Discussion

The results of this experiment support the general hypothesis that anorexics over-estimate their body size. Direct support was also obtained for the hypothesis that the 'demand characteristics' of the experimental setting partly determine the degree of over-estimation. This second finding may account for some of the variation found between laboratories, and lends weight to Garner & Garfinkel's (1981) concern about the uncontrolled aspects of laboratory test settings. The impact of particular instructions is emphasised by the absence of any effect due

to the order of presentation of the instructions: this would seem to indicate that the estimates of body-size are influenced by the immediate stimulus conditions and not by the general laboratory setting. The fact that anorexics appear to be more affected than the control group by the change in the instructions emphasises that particular attention should be paid to the specification of test conditions.

The biggest effect of instructions was found in the contrast between the 'confrontation' and 'ambiguous' conditions. Indeed, confrontation produced a greater reduction in over-estimation than the 'external' instruction.

There are two notable points about our findings. Firstly, it would seem that no special relationship between the experimenter and the subject is necessary for reduction in over-estimation to occur: this study was conducted by an experimenter (L.P.) who had not previously met the subjects and had no therapeutic relationship with them. The second point is

that, without being told the direction of their error, all subjects (controls and anorexics) reduced their over-estimation in the 'confrontation' condition. This finding suggests that the experiment was at least partly free from the experimenter-bias artefact, as the experimenter had no expectations about the direction of the effect for the 'confrontation' condition. This finding is rather surprising and there is no obvious explanation for it. However, one possibility is that giving the subject any information at all about how to judge body-size ensures that she will pay more attention to the available cues. The fact that 'external' instructions produced more accurate estimates in the anorexic group can be construed as supporting this hypothesis. There is supporting evidence from other studies that providing cues for estimation will reduce previous estimates (Norris, 1984) or increase estimates in certain plausible conditions (Crisp & Kalucy, 1974). An explanation of this effect would be worth pursuing, as it may provide a more general explanation of why anorexics over-estimate their body size (Garner & Garfinkel, 1981).

Despite the apparent success of the experiment in differentiating between instructions, we found no effect of different instructions on the variation in the scores: this was contrary to expectations. Moreover, we were unable to account for the variation by examining subject characteristics which were not under experimental control. However, the range of subject characteristics we considered was rather limited and it is possible that allowing for other aspects would have provided a meaningful way of reducing the variance. Another possibility is that the anorexic group used in this experiment differs from those employed in other ones, being a little older than some.

The significant impact of the experimental conditions, and the fact that other studies have indicated that non-anorexics too over-estimate their body-size, suggest that it might be more profitable to investigate experimentally the general processes involved in body-size estimation rather than the supposedly pathological over-estimation by anorexics.

Acknowledgements

We thank Dr M. R. E. Proctor for building the body-size estimator and Dr P. J. Cooper for comments on a draft of this paper.

Appendix

Full text of the four sets of instructions given to subjects

Ambiguous

I am going to move these lights slowly outwards. Please tell me when the distance between them is equal to the width of your face/bust/waist/hips/distance from your back through to the front of your stomach (*each stated in turn*)

Now this time I will move the lights inwards and you tell me when to stop. Are you ready?

Confrontation

Rather than giving you a different set of instructions this time, I would like to repeat the ones we did just now, because in comparing your estimations with you actual sizes I find you really were wrong in your estimations. So listen carefully and let us see if you can be more accurate this time in your estimations. (*Instructions A were then repeated*).

External

Pretend for a moment that you are me, standing right in front of you, looking at you. Would you please estimate how wide I would see that you are across your face/bust/waist/hips/ from your back through to the front of our stomach? (*Each stated in turn*). The first time I will move these lights slowly outwards and you must tell me when to stop.

Now this time I will move the lights inwards and you tell me when to stop. Are you ready?

Internal

This time would you please show me how wide you feel yourself to be/you feel that you are across your face/bust/waist/hips/from your back through to the front of your stomach? (*Each stated in turn*). The first time I will move these lights slowly outwards and you must tell me when to stop.

Now this time I will move the lights inwards and you tell me when to stop. Are you ready?

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Hormones, Mood and Sexuality

SIR: We write in reference to the recent article by Alder *et al* (*Journal*, January 1986, 148, 74-79). The research recorded is in line with the high standards that one has come to expect from the MRC Reproductive Biology Unit at Edinburgh. A number of interesting findings emerged:

Firstly, in terms of hormones, testosterone and androstendione median levels were lower in 5 breast-feeding women taking a progestogen-only pill, in comparison to 14 breast-feeding women who were not taking steroidal contraception.

Secondly, in the breast-feeding women testosterone and androstendione median levels were lower in those assessed at interview as having severely reduced sexual interest.

There is an apparent anomaly here, in that one might presuppose that those women taking steroidal contraception were certainly more concerned about the results of sexual activity (preventing a further pregnancy) and were possibly more interested in sexual activity. As mentioned, their testosterone and androstendione levels were lower, but no comment is made in the paper concerning their *actual* sexual interest, in comparison with the group not taking the pill and who had higher male hormone levels. This information would be of interest in assessing the association between male hormones and sexual activity in this group of people.

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Drs Alder and Bancroft Reply

SIR: Drs Harris and Thomas raise an interesting point in their letter. If the reduced sexual interest in our sub-group of breast-feeding non-pill using women was a result of their lower androgens, did the pill using group whose androgens were also low report sexual interest, and if not why not?

The progestogen-only pill using breast-feeders did not differ from the non-pill using breast-feeders in either sexual interest or activity. This highlights the complexity and often contradictory nature of the evidence of androgens in the sexuality of women which we pointed out in the paper. It is of course

possible that the low level of androgens in the non-pill sub-group was a result, not a cause, of the low sexual interest. Or, alternatively, as we warned in the paper, they may have had lower androgens to begin with (i.e. before pregnancy). Although we have no evidence that our pill users were either more concerned about the result of sexual activity or more sexually interested before starting on the pill, they may have differed in some relevant way, e.g. their level of sexual interest may have been brought down by the pill or lowered androgens to the same level as the non-pill users. It is also possible that the androgen lowering effect of the contraceptive pill is different to that of breast feeding in some obscure way.

As yet there is little other evidence of the effects of progestogen-only pills on androgen levels. In a previous study of combined oral contraceptive users (Bancroft *et al*, 1980) we compared women with and without loss of sexual interest. Both groups had equally low androgens. In the "normal interest" group testosterone levels were positively correlated with self-ratings of sexual interest; this was not so in the low interest group. The role of androgens in female sexuality remains an enigma.

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Homosexuality in Monozygotic Twins Reared Apart

SIR: I was very surprised to read that female sexuality may be an acquired trait and male due to complex interaction, in which genes play some part (*Journal*, April 1986, 148, 421-425). It is quite clear that the authors are aware that their sample is small, but the consequences are not accepted (i.e. they dare make conclusions as outlined above). Homosexuality is not a rarity (despite the fact that collecting a large enough sample of homosexual

monozygotic twins reared apart is) and care should be exercised when trying to generalise about it.

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Dexamethasone Suppression Test

SIR: I refer to the review article by Braddock on the dexamethasone suppression test (DST) (*Journal*, April 1986, 148, 363–374), with particular respect to nonspecific factors which might affect non-suppression. I feel the omission of the effects of caffeine merits updating.

Uhde *et al* (1985) studied 22 normal volunteers and depressed patients (diagnostic criteria not specified). The DST consisted of dexamethasone 1 mg at 11 pm and a 4 pm cortisol sample. Analysis was by radioimmunoassay. All subjects were given a single blind placebo controlled challenge of 480 mg caffeine (equivalent to about 4 cups of filtered coffee) between 2 and 2.30 pm. Caffeine was found to increase the mean post-dexamethasone cortisol value from 2.3 to 5.3 $\mu\text{g/dl}$ ($P < 0.001$). For both volunteers and patients, when using the 5.0 $\mu\text{g/dl}$ standard cut-off point, non-suppression occurred in 14% on placebo and 36% on caffeine. This dose of caffeine is not unusual when compared to the average daily consumption (Gilbert *et al*, 1976), and indeed in psychiatric patient populations more caffeine may be used (Galliano, 1982). In addition, depressed patients in particular may actually increase consumption of caffeine as a result of their illness (Greden *et al*, 1978; Neil *et al*, 1978). Caffeine therefore should be added as an additional non-specific factor which might affect non-suppression.

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Is Hysteria (Conversion Reaction) Still Alive?

SIR: The article by Shalev and Munitz entitled Conversion Without Hysteria: A Case Report and Review of Literature brings out the controversy which exists around the diagnosis of hysteria (*Journal*, February 1986, 481, 198–203).

My experience in a psychiatric hospital in India in a semi-urban area is that there is a well defined group of patients who can be given a diagnosis of hysteria. We have described the clinical features of such a group of 276 patients (Subramaniam *et al*, 1980). Similar patients are very common in most of the psychiatry clinics in India. The following are the common clinical features: presence of physical symptoms, more often monosymptomatic, in motor or sensory system without any organic basis; pre-morbid hysterical personality traits; age of onset of symptoms before 20 years; more among females; dramatisation of symptoms with belle indifference; presence of some unconscious conflict not necessarily sexual in nature and complete recovery with psychoanalytically oriented psychotherapy. Any physical symptom in the absence of an organic basis should not be considered hysterical. If this is established, it is likely that hysteria or conversion reaction will continue in spite of the obituaries pronounced on it by Slater and others.

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On Serious Violence During Sleep-walking

SIR: Drs Oswald and Evans (*Journal*, December 1985, 147, 688–691) presented some interesting material on somnambulism, including a case of a criminal act committed in sleep. We report another case.

Case report: A young man stayed overnight at his mother's. In the middle of the night he was awakened by an unpleasant dream in which his mother criticised and attacked his girlfriend. He sat up and dressed, with the dream going on in his head, but aware of where he was. Suddenly he got the thought—as if a voice in his head told him—that he should threaten his mother not to be so derogatory and aggressive to his girl-friend. So he went to get a hammer in the basement and went to his mother's bed. Just as he was about to wake her she moved in her sleep. To his surprise he hit her again and again with the hammer without a conscious thought, until after a while he

realized what he was doing. Seeing the blood he left and went around the streets. A little later he returned to his mother's house, hoping to see an ambulance in the street. Next he went to her door, hoping to hear some sign of life from inside. Neither of his hopes were fulfilled, so he went to his own home, where hours later, he was arrested for the murder of his mother.

Throughout his earlier life the young man had suffered from frequent nightmares and as a smaller child he had been known to sleep-walk. The relationship between mother and son had never been very good and it had worsened because of the mother's disapproval of the son's girl-friend. In the days before his mother's murder the young man's dreams about his mother had been extremely vivid. Whenever he woke up he had to concentrate for a time in order to tell dream from reality. The young man stressed convincingly—and he was supported by the evidence of others—that it was very far from his nature to react with violence.

In the requested psychiatric report it was concluded that the young man was in good health physically, examination by specialists in neurology and EEG revealed no abnormalities. Psychiatric examination showed that he was immature and insecure, but there were no decisive signs of psychopathology. A similar conclusion was drawn from a full scale psychological test, where, in addition, evidence of difficulties in controlling aggressive impulses were seen.

It was claimed that at the time of the crime he had been in a somnambulistic state, a state of reduced consciousness and consequently he had been in psychosis-like condition. According to the Danish Penal Code, section 16—"Acts committed by persons being irresponsible owing to psychosis or similar conditions or pronounced mental deficiency are not punishable"—it was suggested that the crime should be dealt with by treatment and supervision. The case was presented to the Danish Medico-Legal Council, and the Council too concluded in favour of a psychosis or a similar condition. The court followed the medical experts. Discharge took place soon after, and in the first year the young man regularly consulted his psychiatrist, who described him as still immature and periodically anxiety-ridden. In the periods of anxiety he was not able to sleep without sedative medication. He was again admitted to hospital for a time, mostly because of lodging-problems. In the following years he saw his probation officer regularly, and seemed to undergo a positive development, both emotionally and socially. Seven years after the criminal act he was tested by the same psychologist who examined him during the period of mental observation. The psychologist reported the young man to be quite well functioning and compared with his earlier condition he was now quite well controlled, almost inhibited emotionally. Thus the test indicated character neurosis and thereby confirmed the clinical impression of personal stabilization.

Our case might be found somewhat more controversial than those presented by Oswald and Evans—some motive could be postulated and the degree of consciousness-reduction is not quite clear. Still, as

the border between sleeping and waking is perhaps not very well defined, we have great sympathy for our older colleagues who remembered the possibility of somnambulism.

In his precise and thorough survey of the subject Schmidt (1943) differentiates somnambulism from epilepsy, discusses psychiatric and forensic aspects and presents material on 15 cases of homicide. Furthermore a nosological subdivision is made; along with the sub-type called the dreamy type ("Die traumhafte Form"), there is a discussion of crimes committed—as in our case—while under the influence of a dream. In his conclusion Schmidt advocates that somnambulism should legally be treated like psychosis. The question whether the defendant is responsible or not must be answered with a no ("verneint"). In these cases the medical opinion is therefore fundamental. We close this letter, like Schmidt did his paper, by observing that even the most careful examination can leave the investigator in doubt.

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SIR: The article by Oswald and Evans (*Journal*, December, 1985, 147, 688–691) states that sleep walking can be accompanied by violent injury to the self or others. We would like to report another case.

Case report: A 25 year old divorced man was admitted to our service, having been charged with the attempted murder of his *de facto* wife. The circumstances surrounding the attack were unusual. After an uneventful day he helped his wife to prepare the evening meal, played a game of dice and then watched a movie on TV. On finding this uninteresting they had another game of dice and retired to bed at midnight. His wife's young son was ill and at that time was sleeping, in a single bed, in their room. The boy woke up several times during the night, vomiting, and our patient and his wife at various times got up to attend to him. This occurred till at least 2.00 am. His next recollection was of waking up as it was becoming light and seeing his wife sitting at the corner of their bed with something around her neck. He said that it looked like a piece of string or rope. He pulled it from her neck as a result of which she fell on the floor. He immediately ran down the hallway to the kitchen and telephoned for an ambulance



and the police. His wife was taken to hospital where, fortunately, she made a good recovery. He was charged with attempted murder and taken into custody. He denied any conflict with his wife; she was pregnant and they were both looking forward to the birth of their first child. He specifically denied any disagreement with her before they went to bed, while no sexual activity or attempted sexual activity occurred between them that evening. His wife, who left him after the assault and moved interstate, confirmed his story.

He came from a somewhat troubled background, in that his parents separated when he was a child, and for a time he was placed in a Boy's Home before reuniting with his father. He had a very close relationship with his father, both shared a common interest in black magic and seances. He was very distressed by the death of his father some years earlier and continued to maintain the contact with him by wearing some of his clothes on a regular basis. It was subsequently discovered that the article used in the apparent attempt to strangle his wife was the cord of the dressing gown previously owned by his father. Additionally the attempted strangling occurred very close to the anniversary of his father's death. Although the patient had a history of impulsive suicidal attempts, coupled with alcohol and drug abuse, these behaviours had not been evident for some time and there was no evidence to suggest that he had been so troubled, in any way, at the time of the attack on his wife. Again this history was confirmed by her. Our patient claimed that for some days prior to the attack his sleep pattern had been disturbed with night terrors/nightmares.

Physical examination and a range of laboratory investigations were normal. A skull x-ray, an EEG and CT scan of his brain were also normal. Neurological opinion was sought but no pathology was identified. Neuropsychological testing did not reveal any abnormality. An MMPI demonstrated a hypochondriacal profile.

At his trial, the defence raised the possibility of somnambulism which, at law, would fall within the category of non-insane automatism. He was found guilty by the jury and sentenced to 15 months hard labour, with a non-parole period of 11 months. This was suspended in favour of a probation order of three years with a condition to undertake any medical treatment as the supervising probation officer might direct.

Although it is clear that this man had an abnormal personality structure and some unusual interests, no convincing explanation or precipitant has ever been identified with respect to his assaultive behaviour. He had a history of disturbing dreams dating back to childhood. He had a vague partial recollection of one sleep-walking incident, some years earlier, when having gone to bed he woke up in the kitchen. No violence was exhibited on that occasion.

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Tardive Dyskinesia and Parkinsonism

SIR: Tardive dyskinesia (TD) and drug induced Parkinsonism (PS) both occur in association with the use of neuroleptics. The co-existence of TD and PS has been documented (McCreadie *et al*, 1982) but interest has mainly centred on the unmasking or the amelioration of dyskinesia either by anti-cholinergic agents (Chouinard *et al*, 1979) or of GABA agonists (Gerlach, 1977). The subject has also been studied using multivariate statistics on populations who exhibit neuroleptic induced extra-pyramidal effects (Kidger *et al*, 1980). These studies report that three independent factors emerge which approximate to the clinical syndrome of Parkinsonism, oral dyskinesia and akathisia. No study appears to have specifically considered the relative severities of the TD and PS syndromes when they co-exist and no comparison has been made with a control non-dyskinetic Parkinsonian cohort.

The aim of our study was to test the null hypotheses that the severity of PS in a TD group is no different from that of a control group exposed to neuroleptics but without TD. We also examined the correlation between the severity of TD and PS in our index group. The groups were carefully matched for age, sex, and type of medication. The 16 patients in each group were rated on the Abnormal Involuntary Movements Scale (AIMS) (Guy, 1976) and the Webster rating scale (Webster, 1968). The criterion for diagnosis of TD was a clinically recognisable disorder and a score of two or more on any item on the AIMS. The patients were on long-stay wards and had a primary diagnosis of functional psychosis. All subjects were on neuroleptics at the time of assessment and 9/16 in each group had been on long-term anti-cholinergics.

The mean age of the index group was 64.75 years (range 50–82) and of the control group 63.82 years (range 50–87). The Webster scores were compared between the groups and were not statistically different ($t = 0.926$, d.f. 30, NS). There was no correlation between the total AIMS and Webster scores ($r = -0.08$). We conclude that TD patients over 50 years have PS to a similar degree as an age- and sex-matched group without TD. In addition, in the TD group, the severity of PS did not predict the severity of TD. The co-existence of TD and PS and our findings of an independence of their relative severities calls into question the currently held view that PS results from a blockade and TD a hypersensitivity of dopamine receptors (Marsden *et al*, 1980). In our view the two conditions are either mediated through different dopamine systems or through independent but related neurotransmitter systems which have not yet been fully elucidated.

We thank Drs T. A. Kerr and I. N. Ferrier for their assistance.

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Mania Following Bereavement

SIR: I read with interest Rosenman and Taylor's case report of mania following bereavement (*Journal*, April 1986, **148**, 468-70). The authors state that such reports showing this association are uncommon. I report two further cases.

Case reports: (1) A lady who had no previous psychiatric history, first presented aged 49 years, two months after the sudden death of her husband. He had taken his own life whilst she was at work. At first, she grieved appropriately but six weeks after her bereavement she became restless, irritable and garrulous. She returned to normal mood within one month following treatment with neuroleptic medication and ECT. Four years later her mother died of carcinoma. She grieved initially but soon became cheerful. By the time of presentation, one month after her bereavement, she was restless, overtalkative, sexually disinhibited, giggly and expressed paranoid ideas with regard to her neighbours and sons. She said that nursing her mother for eight years had imposed a great strain on her and that her behaviour was a reaction to the lifting of this strain. She became euthymic within two months on treatment with haloperidol but 18 months later presented with a further manic episode. This occurred three weeks after a celebration in her husband's family to which she had not been invited and she had been initially very upset. She has been well in the nine years since the last affective episode.

(2) A 58 year old lady with a previous history of bipolar affective disorder presented the day after the funeral of her husband who had died suddenly of a myocardial infarct one week previously. Within 24 hours of his death she became restless, overtalkative and insomniac. On admission she talked incessantly and maintained that she felt "hilarious" in spite of occasional tearfulness. She believed she had special powers of healing people and that the television was telling her what to do. She could hear her husband talking to her. Her mood gradually stabilised on treatment with haloperidol, but one month after the bereavement she became depressed. She was then successfully treated with an antidepressant and discharged. One year later she presented with depression requiring treatment with ECT. Her mood stabilised but after a visit to her husband's grave three months later, she became manic with mixed affect. This resolved and she has been well for the last six months.

Rosenman and Taylor discuss the mechanism of manic response to bereavement. They cite the Post *et al* (1981) finding that a previous history of affective disorder predisposes to a rapid onset of mania. These two cases support this: (1) with no previous history of affective disorder did not develop mania until six weeks after her husband's death and (2) with a well established bipolar affective disorder developed mania within 24 hours of her bereavement. That repeated episodes of illness establish a facilitated pathway by which rapid changes of mood could occur, may be further supported by the recurrence of mania following case 2's visit to her husband's grave 15 months after his death, and following case 1's perceived rejection by her in-laws.

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Reference

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SIR: The case report by Rosenman and Taylor (*Journal*, April 1986, **148**, 468-470) of mania following bereavement was of considerable interest. I report another two cases.

Case reports: (1) A 46 year old divorced engineer was admitted as an emergency in a hypomanic state on the evening of his mother's funeral. Instead of returning home from the funeral he had gone to his place of work where his behaviour had caused concern, the work's medical officer had arranged admission. On admission he was dressed in a

morning suit and was constantly pacing the floor, his mood elevated, he was overbearing and slightly aggressive in manner. There was marked pressure of speech and flight of ideas. This state persisted for six weeks and was followed by a period of depression. There is no evidence that he was hypomanic prior to his mother's death. There had been a hypomanic episode some two months beforehand but this lasted less than a month and there is well documented evidence that his mood remained stable up to the time he had leave from work after his mother's death.

Almost two years later his father died and five days afterwards he was again admitted in a similar state. Again there was no evidence to suggest that his mood was elevated before the event.

This patient had an established manic-depressive disorder and had been subject to mood swings with no apparent precipitant. On these two occasions and on a possible third occasion when a hypomanic episode followed his wife leaving him, there was a clear precipitating event.

(2) A 24 year old single woman was admitted from the police station in a manic state. She had been found wandering naked and was grossly overactive, her mood alternated between elation and aggression, her speech was constant, with flight of ideas, punning and clang associations. Three weeks previously her adoptive father had committed suicide. She had felt pressure from the family as there had been a suggestion that she had been responsible for her father's depressive illness. During the three weeks after his death she had become progressively more disinhibited, over-active and promiscuous. Again, there is no suggestion that her mood was different before the death.

She had no previous history of affective disorder. However, a year before this episode she had been diagnosed as having a personality disorder. It is possible that her behaviour at this time indicated a mild hypomanic episode. Since the reported incident she has had one depressive swing starting three days after she gave birth. This depressive swing was followed by a period of mild hypomania.

I am grateful to Dr F G Spear, Consultant Psychiatrist, for permission to report these cases.

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Lithium Carbonate and Piroxicam

SIR: The report by Walbridge and Bazire (*Journal*, August 1985, 147, 206-207) of an interaction between lithium carbonate and piroxicam prompts us to report details of a patient who also experienced lithium toxicity, probably attributable to this combination.

Case report: A 62 year old female with a 20 year history of manic depressive illness was started on lithium in March 1984 during her twelfth admission to hospital. Initially her renal function was checked and showed a plasma creatinine of 80 $\mu\text{mol/L}$. Her plasma lithium was stabilised at

0.6-0.75 mmol/L (checked monthly) with a dose of 800 mg of lithium carbonate at night. Ten months later, on 24.1.85, piroxicam 20 mg daily was prescribed for osteoarthritis of her right knee. Concurrent medication was as follows: amitriptyline 150 mg at night from 16.1.85 (previously on dothiepin 75 mg at night started before 1984), chlorpromazine 175 mg daily (from before 1984), procyclidine 5 mg tds from 14.1.85, and temazepam 20 mg at night from 11.1.85. Her January lithium level (taken on 8.1.85) was slightly raised at 0.85 mmol/L, possibly due to a recent loss of weight. At the end of January, she became noticeably agitated and tremulous: and during the next few days developed ataxia. During this period she was eating and drinking very little. A lithium level, done on 7.2.85 was 2.85 mmol/L with a raised plasma urea of 12 mmol/L and plasma creatinine of 142 $\mu\text{mol/L}$. All drugs were stopped and i/v fluid replacement started. Plasma lithium fell in four days to 0.35 mmol/L and plasma creatinine was inside the normal range (60-120 $\mu\text{mol/L}$) within 48 hours. Renal function tests continue to show mid-range values at the present time. She was also treated for an *E. coli* urinary tract infection with Amoxycillin starting 8.2.85. The lithium toxicity in this patient was probably exacerbated by dehydration and a urinary tract infection, but we consider that a piroxicam/lithium interaction could have contributed significantly.

Although the lithium/non-steroidal anti-inflammatory drug (NSAID) interaction has been well established, the mechanism is not completely understood. One mechanism, that of suppression of renal prostaglandin synthesis by NSAIDs leading to a fall in GFR and thus a reduced clearance of lithium has been clearly implicated in some cases (Nadarajah & Stein, 1985). This is reversible but the risk may be greater with NSAIDs with a long half-life (Adams *et al.*, 1986). Walbridge and Bazire suggest using an NSAID with a short half-life, e.g., ibuprofen, to reduce the extent of the interaction; however reports have shown marked increases in plasma lithium concentration within a short time of initiation of ibuprofen therapy (Ayd, 1985; Leftwich *et al.*, 1978).

In the light of this, it has been of concern to us that ibuprofen is now a non-prescription drug in the UK and can be bought freely over the counter. With this in mind, we now counsel our lithium patients on various aspects of their therapy with the use of information cards which specifically refer to the possibility of an interaction with ibuprofen. We ask our patients to show their card to any chemist they buy medicines from.

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Seasons of Birth and Sub-Types of Schizophrenia

SIR: Many studies show disproportionately higher rates of schizophrenic patients born during the winter months (Hare *et al*, 1974; Torrey *et al*, 1977). We undertook a study to find out if there is an excess of winter births among schizophrenic patients, and in particular if seasonal variations are related to the gender and sub-types of schizophrenic patients. We examined the seasons of birth of 472 patients with the diagnosis of schizophrenia admitted consecutively to a teaching hospital over a period of 5 years. We also studied the gender and paranoid vs non-paranoid sub-types of this subject group.

The total group did not show an excess of winter births, neither did the total male ($n=193$) and total female ($n=279$) schizophrenic patients. However, the male paranoid schizophrenic group ($n=102$) showed a significant ($P=0.05$) increase of births during the first quarter of the year. The different sub-group of female patients showed a homogeneous distribution of seasons of birth. We found a fairly stable distribution of seasons of birth among both the male and the female non-paranoid sub-groups (those with a greater genetic loading). According to Huxley's "genetic morphism" hypothesis (Huxley *et al*, 1964), the genes endow these sub-groups of patients with better capabilities of resisting various environmental factors. The homogeneous distribution of birth among the female paranoid sub-group could be explained on the basis of certain "built-in" protective factors for the female population (Seeman, 1981). Nature has provided the female with protective factors which may be responsible for their later onset of illness, better response to neuroleptics, fewer relapses, better work record, greater social adjustment, and significantly fewer peri-natal problems as compared to their male counterparts. Although there have been several explanations, the significantly greater number of

births of males in the paranoid sub-group during the winter months has been explained on the basis of the two hypotheses, viz. pre- or perinatal birth trauma or the viral infection hypothesis (Torrey & Peterson, 1976). They contend that because of the higher prevalence of viral infection during colder months, the schizophrenic patient is infected with slow or latent viruses, which alter the function of nerve cells in critical areas of the brain without altering the histological structure. Since the direct evidence of viral infection is lacking, we proceeded to test the first hypothesis, i.e., that subtle birth trauma later predisposes to the development of schizophrenia in susceptible individuals. For this purpose we examined the birth dates of 44 (38 males and 6 females) patients with the diagnosis of minimal brain dysfunction admitted consecutively during the same period as those of our schizophrenic sample. This group of patients is known to have high incidence of subtle brain insults. However, we failed to find any seasonality of birth in this sample. Therefore, we may infer that in our sample the significant increase of births among male paranoid schizophrenics during the winter months could not be explained by factors related to peri-natal trauma. This finding tends indirectly to give more weight to the viral hypothesis of schizophrenia, especially among male paranoid schizophrenics.

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Book Reviews

Dangerousness: Probability and Prediction, Psychiatry and Public Policy. Edited by Christopher D. Webster, Mark H. Ben-Aron and Stephen J. Hacker. Cambridge: Cambridge University Press. 1985. Pp. 236. £25.00.

It is sad to reflect that of all living creatures, man has the greatest propensity to be in conflict with himself and with others. Aggression, and its counterpart violence, both of which carry distinct overtones of dangerousness, would appear to be innate. If this is so—and the evidence is pretty irrefutable—then, given unusual or exceptional circumstances, we can all behave dangerously. For example, the *crime passionnel*, so often carried out with unbelievable violence and ferocity, is equally often at odds with the day-to-day behaviour of the assailant. And what happens to Joe Bloggs when he is transformed into Private Bloggs is the stuff that history books are made of.

What, I suppose, this book attempts to do is to narrow the field to the probability and prediction of dangerousness in those suffering from mental disorder, although, to my mind, this is a factor which must add yet another dimension of uncertainty to the dilemma.

Professor John Gunn of the Institute of Psychiatry, London, who is, incidentally, the only non north-American contributor, sets the scene unambiguously in his Foreword. He writes "... we learn, yet again, that our ability to forecast which people will do harm in the future is very poor". The implicit pessimism is almost painfully amplified in an excellent, yet hardhitting, chapter by Professor Alan A. Stone. He alleges—and few of us who turn an honest penny in the practice of Forensic Psychiatry in Courts of Law in the UK would dare disagree—that "the question of dangerousness, the statistical prediction of violent behaviour... has become the chief battlefield in the struggle between law and psychiatry". He goes on to deliver a flurry of telling blows to the psychiatric chin. "I found the published studies of prediction woefully inadequate, poorly conceived, wrongly interpreted and well below any acceptable standard of scientific research or even solid clinical experience", he opines.

The same sort of scepticism, although far more delicately expressed, is to be found in the contribution of Professor Cyril Greenland. He quotes Dr Barry Boyd as saying that only 10% of the patients detained under his care in a Canadian maximum

security hospital would kill again on discharge, and that he would be delighted to set the remaining 90% free if someone could tell him reliably who they were. It could be added that our own Dr Patrick McGrath, lately of Broadmoor Hospital, has oftentimes been heard to express the same opinion, it could be added.

For my money, far and away the most intriguing chapter is that by Professors Christopher Webster and Mark Ben-Aron entitled, "Dangerous Doctor Fischer: a case history in clinical assessment." Dr Fischer is the central figure in Graham Greene's allegorical, spine chilling novel, *Doctor Fischer of Geneva or The Bomb Party*. Greene with his incomparable insight into the vagaries of the human psyche brilliantly portrays one of that most evil and dangerous breed of psychopaths (Shakespeare does a similar job with Iago), the jesting destroyer. It matters little that in the end, after repeatedly corrupting and humiliating a coterie of greedy toadies, Fischer falls victim to his own corruption and kills himself.

All in all, for those of us who like myself had hoped to find in this book some sort of philosopher's stone that would transmute the base metal of educated guess-work into the gold of certainty, will be disappointed. Perhaps, for the time being at any rate, we will have to fall back on that well-worn saw—nothing predicts behaviour like behaviour.

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Using Metaphors in Psychotherapy. By Philip A. Barker. New York: Brunner-Mazel, 1985. Pp. 221. \$25.00.

The title of this book excites the hope of a general examination of the therapeutic use of metaphors in psychotherapy. It soon becomes apparent however that the major emphasis is on strategic and systemic approaches in child, adolescent and family psychotherapy. A paragraph is given to the use of metaphor in psychoanalysis and behaviour therapy and the other therapies are left to fend for themselves. The same may be said of the examination of metaphor. The definition of the word is extended, not without justification, to focus on the use of stories, anecdotes, metaphorical objects and activities as indirect instruments of psychotherapeutic change. The relationship of metaphors to symbols and

symbolic function in dynamic psychology referred to by well-known psychoanalysts is not discussed.

Once the reader realises that what appears to be a whole story is only a part of it and that this is a primer or guidebook on the therapeutic use of stories, anecdotes, and analogies, it can become a helpful and thought-provoking book.

The first three chapters discuss the past and present use of extended metaphors as an indirect, entertaining, informative and flexible way of communicating, and of their place particularly in the more directive strategic and systemic therapies. The author reflects on their power to reach hidden emotions, to reduce resistance, to reframe, redefine and suggest solutions to problems, to restore confidence and competence and what is rather chillingly called "embed" directives. Then there is a classification and discussion of various types of therapeutic metaphor. These chapters are useful in setting out some of the principles underlying the therapeutic use of metaphors and will be of particular interest to family and eclectic psychotherapists and students of Milton Erickson's approach to whom the author expresses his indebtedness.

Succeeding chapters focus on the use of metaphors in the establishment of different kinds of treatment goals; the treatment of developmental problems; conduct disorders and other behavioural problems; anxiety and depression; family problems and difficulties in wider social contexts—and a variety of miscellaneous issues, and finally with the practical delivery of metaphors, and the important issue of gaining rapport.

The book is structured so that each chapter starts with a story, ends with a summary and with a practical exercise in constructing "metaphorical approaches" to particular problems. I think some readers may find one or two of the stories too long, some aspects rather repetitive and the practical exercises particularly daunting if worked on alone, rather than in a group or seminar. In fact, the author's statement in the introduction, that "the use of metaphors is surprisingly easy once rapport is established" sets a tone of untroubled optimism which lingers on despite a warning illustration of a possibly resistant family, statements on the importance of fitting the metaphor to the client and observing verbal and non-verbal responses.

Nevertheless, so long as these limitations are kept in mind, the book contains a great deal of valuable information, and deserves a place as an introductory text to this intriguing area of communication.

BRIAN LAKE, *Consultant Psychotherapist,
St James's University Hospital, Leeds*

General Hospital Psychiatry. By Michael Alan Taylor, Frederick S. Sierles and Richard Abrams, London: Collier Macmillan. 1985. Pp. 463. £35.00.

Let us immediately clarify a misunderstanding about the title of the book which may well arise among psychiatrists working in general hospitals. The term general hospital psychiatry is beginning to be used in favour of the more cumbersome consultation/liaison psychiatry to connote the professional activities of psychiatrists who work within district general hospitals as opposed to mental hospitals.

This, however, is a textbook of in-patient general psychiatry. The idea is that no previous text is geared towards the modern style of psychiatric practice; biologically and pharmacologically based usually occurring over fairly short periods of in-patient stay in wards "designed to look like living rooms". Thus the authors write as much like physicians as they can—under the circumstances. The importance of making a correct diagnosis is emphasised and a few "diagnostically powerful" symptom patterns described, although I found these often contentious and unconvincing. Happily, the authors place great importance on observation of the patient for a few days before starting treatment, a rule which they admit is nearly always broken.

The mental state examination is given the grand but misleading title of Neuropsychiatric Examination. A separate Behavioural Neurologic Examination is then described which is, in essence, the detailed cognitive mental state examination. A second section then deals with patient management including chapters on various forms of treatment except psychotherapy. There is one chapter on Consultation/Liaison Psychiatry. The final section has 16 chapters on the major diagnostic groupings in psychiatry.

This is an interesting and well-written book. It covers psychiatry from a specific viewpoint, making no attempt to include all available treatment options and sticking rigidly to a determinist, behavioural and neurologic framework. As a result, the great value of this approach in some areas is apparent and therefore it is a book which, particularly in the USA, should be of considerable interest. It is highly critical of pseudo-mechanistic interpretations of disease as found for example in some Freudian terminology.

However, in other areas, the failure to consider any form of psychotherapy causes some passages to appear lightweight and superficial. Neuropsychiatry is still in its infancy and to attempt to expand it to cover all conditions presenting to the psychiatrist

threatens to break the bounds of credulity. To say that eventually we will know enough about the brain to carry out that exercise successfully presupposes that the materialist-determinist view of the human being proposed by Ryle is correct. We may however quite legitimately conclude that Karl Popper was right to derisively call this a "promissory materialism", and that therefore it is likely that psychotherapeutic skills will be in demand for some considerable time.

CHRISTOPHER THOMPSON, *Senior Lecturer in Psychiatry, Charing Cross and Westminster Medical School, London*

Ethical Issues in Psychosurgery. By John Kleinig
London: George Allen & Unwin. 1985. Pp 152.
£15.00 (hb) £5.95 (pb).

This is an interesting book, written by an academic philosopher, which is easy to read. It could almost as well have the title, "Ethical Issues in Psychiatry", because it is noted early on that a number of psychiatric treatments, including "...drug therapy, aversive conditioning and even token economies must confront the problem of diagnosis, irreversibility and consent that psychosurgery so sharply raises". Thus, the general psychiatrist will find relevant reviews in, for example, the sections dealing with the classification of psychiatric conditions (illness or deviant behaviour; treatment or punishment), placebo responses, and alleged social influences on psychiatric attitudes.

Those who have managed to get through the many pages of the "Consent to Treatment" draft document prepared by the Mental Health Act (MHA) Commission, may turn to the chapter on consent in this book with some eagerness. The subject is dealt with sensibly although much will be well known to psychiatrists (such as, why get consent?) and it does not become so confusingly entangled with law as does the Commission's document.

Understandably the need for more information and more scientific method in assessing psychosurgery is frequently stressed in the book, although the author himself indulges in some speculation. He considers that the wide-spread use of standard pre-frontal leucotomy in the 1940s was because the "The Second World War produced a large number of psychological casualties (which) constituted a serious logistical problem" (p. 7). He asserts that this situation created pressures for the too frequent use of psychosurgery, but no evidence is offered in support of this view, which is unique to this book as

far as I am aware. Similarly, the author feels able to state, with regard to the development of psychosurgery, that concern for patients "was clouded by... the desire to enhance the scientific or other ambitions of its practitioners" (p. 12). This acid comment could also do with some supporting facts.

The author certainly foretells of the coming of our MHA Commission. His answer is "no" to his own question, "is the patient's consent sufficient to justify psychosurgery?". The patient's consent cannot be relied upon because "some... would probably be willing to consent to an inappropriate treatment" (p. 60), which assumes that the neurosurgeon and psychiatrists advising the patient cannot give reliable opinions about appropriateness (perhaps because of "ambitions", see above). It follows, one supposes, that unambitious MHA Commissioners are needed.

An indictment is clear but not explained: the author observes that some "may resent... monitoring and regulation, especially if enshrined in law, but it is only because their profession has shown itself incapable of, or unwilling to, regulate itself that such measures are proposed" (p. 21). In what way did we not regulate ourselves?

P. K. BRIDGES, *Physician in Psychological Medicine & Senior Lecturer, Guy's Hospital, London*

Psychosomatic Medicine and Liaison Psychiatry: Selected papers. By Z. J. Lipowski. New York: Plenum. 1985. Pp. 453. \$39.50.

It is a bold step to publish one's collected papers, since it assumes an audience for work written long ago. Usually, such books lie unread in libraries, but this book deserves a better fate since Professor Lipowski has played a rather remarkable role in the development of what has become known as "liaison psychiatry". During the last 20 years, this has been established as a North American sub-specialty, becoming a part of almost every training programme and accepted in every substantial general hospital. Although, for most of this time, the predominant mood has been one of optimism, recently reductions in funding and the swings of fashion have led to doubts, falling morale and pessimism about the future. It is a good moment to take stock.

What has Professor Lipowski's contribution been to liaison psychiatry? He has undoubtedly been a man of his time who had the good fortune to begin his career as traditional psychosomatic medicine was expiring and as psychiatry was turning back to clinical medicine. However, there can be little doubt that his vigorous promotion of the clinical and academic aspects of his subject have helped to establish

its public image. By wide reading and by judicious use of what must be a remarkable card index, he has been able both to reflect what has been going on around him and to be in the forefront of crystallising new ideas. Starting with a major three-part review of psychosomatic medicine in 1977, he has closely chronicled the development of clinical practice and thought. Although he has described his own clinical services his main creative contributions have not been of clinical innovation or research. They have been theoretical and descriptive, the formulation of the principles of liaison practice, description of the main factors affecting individual response to physical illness and, most recently, a clarification of the syndromes forming organic mental disorders.

The book begins with a very short and anonymous introduction which outlines Professor Lipowski's career, a career which began in Poland immediately after the Russian occupation and continued in Ireland and Britain before he moved to North America. After working on the Psychiatric Consultation Service at the Massachusetts General Hospital, he set up liaison psychiatry units in Montreal and Dartmouth. Since 1983, he has been Psychiatrist in Chief at the Psychosomatic Medicine Unit of the Clarke Institute and Professor of Psychiatry of the University of Toronto. It has been an eventful life in several ways and it is a pity that Dr Lipowski has not felt able to tell us more about his personal experience. One would like to know more of his life and more of his personal views, even his prejudices. Seven of twenty-seven chapters are new pieces, or more correctly re-written papers, and these help the book to provide an up-to-date view of the current state of liaison psychiatry. There are four sections; Psychosomatic Medicine: Theoretical Concepts; Psychosocial Reactions to Physical Illness (two useful new chapters); Physical Illness and Psychopathology (chapters on epidemiology and organic disorders); Consultation-Liaison Psychiatry.

Professor Lipowski is usually an enthusiast and an optimist and he is a kindly reviewer of research. He can be stern about unsubstantiated speculation but has been reluctant to criticise either the methodology or the very disappointing literature on evaluation. He is somewhat inclined to accept as definitive conclusions what should be regarded as provisional or even speculative. This kindness is probably an inevitable accompaniment of his unrelenting work to establish liaison psychiatry. A harsh scepticism about theory and methodology is also necessary but could not have accomplished much for a very young area of interest.

Much of Professor Lipowski's writings and much

of this book have been concerned with the organisation of services in North American hospitals. The British reader will have quite different anxieties about funding and must also keep in mind that American liaison is providing psychiatric care in a system in which there is little primary care, no psychogeriatrics and no comprehensive catchment area psychiatric services. Very few will attempt to read the whole book but it provides an important and well-indexed reference work by our leading liaison psychiatrist. It is not a textbook of liaison psychiatry but it is the nearest thing that we have. Long may Professor Lipowski continue to scan the medical literature and keep us informed of what is going on.

RICHARD MAYOU, *Clinical Reader in Psychiatry, University of Oxford*

A Manual of Practical Psychiatry. By P. E. Bebbington and P. D. Hill. Oxford: Blackwell. 1985. Pp 405. £19.50.

This large, strong paperback is introduced as a "bench book for the ward or clinic", that is, it is designed to be full of practical information for psychiatrists in training. It is committed to "clarity in assessment and the principle that this should lead to a logical programme of management". Detailed accounts of psychotherapy are deliberately not attempted, but plans of management do discuss such matters as family and social management, and ways of talking to patients.

There are chapters on organic psychiatry, schizophrenia, affective disorders (which comprise mania, depression, anxiety, phobias and obsessional neurosis) and behavioural disorders (alcohol, drug abuse, anorexia nervosa, obesity, insomnia, sexual disorders and personality disorders). Investigations and physical treatments are treated separately in a chapter of their own, and there are helpful sections on the rehabilitation of chronic patients, manipulative patients, violent patients, general hospital psychiatry and "working with colleagues". There is a very full chapter on child and adolescent psychiatry, another on mental handicap, and one on "UK law" (actually British law, as Scottish law is covered but not Northern Irish).

The result is very unusual for a general textbook on psychiatry. There are few discussions of aetiology—none at all for depression, and less than a page for alcoholism. Some ubiquitous old friends such as Kraepelin, Bleuler and Schneider are not mentioned. Instead there are discussions of symptoms and syndromes, and many long lists of headings under which to collect information for assessment of

patients, and for drawing up many-sided plans of treatment. Direct advice is frequently given to the reader on how to handle situations, and specimen remarks to try out on patients. In general the standard is elementary and would be most appreciated by a beginner trainee, but there are also detailed sections where the authors' fondness for lists is impressive and unusually useful (a clear account of admission procedure under Section 47 of the National Assistance Act; 60 syndromes associated with mental handicap; 13 different powers of criminal courts). The flavour overall is strongly of medical psychiatry: this is of course emphasised by the omission of advanced psychotherapy, and can be illustrated by, for example, the 2½ pages on neurosyphilis, the full account of rare physical complications of alcoholism in contrast with only a paragraph on depression and alcoholism, the limitation of psychological testing to tests for organicity, and clinical psychologists doing counselling but not psychotherapy.

There is a mistake on p. 349 concerning consent to treatment under the Mental Health Act: contrary to what the authors state, the patients whose urgent treatment is covered only by principles of common law are those who are informal and those detained under 72-hour sections. Those detained for assessment for 28 days or for treatment are covered by Part IV of the Act. It is unfortunate that the section on Scottish Law is stuck with the transient Mental Health (Amendment) (Scotland) Act 1983, rather than the definitive Mental Health (Scotland) Act 1984, because the numbers of sections are different and, by bad luck, seriously confusing (Section 24 in 1983 was the standard admission procedure for treatment, but in 1984 Section 24 has become the procedure for emergency admission).

Unfortunately there are many flaws in the presentation of the book, arising apparently from poor revision. There are numerous grammatical mistakes, awkward sentences, colloquialisms, terms not explained, and minor inconsistencies between one part of the text and another, which should be improved before any re-issue or new edition.

ANDREW C. SMITH, *Consultant Psychiatrist,
Greenwich District Hospital, London*

Observing the Erotic Imagination. By Robert J. Stoller. London: Yale University Press. 1985. Pp. 228. £20.00.

The central theme of this book, one that Stoller has argued for some time, is that erotic excitement depends upon some measure of hostility towards the opposite sex, and that this arises from early

childhood maltreatment. In their sexual fantasy and behaviour people are seeking (unconsciously) to gain revenge for perceived injustices in infancy, constructing for themselves scripts or scenarios that relate to the critical childhood incidents but which are twisted in such a way that "trauma becomes triumph".

This is a fascinating hypothesis; unfortunately, I do not find it convincing. The idea of a link between sex and aggression is reasonable and does have scientific support, but the dynamic interpretation strikes me as far-fetched. One obvious objection is that it does not explain how non-human animals are sexually aroused; are we supposed to presume that monkeys and rats are also trying to undo childhood traumas in their mating behaviour? Surely it is more parsimonious to adopt the evolutionary view which supposes that male animals are genetically programmed to be sexually aroused by reproductive females, even though the natural course of events may be distorted by brain damage, biochemistry, imprinting errors and learning experiences.

Within this framework, the major facts of sexual behaviour and deviation, which psychoanalysts find so puzzling, are easily understood. For example, it is no surprise that perversion is an almost entirely male phenomenon; females do not have to acquire sex targeting mechanisms in the same way as males. The "dehumanising" of women that is so characteristic of male fantasies and pornography is not for the purpose of gaining revenge against mummy or nanny but is a natural aspect of male novelty-seeking (The Coolidge Effect). Relationship building can be detrimental to the survival of male genes even though it may benefit female genes.

In discussing the origins of male homosexuality, Stoller makes valid criticisms of other psychoanalysts and naive conditioning theorists but the "logic" by which he arrives at his own Oedipal theory is totally unsatisfactory. He cites a Melanesian society called the Sambia in which young boys are taught to fellate older boys in order to gain the masculinity provided by their semen. Since learning theorists would predict that this would lead to near-universal homosexuality in adulthood, yet nearly all Sambia men adjust easily to marriage, Stoller says this proves that childhood intimacy with the mother must be where heterosexuality is learned. If that is so, then why do girls not turn out to be lesbian (since they are equally intimate with their mother in childhood)? And why is the epigenetic (biological) alternative of natural heterosexuality not considered, when it is more parsimonious and at least equally compatible with the data?

Stoller found one Sambia man who did turn out

to be homosexual in adulthood and he explains this as being due to the absence of the father. "It is common knowledge among analysts that one of the contributions to some of the homosexualities is a hopeless, awful yearning for an unreachable father" (p. 128). This typifies what is wrong with this book and psychoanalysis in general. What Freud "says" or Fenichel "believes" is treated as more important than what researchers have found through carefully designed empirical studies. There is a vast scientific literature on sexual arousal and the origins of male sex variation which Stoller ignores (and one of its conclusions is that absent fathers do not produce homosexual sons).

GLENN D. WILSON, *Senior Lecturer in Psychology, Institute of Psychiatry, University of London*

Repertory Grid Technique and Personal Constructs: Applications in Clinical and Educational Settings. Edited by NIGEL BEALL. Beckenham: Croom Helm. 1985. Pp. 407. £22.50p.

According to its dust-jacket, this book has three major aims: (1) to illustrate the use of grids in clinical and educational settings, (2) to demonstrate how grids are being used both in research and practice, and (3) to provide a source book for students and practitioners. It is organised into six main sections. The first gives examples of various clinical applications to which grids have been put and includes articles in which the subjects were children with emotional and behavioural problems, young offenders, women with weight problems, and female agoraphobics. The second section reports studies of the use of grids with people with various disabilities: aphasia, amputations and severe physical disabilities. The third section comprises two chapters concerned with the use of grids in the evaluation of change. Next comes a section of papers demonstrating the use of grids in exploring aspects of interpersonal relationships. The fifth section reports a number of educational applications and the sixth comprises two papers examining the construing by (non-handicapped) adults of mentally handicapped children. In addition to these six sections there is a good introductory chapter on the main approaches to administering and analyzing grids and an equally good final chapter devoted to a critical analysis of the validity of repertory grids.

The 22 British-based contributors maintain a uniformly high standard of writing. The book as a whole conveys the diverse and imaginative ways in which repertory grid methods and personal construct ideas are now being used. My only regret is that the section concerned with mental handicap could not have included something on the use of grids with the mentally handicapped themselves, rather than with their parents and carers. In general, however, this book succeeds admirably in its three main aims.

TOM TRAUER, *Top Grade Clinical Psychologist, King's College Hospital, London*

A Guide to Medical Endocrinology. By TAREK HASSAN. London: Macmillan. 1985. Pp. 269. £17.00 (hb) £8.95 (pb).

The author's premise for this short book is outlined on the inside flap of the front cover. Prof. Hassan's intention was to produce a book primarily for undergraduates which not only incorporated up-to-date facts about medical endocrinology (including all the essential endocrine physiology as an "integral part of an intellectually convincing clinical approach") but also to remind readers of the importance of a patient's non-medical needs. Unfortunately, neither of these commendable intentions are met. It is an extremely difficult objective to combine a comprehensive account of fundamental endocrine principles with a philosophical discussion about their biological and social relevance and still maintain an undergraduate's interest. Moreover, I suspect that few clinical medical students are intellectually adjusted to appreciate, as the author rather laboriously puts it "a perception of the dynamic and multidimensional nature of life". In addition, any text which includes statements such as "... seen through that tragic misorientation, biological realities are just elements to be submitted and subdued or, at best, tolerated grudgingly until they are cured or grown out" will serve to distract and dissuade rather than to inform an enthusiastic mind. The hope that this approach might appeal more to postgraduate readers, particularly those with an interest in the psychological sciences, is again unfulfilled by the nature of too basic an approach and presentation of endocrine detail. Indeed, some chapters are more suitable for sixth form biology text rather than a clinical manual, and highly important information about recent advances is omitted. Disappointingly, there are only a few obscure paragraphs about the psychological and psychiatric elements of endocrine illness. For example the statement that "bodily enactment of repressed material together with body and emotion integration in communication within the group sometimes leads to relevant progress in the management of the problem of obesity" gives little practical guidance for the management of this condition whatever the experience of the reader. It is also disappointing that the author has not included any reference to altered endocrine function in association with psychiatric disease nor the psychiatric manifestations of endocrine disorders, two fields of increasing importance. The author's reasoning behind this book is admirable but I fear that it has merely highlighted an important need. If this should be the case then the book itself will have served a useful purpose, but I cannot recommend it as a necessary reference text for any bookshelf.

P. G. KOPELMAN, *Senior Lecturer in Endocrinology, The London Hospital*

The Ego Ideal: A Psychoanalytic Essay on the Malady of the Ideal. By JANINE CHASSEGUET-SMIRGEL. (Translated by Paul Barrows. Introduction by Christopher Lasch). London: Free Association Books. 1985. Pp. 271. £18.50 (hb), £7.95 (pb).

In the subtitle of this book: *A Psychoanalytic Essay on The Malady of the Ideal*, Chasseguet-Smirgel describes

the essential paradox explored in her essay. By speaking in the same breath of a "malady" (which she states is universal) and of an "ideal" (the ego ideal), an apparent tension is exposed, she argues that this tension can promote psychosexual maturation, but also offer developmental hurdles and cul de sacs. From the outset, however, there seems to be an intention to establish the notion of the ego ideal as a natural force which can function as an internal guide or agency—a maturative force.

This is a scholarly work which is at times acutely intellectual and at others feelingful. Chasseguet-Smirgel refers skilfully to the relevant works of Freud and others. In contrast to Freud, she establishes that a clear distinction is necessary between the function of the ego ideal and that of the superego, and argues this is helpful both in understanding clinical material and in accurately describing developmental processes. Her examples which flesh out this argument, are drawn from art, literature and patients, and give the book a flow and life which was invaluable for the reviewer.

A reading from start to finish will be a not inconsiderable undertaking, but one which is likely to stimulate thought in different and perhaps surprising directions. If a "dip" into this work is made by reading chapters five and eight, the reader is likely to gain access to the core and spirit of the book. A number of her theoretical assumptions may be experienced as arguable. However, Chasseguet-Smirgel has written a challenging and thought-provoking book, which extends psychodynamic thinking in a style which seems to have intended debate.

PETER FULLERTON, *Social Worker, Emanuel Miller Centre for Families and Children, London*

Adolescent and Developmental Breakdown: A Psychoanalytic View. By MOSES LAUFER and M. EGLE LAUFER. London: Yale University Press. 1985. Pp 225. £18.00.

This curiously timeless book, well referenced, by two practising psychoanalysts who are trainers and researchers, is of interest to the student of psychiatric history and presents a clear, though uncritical, limited perspective on adolescence as a theoretical concept. The authors' working framework remains undefined and undiscussed and their book does not offer guidelines to practitioners within any setting with service commitments and limited resources.

Referrals, from unidentified sources, are of men and women of sixteen years and upwards and analysis lasts for periods of three to five years with attendance less than five times weekly being a cause for concern. Money is never mentioned.

The authors, although they emphasise the importance of engaging with the parents of parasuicidal men and women, their only concession to systems theory, work exclusively with their patients and only once is a patient seen first, with her parents, at under sixteen years of age. Hospital admissions are discussed only within the context of the analyst's visits to his patients.

Thus the authors avoid discussion of consent to treatment by under sixteen year olds, nor do they discuss direct analytical work in early adolescence, their theoretical construct of this period being obtained by retrospective

discussion of data obtained from work with young men and women.

Their account of adolescence is relentlessly sexual yet, though some histories indicate the possibility of infra-familial sexual abuse, this term does not appear in the index. "Incest barrier" and "incest fantasies" are indexed but not "incest". Outcome is not discussed.

This is a frustrating book. One might welcome fewer case histories and rigorous, critical discussion of psychoanalytic theory or a contextualised study, with regard to economic reality, or a service for alienated young men and women attending the Brent Consultation Centre. Better still would be a revised book expanded in both directions.

JEAN HARRIS, *Consultant Psychiatrist, Child and Family Psychiatric Clinic, Dunstable, Bedfordshire*

A Method of Psychiatry. Edited by STANLEY E. GREBN, VIVIAN M. RAKOFF and GEORGE VOINESKOS. Philadelphia: Lea & Febiger. 1985. Pp. 475. £32.75.

Faculty members of the Department of Psychiatry at the University of Toronto wrote this book because they could not find a 'good and comprehensive' text which reflected their method of teaching the basis of psychiatry: i.e., 'broadly-based eclecticism'.

On epidemiological grounds, some might question their neglect of the role of the primary care physician in the provision of mental health services, and the scant attention paid to mental retardation (10 pages) and disorders of old age (7 pages). An academic might be pleased by the extensive reference lists but disappointed that more attention was not paid to emphasise research as a component of education.

GREG WILKINSON, *Honorary Lecturer, General Practice Research Unit, Institute of Psychiatry, London*

Child Sexual Abuse within the Family. CIBA Foundation, Edited by RUTH PORTER. London: Tavistock. 1984. Pp. 156. £12.95 (hb), £5.95 (pb).

In dealing with child abuse we are dealing with a matter of public concern. This account of the CIBA Foundation Study Group's examination of the issue of child sexual abuse and its management is a welcome addition to the literature in this area. It forms a basis of good practice for multidisciplinary groups dealing with children and their families with these problems. Its management guidelines are clear and concise for the beginner in the field. It examines the problems from psychiatric, social work and legal standpoints and emphasises the present punitive attitude to perpetrators of child sexual abuse, whilst recommending a more constructive approach within a legal context, a necessary comment in view of the fact that whatever the faults of the present situation, the abuse certainly ceases with the legal consequences of such abusive acts coming to light. It is perhaps less helpful as one might expect in a new field in those grey areas of practice where suspected cases are discussed rather than proven cases.

As in so many works in this area the biggest problem is that we do not know the prevalence of the disorder or its actual acute effects or long-term consequences in any detail. Most accounts are written from a somewhat partisan point of view, a criticism not shared with this particular work. I would recommend it to trainees and their trainers in psychiatry, social work education and the law. The climate of opinion about child abuse, and in particular child sexual abuse is changing. There is a danger that many of the statements made cannot be supported by facts. This book is a healthy corrective to some of the more vehement public statements and should be seen as a reasoned approach to this group of disorders.

L. G. SCARTH, *Consultant Psychiatrist, Royal Hospital for Sick Children, Edinburgh*

Handbook of Clinical Behaviour Therapy with Adults.

Edited by MICHEL HERSEN and ALAN S. BELLACK. New York: Plenum Press 1985. Pp. 732. \$75.00.

Is there an up-to-date book that tutors can confidently recommend as a basic handbook for the increasing numbers of psychiatrists who want to learn more about clinical aspects of behavioural psychotherapy? My hopes were raised on receiving this book, since both editors are well known for an extensive contribution to the behavioural literature over the past 15 years. However, I was soon disappointed.

In the second chapter we are told that systematic desensitisation based on relaxation is the major orthodox behaviour therapy approach to simple phobias. In the next, that most clients with agoraphobia require between six and 18 months of therapy. Both claims are grossly out of touch with orthodox teaching. Instead they reflect the idiosyncratic views of individuals; the first faithfully reiterating the teaching of Joseph Wolpe; accurate 30 years ago but long since superseded; the second, the provocative but unsubstantiated speculations of a worker committed to broadening the base of the subject. This is not a handbook at all. It is a collection of articles which could better be titled "The way we do it in . . . Rhode Island/Pennsylvania State University/Gainesville/New South Wales/ etc . . ." Inevitably there are some first-rate chapters particularly those on the treatment of obsessive-compulsive neurosis; stress and generalised anxiety; cognitive approaches to depression and work and study problems. But this excellence is more than off-set by the rest. It is intensely irritating for the reader of a clinical handbook, 732 pages long, to find many techniques, such as Progressive Muscular Relaxation, Biofeedback, Operant methods for Control of Pain, and Stimulus Control advocated, but then to find no description of how to carry them out. Even worse, as in the case of Autogenic Training is to be referred for further details to an account which is "in press". Many chapters are concerned with speculative accounts of psychopathology rather than therapy, and much of the case material is unhelpful. Feminists will welcome the chapter on sex role considerations, but find it of little clinical relevance.

In such a large book there are surprising omissions. For example, no description of the treatment of habits and tics,

and no mention of sensate focus. The index is woefully inadequate for a book that must aspire to be a reference work.

We need a new handbook which is both practical and basic, and whose editors opt for quality not quantity. This attempt fails on all these criteria.

JOHN COBB, *Consultant Psychiatrist, The Priory Hospital, London*

Psychiatric Nurse Therapists in Primary Care. By ISAAC M. MARKS. London: Royal College of Nursing of the United Kingdom. 1985. Pp. 117. £4.00 (UK), £6.00 (overseas).

Isaac Marks has played a significant and pioneering role in fostering the growth of psychiatric nurse therapists. He has also been tenacious in documenting, analysing and evaluating their roles and functions. This publication, written in collaboration with colleagues, is Isaac Marks' latest contribution to the body of knowledge about nurse therapists. It presents the methodology used in, and the clinical and economic outcomes of, the first controlled study of chronic neurotic patients who were cared for by psychiatric nurse therapists in primary care settings.

The study was conducted over three years: 'control' patients were treated by general practitioners. The nurses were more effective. Health questionnaires to GPs were also used to determine the most suitable catchment population for a nurse therapist. The report suggests 'a population of about 30,000 served by 12 general practitioners'. A national follow-up study of British nurse therapists was also conducted, which indicated that most continue as nurse therapists after training.

An interesting aspect of the research is the cost-benefit analysis which suggests that using nurse therapists is cost-effective for the NHS and the patient. But this is based on nurses' salary levels! Much more rigorous work still needs to be done on cost effectiveness—in all aspects of care within the NHS.

This report must be satisfying to the author for it supports his own commitment and ideology. But if nurses are taking over functions from psychiatrists and GPs, it would be interesting to know how the medical resources saved as a result are used. Do other patients get more and/or better care? Are the saved resources transferred to nursing budgets? Furthermore, does the usefulness of nurse therapists suggest that the expensive training and salary levels of psychiatrists and GPs is an unnecessarily costly burden for the NHS? Perhaps some brave researcher might like to try to find out.

JAMES P. SMITH, *Assistant Director, Long Term and Community Care, King's Fund Centre, London*

Animal Models in Psychopathology. Edited by NIGEL W. BOND. Orlando, Florida: Academic Press. 1985. Pp. 318. \$29.00.

Bond, like editors of similar volumes before him, proposes that an adequate study of human psychological disorders

and the mechanisms underlying them can be made through a consideration of animal models. These he defines as "heuristic devices" from which one extrapolates to the human condition by the argument from analogy. Little emphasis is placed on the "accuracy" of these models: indeed no existence claims need be made for them. Bond argues that an animal model of schizophrenia, for example, need not show characteristics that are homologous with the clinical condition.

These propositions are likely to raise the hackles of those who see little scientific or ethical value in animal experimentation. They leave wide open the criticism that the choice of animal models in psychiatric research is based on vague, arbitrary or simplistic criteria. Although due consideration is given in the introduction and by some of the contributors to the validity of their models and the ethics of their use, the overall case is less than totally convincing.

A more advanced and satisfactory scientific approach is to develop theories based on a synthesis of careful and well-controlled clinical, behavioural, anatomical and physiological observation. Animal studies must then be necessary for the testing of hypotheses, including behavioural ones, that cannot practically or ethically be carried out in humans. Thus there is no further need to speak of experimental preparations as "models". Within this framework, perhaps the best elaborated multidisciplinary theories in psychopathology concern the affective disorders and anxiety/the neuroses, neither of which are considered in detail in this book; although they have been given extensive treatment in other texts. Nevertheless, the most satisfying contributions to this volume pursue their subjects with a strong interdisciplinary approach. Particularly noteworthy are the chapters on obesity, anorexia and the memory disorders.

The canvas of psychopathology painted in this volume is very wide, and there is little overlap in content with other volumes of this general title. Apart from the above topics, the subjects presented range from the teratogenic effects of alcohol and the toxic effects of environmental substances to the aetiology of stress influences on cardiovascular disease and the effects of psychoactive drugs on brain reward processes. In some chapters there is excessive concern with individual experimental methodologies, which, while of interest to scientists in the field, is perhaps beyond the scope of the general overview presented by this volume.

J. D. SINDEN, *Psychologist, Institute of Psychiatry, London*

Advances in Psychosomatic Medicine. Volume 13: Interface between Neurology and Psychiatry. Edited by M. R. TRIMBLE. Basel: Karger. 1985. Pp. 192. \$48.50.

This authoritative volume advances the cause of psychosomatic medicine. Volume 13 of the series selects problems, disorders and systems in the neurological field and presents eight chapters written by acknowledged experts who explore the psychological aspects. Trimble has gathered work on the limbic system, head injury, cerebrovascular disease, multiple sclerosis, movement disorders and epilepsy with chapters on the social environment and behavioural psychotherapy.

The articles are well documented and researched and the bibliographies appended at the end of each chapter and referenced in the text, are wide and exhaustive. This is a mature view of psychosomatic medicine, there is no analytic or dynamic softness but a genuine attempt to link observed behaviour with the emotions experienced and to identify the neurological pathways and the areas in the brain mediating these psychological events.

Gray's chapter on 'Emotional Behaviour and the Limbic System' sets the scene but because the necessary research examples are animal based the view of anxiety presented is at the level of animal behaviour and misses the opportunity to discuss anxiety created in man by moral and spiritual conflicts and relationship dilemmas.

Grant's chapter on the 'Social Environment' moves towards the clinicians interest with consideration of life events and their importance to various neurological crises, to be explored by later authors in more depth. Sims' interesting chapter on 'Head Injury, Neurosis & Accident Proneness' includes a history of the development of psychosomatic ideas and usefully discusses the concepts, before postulating a triangular relationship between his three key conditions.

In the last chapter Cobb gives disappointing views of the limitations of behavioural psychotherapy, but this realistically shows that learning theory must be applied cautiously to man.

The book is well produced in hardback with good quality paper and printing. Medical and psychiatric libraries should stock this challenging volume and will probably wish to examine the other volumes in the series.

DAVID TOMS, *Consultant Psychiatrist & Clinical Teacher, Nottingham University*

Psychiatric Aspects of Chronic Pulmonary Disease. Edited by W. L. THOMPSON and T. L. THOMPSON II, Basel: S. Karger. US\$47.00, SFr. 110.00 DM 132.00

The editors of the book rightly comment in their introduction that although psychological and social problems associated with chronic lung disease are common, little has been written about them. To this extent, this book is welcome, since it provides a source-book of ideas and references for those with a particular interest in this area, even though there appear to have been few advances to justify the series title. It is much less likely to satisfy the wide audience to whom it is said to be directed. I found the sections on medication and occupational diseases particularly interesting but few of the chapters are easy to read and the content is by no means comprehensive as there are no chapters on the nature and assessment of breathlessness, on lung cancer or on hyperventilation. It is also a rather parochial book since six of the eleven chapters are by authors in Denver and another chapter on the important subject of chronic bronchitis is by a writer whose partial view is buttressed by nineteen references, sixteen to himself. It is sad to see this series becoming ever more expensive for ever slimmer volumes. A book for the experts.

RICHARD MAYOU, *Clinical Reader in Psychiatry, University of Oxford*

The Clinical Guide to Child Psychiatry. Edited by DAVID SHAFFER, ANKE, A. EHRHARDT and LAURENCE L. GREENHILL. New York: The Free Press. 1985. Pp. 619. \$45.00.

This book is described by its publishers as *the* definitive source of practical guidance for those who need to deal with the full spectrum of childhood psychiatric disorders. It contains 31 chapters divided into three groups; the treatment of specific disorders, diagnostic techniques, and treatment approaches. Other than Michael Rutter, who writes on autism, all the authors work in the United States, mainly child psychiatrists, but also some psychologists and social workers.

I found myself torn over this book. There is much that is very good and many of the individual chapters are excellent. I could not, however, help comparing it unfavourably with the major text book published in this country by Rutter and Hersov (Oxford: Blackwell. 1984). It is not that one would necessarily obtain a more authoritative account of a particular syndrome in the latter. It is rather that a different approach seems to underlie the two books, approaches which probably reflect the current state of child psychiatry in Britain and the United States. The disquiet which I found myself experiencing over the Shaffer book relates more to where child psychiatry is and where it is going in the United States, rather than the book itself. It reflects a scene where child psychiatry is becoming a uniprofessional medical speciality. Cynically, one might wonder whether this is to justify insurance payments. There can be few psychiatrists in this country who have not at times wished that they could free themselves from

the constraints of multi-disciplinary work and concepts. Reading this book however had a salutary effect on me and I thirsted for a wider bio/psycho/social model for both the origins and treatments of childhood disorders. There are only limited attempts to see the child as a developing organism, fully understandable only in the context of both its family and the wider society in which he or she lives. The narrow view is seen at its most extreme in the chapter on major depression. There is a detailed account of the recognition of the disorder, the use of the laboratory tests and pharmacotherapy. There is no mention of the possibility that the depression could, in a child, be due to abuse by parents or others.

There seems throughout relatively little doubt and insufficient self criticism. This approach is accompanied by a great need to emphasise that child psychiatrists are identical to any other physician who deals with individual patients. For example, child psychiatrists are justifiably criticized for their negative attitudes to physically examining a child, but should a rectal examination really be part of the normal routine?

Having made these complaints, I must again reiterate that there is much to learn from this book. There are for example, useful chapters on stammering, abnormal puberty and the divorcing family. This probably is the best guide to American child psychiatric practice at the present time. It is not, however in itself sufficient to educate the trainee child psychiatrist.

STEPHEN N. WOLKIND, *Consultant Child Psychiatrist, Maudsley Hospital, London*

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Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey

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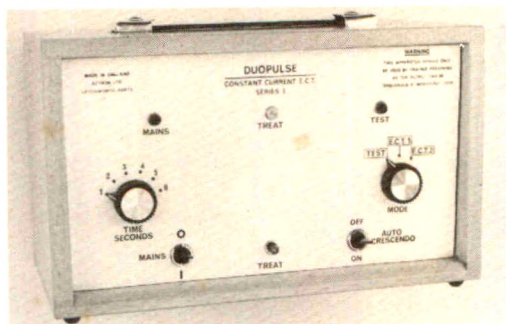
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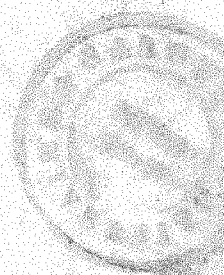
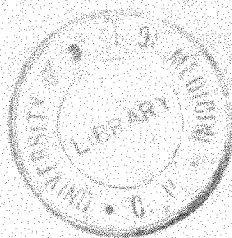


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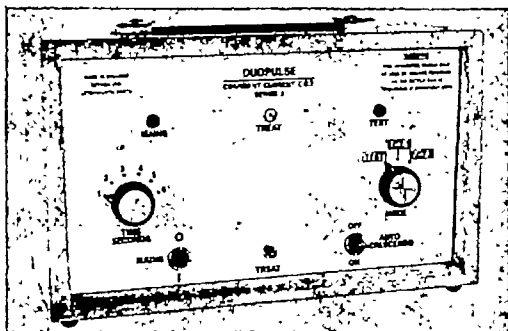
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Community Care of the Acutely Mentally Ill*

JOHN HOULT

Deinstitutionalisation is a two-part process: the first involves discharging patients from hospital to the community, which has been done in many countries. The second part—the provision of adequate resources for good community care—has still to materialise almost everywhere. In New South Wales, the inpatient mental hospital population dropped by two-thirds in 20 years, yet the total number of hospital staff increased considerably during this time; on the other hand, in the community, where most of the patients are, there are relatively few staff. This has important consequences: it means that for many patients, their episode of illness has to progress unchecked until it is of sufficient severity for them to be admitted to hospital. When that happens, there will then be enough staff to give them good quality care, so that their symptoms can subside, but afterwards there will again be hardly anyone available to help maintain them and to prevent relapse.

To the staff working in a hospital, a patient's admission is the beginning of a treatment episode, but for the patient and his family, it is the culmination of weeks or even months of increasing suffering which often could have been relieved much earlier, if help had been available. Instead, they may be forced to wait, as their distress intensifies, until the patient's symptoms and behaviour are of such severity that admission to hospital, sometimes even compulsorily, becomes the only option. Hospital admission gives the relatives temporary relief, and alleviates the patient's symptoms and more disturbed behaviour, but before long he returns home; in the case of a patient with schizophrenia, either his apathy or his unpredictability may then again cause concern to his relatives, who have to wait for the next relapse.

A relative of one of the control group patients in our research project described an experience common to many: "The only relief we get is when she is in hospital. She gets violent and doesn't seem to be managed in any other way. She never comes out cured—she gets much better, but it doesn't last more than a month."

Excessive reliance on admission to hospital as the way to deal with the acutely mentally ill involves another problem. Doctors working in hospitals focus on psychopathology: that is what they have been trained to do. Patients and relatives quickly learn that this is what doctors are interested in, and report their difficulties in these terms. This is not to deny the importance of symptoms and signs; patients suffer much distress from them, and it is our duty to offer relief. But many times, the underlying interpersonal stresses which have precipitated the symptoms and signs are ignored, or are not adequately dealt with, and so they persist to precipitate further episodes, which are again dealt with only in terms of psychopathology. Eventually, the patient takes on the role of patienthood, community coping skills get lost, and relatively minor stresses lead to symptoms which lead to further admission. Several research studies have shown that the best predictor of a patient's future use of hospital is the amount of previous hospitalisation he has had (Strauss & Carpenter, 1984; Wynne *et al*, 1978).

In order to interrupt the "revolving-door syndrome", with its high readmission rates, so that resources can be more equitably distributed from the hospital out into the community, where the bulk of the patients are, a means is needed for dealing with the acutely mentally ill in the community.

Review of the published research

The two major changes in the care of the mentally ill—institutionalisation, then deinstitutionalisation—were not done on the basis of research results; it is

*The First Upjohn Lecture on Mental Health Care Delivery given at The Annual Meeting of the Royal College of Psychiatrists 11 July 1985.

only in the past 20 years that controlled studies, comparing one form of service delivery with another, have appeared in the scientific literature. Those studies which compare community alternatives to mental hospital care for the acutely mentally ill have been reviewed by Braun *et al* (1981) and by Keisler (1982). In none of these studies is there any instance in which hospital treatment was superior to the community alternative for the average patient. It could be that, concealed in these aggregate results, are sub-groups who would show marked benefit from hospital treatment. Should further research show this to be the case, it would mean that for some or all of the rest of the patients, hospital treatment was correspondingly less beneficial. The alternative treatments produced outcomes which were equal to, and occasionally superior to that of the control groups, but good outcome appeared to depend on comprehensive and continuous programmes of care in the community. Some of the researchers (Davis *et al*, 1974; Stein & Test, 1980; Fenton *et al*, 1979) followed-up the patients after the experimental treatment ceased; there was little carry-over effect. The improvement of the experimental group patients soon faded, and before long their outcomes were indistinguishable from those of the control group patients.

Stein & Test's study is generally regarded as the best; they had good methodology, excluded the fewest patients from their study, and provided a comprehensive community care programme, delivered in an assertive manner by a relatively large team. Their results were impressive: at each of the four-monthly evaluations during the experimental programme, the experimental group patients were less symptomatic and had better social outcome, compared to the control group. They were not an increased burden on their relatives or to the general community: only 12 out of 65 experimental group patients were admitted to hospital, compared to 58 out of 65 control patients.

Background to the Sydney study

Since 1974, I had worked in a community mental health centre in North Sydney, and had experienced frustrations in my work because of the problems mentioned earlier. In searching the literature for a model for improving the quality of the service, I came across the early reports by Stein & Test. Although Sydney is a city of over 3 000 000 people, and Madison, Wisconsin had only 300 000, their programme appeared to have the necessary ingredients to make it a model for a general method of service delivery, suitable for Australia. However,

the implications of this way of working seemed so important that the original research ought to be replicated, and at a level more in keeping with the level of staffing which could be expected in Australia. Consequently, I obtained a grant from the Australian Government to conduct the research, while the New South Wales Health Department provided extra assistance, in what were increasingly difficult financial times.

The aims of the study were: (1) to demonstrate that it is feasible to treat psychiatric patients in the community as an alternative to mental hospital admission; (2) to show that this can be done without detriment to the patients, their relatives, or the community; and (3) to demonstrate that such treatment costs no more than standard hospital care and after-care.

Method

The sample consisted of those patients who presented voluntarily or were taken involuntarily to Macquarie Hospital for admission over a five-month period, beginning September 1979, and who met the following criteria: (1) resident in the Lower North Shore or Manly-Warringah local government areas of Sydney; (2) aged between 15 and 65 years; and (3) not having a primary diagnosis of drug or alcohol dependence, organic brain disorder, or mental retardation.

Macquarie Hospital is the only state psychiatric hospital serving these two local government areas; it was built in 1959, and has a capacity of 300 patients. It is well staffed, of good standard, and used as a psychiatric teaching hospital for medical undergraduates of the University of Sydney and for post-graduate training by the Royal Australian and New Zealand College of Psychiatrists. In the two areas studied, there is a great diversity of housing—ranging from spacious bungalows to high density multi-storey accommodation; although largely middle-class, it contains significant pockets of poverty and social disorganisation, where many ex-hospital patients congregate in cheap boarding houses. The hospital tends to cater for those of lower socio-economic status and those with the more severe and chronic conditions.

As each patient presented, the Admission Office nurse opened a sealed envelope in which there was a card, allocating him to either the community treatment (experimental) or the standard hospital care and after-care (control) group. No patients who met the eligibility criteria were excluded from the study, so that even the very ill (e.g. acutely psychotic or suicidal) were included. The intake period ended when each group contained 60 patients.

The control group received standard hospital care and after-care, which usually consisted of admission for an average of three weeks to one of two 35-bed wards, each staffed by a fully qualified psychiatrist, three to four trainee psychiatrists, 15 nursing staff, two social workers, one psychologist, and one occupational therapist. Most patients were initially seen by medical staff every day, and

medication was given on the basis of their assessments. The majority were also seen by social workers. The ward treatment programme included large and small group discussions, arts, crafts, work, and sporting activities. On discharge, most patients were referred for after-care to one of the six Community Mental Health Centres in the catchment areas; these are situated in major shopping areas, close to public transport, and after-care of discharged patients is one of their main functions. At the centre, the patient is assigned to a case manager from amongst the multi-disciplinary team; his task is to organise follow-up care, which may consist of regular medication review by a staff psychiatrist, counselling or psychotherapy, as well as individual, social, and family support. However, standard aftercare tends to be of a "passive-response" nature, because the initiative is often left to the patients and their relatives, while home visits are not common. Also, centre staff mostly work normal office hours, and are not available in the later evenings or at week-ends.

For experimental group patients, a member of the Community Treatment Team and its psychiatrist (myself) were called to the admission office. What we did there and later at the patient's home had basically the same ingredients as the management carried out on behalf of the control group patients—only the time frame and the commitment to community treatment were different. We used no special treatments which are not used routinely for patients who are admitted to hospital and later need maintenance care.

First, we made an assessment. Of necessity, this had to be much more detailed than usually happens at hospital; since the patient had a good chance of being taken back to the community, we needed to be sure we were making correct decisions. Relatives and other members of the social network were involved in this initial assessment, where possible. The areas we assessed included: (i) *The presenting problem*—it was important to define this accurately, to be sure of attending to it; while relatives may sometimes say they want help for the patient's delusions or hallucinations, what really has made the situation intolerable is, e.g. the patient's restlessness, or unpredictability, or the potential for violence. Successful community treatment demands that this be confronted from the outset. (ii) *Clinical symptoms and signs*—leading to a diagnosis. A physical examination was carried out by the psychiatrist at the admission office. (iii) *Behaviour*—it was essential to be sure that this would be tolerable to the people who would be in the patient's environment, otherwise community treatment would quickly break down. (iv) *Interpersonal relationships*—the nature and quality of these determined whether the acutely ill patient could return home or needed alternative accommodation to be found immediately. It also alerted us to the need for treatment at a later date. (v) *Social supports and needs*—accommodation, money, and supervision were the immediate needs of all patients treated outside hospital. If they were not available, we would have to provide them. (vi) *Willingness to co-operate*.

Both the patient and his relatives were then actively involved in making management plans: options were examined, and explanations and advice offered. The team was prepared to negotiate to ensure that a plan acceptable to all was arrived at, and that it had a reasonable chance of

being adhered to. These plans included medication (with rapid tranquillisation if necessary), an expectation that the patient should behave as "normally" as possible, decisions as to where the patient was going to go, and how he would be supervised. Patient and relatives were given information about the illness and its expected course, explanation about the Community Treatment Team's methods of working and for the relatives, guidance about how to deal with the patient's symptoms and behaviour. Threats of violence and suicide usually subsided with support, firmness, and medication. It is surprising how often a solution appears for a seemingly intractable problem in a disturbed patient, if one talks to him and his relatives for sufficient time.

If the psychiatrist considered it feasible, the Team then returned with the patient to the community setting, and immediately continued treatment. However, if the situation in the home between the patient and family appeared untenable, the Team took the patient to alternative accommodation, such as a boarding house, and were responsible for his care there. These were patients whose symptoms and behaviour were predominantly the result of disturbed interpersonal relationships, and whose behaviour quickly improved when they were removed from the relationship. This improvement—in a location other than hospital—was important learning for the relatives, helping them to realise the contribution to the disturbance that the interpersonal conflict made, that the patient could control his behaviour and that repeated hospital admission was not a solution to the recurrent disturbances. Initially, staff might stay with the patient for many hours, monitoring his behaviour and the side-effects of medication given, and reassuring patient and relatives. Once the patient became more settled, the staff withdrew, but if necessary, relatives, boarding house managers and patients were encouraged to page us if there was any disturbance or if they were in any way worried, whereupon we would return, if necessary within 30 minutes. The 24-hour availability greatly reassured the relatives and the managers. We had previously arranged with certain boarding house managers for permission to use their facility for our patients; once they knew that help would be quickly available and we did not expect them to tolerate disturbed behaviour, they readily accepted our patients (and their money) and were helpful supervisors. The Team visited frequently for the first few days and even took the patient out with them for some hours to give relief to families. The medication was adjusted frequently, according to the patient's response, and necessary clinical investigations carried out at this stage. In most cases, quite a strong and enduring treatment alliance was formed over the first few days; patient and relatives learned to regard the illness in quite a different light, and learned new ways of managing it. As quickly as possible, the patient was expected and encouraged to be responsible for himself and resume normal duties. Where necessary, staff accompanied the patient on tasks of community living in order to assess, supervise, and train him in these tasks in the natural setting. For most patients, these activities by the staff soon decreased as the patient became less psychotic, but for those with chronic, severe handicaps, they continued on an on-going basis. Staff assertively

trained patients in adequate hygiene and grooming, as well as in the use of community facilities such as transport and supermarkets, day centres, and sheltered workshops.

Subsequently, the Team remained responsible for all experimental patients. In some cases, patients had a recurrence of their symptoms during the study year, but the Team was usually contacted early, and were able to intervene quickly and deal with the symptoms and the associated problems without needing to admit the patient to hospital.

Some acutely psychotic patients did need admission. These were patients who either fell asleep when given tranquillising medication at the admission office and so stayed in hospital overnight, or who refused initially to co-operate with treatment, and so had to be admitted so that they could be compulsorily treated. After two or three days, most agreed to co-operate, and so were discharged. A few chronic patients had an exacerbation of symptoms and were too disorganised in behaviour to be tolerated outside hospital; these patients needed longer stays. Patients who were admitted remained in the experimental group; they were supervised in hospital by the Community Treatment Team psychiatrist, and on discharge, were offered the same treatment as those who were not admitted.

The Community Treatment Team for the 60 patients in the study consisted of three psychiatric nurses, two social workers, one occupational therapist, one psychologist, and one part-time psychiatrist—none of whom had had previous experience with this kind of treatment programme. The staff were rostered on duty for two shifts every day: one staff member remained on call from 11.00 p.m. till 8.00 a.m. Training consisted mostly of role-plays of likely situations to be encountered at the admission office and at patients' homes; a problem solving approach was used. There were formal lectures on medication and recognition of its side-effects, and on systems and family theories; staff also familiarised themselves with the area and its resources. Two or three week full-time training seems to be adequate for professional staff.

Assessments were carried out at presentation and 1,4,8, and 12 months later by two research psychologists, who were independent of the Community Treatment Team. Structured questionnaires were designed to measure a wide range of problems and attitudes, both from the patient's perspective and that of his relatives. Clinical status and outcome were measured by, amongst other scales, the Present State Examination (Wing *et al*, 1974), while a cost-benefit study was also done.

Results

At baseline, there were no significant differences between the two groups on any of the characteristics measured. Three-quarters of the patients had been previously admitted to a psychiatric hospital, and almost one-third had spent one year or longer there. Three-quarters were diagnosed as suffering from one of the functional psychoses, and one-half suffered from schizophrenia. There were few (6%) neurotics. About 60% of both groups presented as involuntary patients. At 12 months, 88% of the experimental and 84% of the control group patients

were interviewed, while of the relatives, 80% and 78% respectively were interviewed.

The full details have been presented in the major report of the study (Department of Health, N.S.W., 1983a); only the main findings will be presented here. Tables I and II show a highly significant ($P < 0.001$) difference between experimental and control group patients in terms of number of admissions and length of stay in psychiatric hospitals or clinics (public and private) during the 12-month study period. With one exception, experimental group patients who were admitted for one week or longer were chronic patients (i.e. they had previously been admitted for one year or longer). Control group patients spend an average of 53.5 days in psychiatric hospitals; experimental group patients spent an average of 8.4 days.

Community treatment achieved a superior clinical outcome. At 12 months, PSE results showed that experimental group patients were significantly lower on the total score and on each of the four sub-scores (Table III). For both groups, psychotic symptoms had greatly declined since baseline, but non-specific neurotic symptoms were still prevalent, and 29% of the experimental and 43% of the control group patients still had sufficient symptoms for a diagnosis of psychosis.

Both patients and their relatives considered community

TABLE I
Number of admissions to psychiatric hospitals or clinics during 12 months study period

	Experimental (n = 53)	Control (n = 47)
None	32 (60%)	2 (4%)
1	17 (32%)	21 (45%)
2	2 (4%)	17 (36%)
3	1 (2%)	5 (11%)
4+	1 (2%)	2 (4%)

TABLE II
Length of stay in psychiatric hospitals or clinics during 12 months study period

	Experimental (n = 53)	Control (n = 47)
Not admitted	32 (60%)	2 (4%)
Less than 1 week	14 (26%)	11 (23%)
1-2 weeks	0 (0%)	6 (13%)
3-4 weeks	3 (6%)	5 (11%)
5-6 weeks	3 (6%)	8 (17%)
7-10 weeks	0 (0%)	4 (9%)
11-15 weeks	0 (0%)	6 (13%)
16 weeks or more	1 (2%)	5 (10%)

TABLE III
Mean Present State Examination total and sub-scores at baseline and 12 months

	Baseline			Twelve months		
	<i>Experimental</i> (<i>n</i> =58)	<i>Control</i> (<i>n</i> =57)	<i>Sig</i>	<i>Experimental</i> (<i>n</i> =52)	<i>Control</i> (<i>n</i> =46)	<i>Sig</i>
Mean total PSE score	30.5	30.9	NS	9.8	15.4	$P<0.001$
Mean DAH (delusional and hallucinatory syndromes)	8.2	8.4	NS	1.9	4.0	$P<0.001$
Mean BSO (behaviour, speech and other syndromes)	7.0	7.3	NS	2.0	3.1	$P<0.001$
Mean SNR (specific neurotic syndromes)	4.5	5.0	$P<0.01$	1.4	2.4	$P<0.001$
Mean NSN (non-specific neurotic syndromes)	10.5	11.6	$P<0.01$	4.2	6.0	$P<0.001$

TABLE IV
Summary of patients' and relatives' satisfaction with care received during the 12 months study period

	<i>Control*</i> (%)	<i>Experimental*</i> (%)	<i>Sig</i>
Patients very satisfied with advice and information	29	60	$P<0.01$
Patients very satisfied with support and help	33	65	$P<0.01$
Patients very satisfied with amount of supervision	12	56	$P<0.001$
Patients preferring admission to community treatment	25	12	NS
Relatives very satisfied with support and help patients received	28	83	$P<0.001$
Relatives very satisfied with the amount of supervision patients received	30	85	$P<0.001$
Relatives who themselves received enough support and information	39	88	$P<0.001$
Relatives coping better during study year than before	28	70	$P<0.001$
Relatives preferring hospital admission to community treatment	35	2	$P<0.01$

*The numbers were 53 experimental and 48 control patients and 48 experimental and 47 control relatives.

treatment to be significantly more satisfactory and helpful than standard hospital care and after-care (Table IV). The majority (80%) of experimental group patients who were not admitted to hospital were pleased and grateful about it; only 30% of control group patients were pleased and grateful about being admitted to hospital, whereas 39% were upset and angry about it. Nearly all relatives regarded the community treatment experience positively; they considered it to be significantly more helpful to the patients and themselves than standard hospital care and after-care (Reynolds & Houlst, 1984). A separate analysis of the responses of the 54% of relatives who lived with patients indicated that, for them, community treatment was no less satisfying and helpful: a relative of an experimental group patient said—"I like the idea that help can be reached any time day or night. I feel I am sharing the load. Mental hospital terrifies him and now he knows that he won't be admitted, he's not afraid to admit when he is in need of treatment, so he gets treated quicker and does not have such a severe relapse."

Community treatment did not result in more legal offences or police contacts, but it was not as effective as standard care in reducing the number of suicide threats and attempts. However, none of the community treatment patients, but two of the controls, died during the 12-month study period. A brief survey of all patients, 3½ years after presentation, revealed that seven control group patients were dead: three had definitely committed suicide, two had drowned (possible suicides), and two died from natural causes. No experimental group patients were dead at that time.

For two sub-groups of patients, the data were analysed separately. Firstly, for those given a diagnosis equivalent to schizophrenia by the Catego Programme of the PSE, the results mirrored the results of the total sample (Houlst & Reynolds, 1984). Secondly, those patients who had never had a psychiatric admission prior to entering the study: of 11 in the community treatment group, only four were admitted during the 12-month study period, none for longer than three days, and none were re-admitted during this time. In the control group, 11 out of 12 patients presenting for the first time were admitted and five (42%) had two or more admissions during the study year. Although these numbers are small, it could be hypothesised that the prevention of that first psychiatric admission may well prevent a patient starting on a career and identity as a mental hospital patient.

During the 12-months study period, the average treatment cost (public and private, direct and indirect) for each experimental group patient was A\$4,489 and for each control patient A\$5,669; i.e. standard hospital care and after-care cost 26% more than community treatment. For experimental group patients, 81% of the treatment costs were incurred in the community, mainly the cost of the Community Treatment Team, but only 7% of the treatment costs to control patients were incurred in the community. Cost of treatment in Macquarie Hospital accounted for 79% of all treatment costs incurred by control patients, and closely approximated the total costs incurred by the Community Treatment group (Cass & Lapsley, 1983).

Discussion

Our findings confirmed our aims: it is feasible to treat most psychiatric patients in the community as an alternative to mental hospital care. It does not transfer the burden to the community; it is preferred by patients and relatives, and it is possible in this way to get a better clinical outcome for less cost. This latter fact indicates that the effective ingredients lay in the mode of treatment rather than the amount of resources. While improvements in the quality of standard hospital care and after-care (e.g. more community staff, more support to relatives) may have resulted in improved outcome or more favourable attitudes, it would have required even more resources, and hence would increase the cost differential between the two modes of treatment.

The research finding that the amount of previous hospitalisation is the best predictor for future hospitalisation has been called a circular one; and that these are the most severe illnesses which actually do require the most hospital care. I think this explanation is only partly true; the finding from our study that amongst the sub-group of control patients with no previous hospital admissions there was a 42% re-admission rate within 12 months and no re-admissions from the experimental group gives credence to the idea that once a patient is known to have had a psychiatric admission he is much more likely to be re-admitted. Unfortunately the numbers in the sub-group are too small for definite conclusions to be drawn.

The Stein and Test study and our replication of it were the two studies which had the most comprehensive and continuous programmes of care in the community; they also were the ones with the fewest exclusions of patients, and included dangerous, suicidal and acutely psychotic patients. Quite a number of these could be managed safely in the community, but there were still some in both studies who could not. Clearly, hospital care is still needed for a few patients, though many fewer bed days are required than at present.

In his Annotation on "Alternatives to Psychiatric Hospitalisation", Tantum (1985) puts the view that one of the main lessons from this research "is not that hospital admission is no good but that conventional after-care could be improved," and that brief admission followed by intensive community support, such as was available to patients in the experimental projects, would be equally satisfactory. These statements are the common response of hospital-based workers to the challenge to what Tantum calls their "traditional hegemony". Currently, the hospital is the focal point of most psychiatric services; the highest status is accorded to it,

academics are attached to it, the bulk of the staff work there, and most of the funding is spent there. Domiciliary assessment is often merely a funnel to hospital which, in the minds of most professionals, is the place where any treatment of importance takes place. Even the word "aftercare" implies just that; care *after* the main treatment has taken place. Although research has pointed out the continuing high relapse rate, and has identified family and psycho-social factors and non-compliance with medication as being responsible for these relapses, services are still organised to treat the advanced relapse, rather than to prevent the relapse occurring or to treat it vigorously in its early stage. If conventional aftercare were to be improved on a nation-wide scale so that the problems of deinstitutionalisation could be rectified and good care was available for all, the financial cost would be very high. Governments in all countries have baulked at it. Extrapolating from our study, it would mean doubling the cost of services unless there was a corresponding decline in the use of the hospital. Whether brief hospital admission followed by intensive community support would produce an improved outcome without being prohibitively costly remains to be demonstrated. I personally doubt it; given human nature, I think the hospital service would soon build up again if it remained the central part of the service.

The studies on alternatives to psychiatric hospitalisation tell us, in essence, to change our focus from the patient in *our* hospital to the patient in *his* community. If our psychiatric services are organised to ensure that most of the staff and most of the funding are directed towards the patient in his natural environment, then help can be provided early, can be intensive and extensive, and can be capable of producing an outcome as good as, or better than what is currently available, and at about the same cost.

The problems of deinstitutionalisation are in large part due to our present imbalance in service organisation. Most patients in the community are unsupported most of the time; most staff are in the hospital treating episodes, many of which could have been prevented or aborted. The perils of both deinstitutionalisation and institutionalisation can best be addressed, as Tantum concludes, "by considering hospital care . . . as one of a range of carefully judged, social interventions which psychiatrists can deploy in assisting the patient to lead his life as little hampered by his illness as possible." This will mean re-focusing our service orientation and surrendering the "traditional hegemony" of the hospital.

From our experience and that of others, the following principles of care seem to be important ingredients: (1) the intensive nature of the help at the beginning; the 2-4 hours spent at the initial interview and the frequent visiting or even continuous period spent with the patient and his family during the first few days helped form a therapeutic bond between the patient, family, and staff. It also relieved the burden on the family, and in many cases allowed them to resume normal routine. (2) A willingness to involve actively both patient and relatives or care-givers in the management programme and to provide them—where appropriate—with information, support, guidance, and counselling. (3) Consistent care by the one team. (4) A personal case manager, who accepted responsibility for ensuring that the patient's needs were met and for co-ordinating with other agencies providing service. (5) A preparedness to go out to the patients and their relatives assertively, but without being unnecessarily intrusive. (6) Help with practical problems of living; this included the teaching of living skills at the place where the patient needed to use them, so that the problem of learning not generalising from the hospital to the community (Test & Stein, 1978) was avoided. (7) A service available 24 hours a day, easily accessible, mobile, and rapidly responsive, so that patients and relatives feel secure, and early intervention can prevent relapse. (8) An on-going and extensive service, rather than a time-limited service.

All staff on this project quickly came to prefer this way of working. It was more satisfying and meaningful than their previous work; two have since set up, and are still running, crisis teams in other parts of New South Wales. Staffing levels for a crisis team which is part of an on-going community service cannot be easily determined from our study, as methodological requirements for research projects create a degree of artificiality, e.g. while one psychiatrist can be on-call continually for the limited number of patients in the project, and for the limited time of the project, he cannot continue indefinitely like this. A mobile crisis service should be part of a network of community-based support services, and either the latter services pick up on-going care once the crisis has resolved, or the crisis service is manned by regular community mental health staff on a rostered basis. At least two staff should be available for the crisis service for two shifts seven days per week, and fixed rosters are preferred by staff so that they may organise their private lives. Psychiatric cover for the crisis service should come from the consultant and trainee psychiatrists covering the rest of the catchment area service.

In New South Wales, a Department of Health (1983b) inquiry into psychiatric services has recommended that, as a matter of policy for State Government services, the highest priority be given to the development of adequately staffed, community-based teams throughout the state to provide crisis care, treatment, and rehabilitation for the severely mentally ill. The criteria for these services are similar to those mentioned above.

The State Government has accepted and is now implementing the recommendations, transferring resources from the mental hospitals to the community for this purpose.

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Review Article

Witchcraft and Psychotherapy

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Belief in witchcraft, which serves a variety of social functions and personal defences, is bound to emerge in psychotherapy with individuals from a culture that holds such beliefs; endeavouring to understand it can open up new therapeutic possibilities. The nature of witchcraft, the profiles with which it intrudes into therapy, and the socio-psychological functions it fulfills are considered. Referring such patients to witchdoctors is morally unjustifiable, but the witch-doctor's folk-image provides a floating transference, around which the therapeutic relationship can be built. In dealing with witchcraft-ideation, understanding is based as much on cultural as on personal empathy, and to enhance its relevance, therapy may appropriate some of the functional dynamics of the witchcraft system into its own therapeutic manoeuvres.

Increasing emphasis on availability and accessibility of mental health services to all populations in the developed world has brought into focus the centrality of racio-cultural interests. The training of the Western therapist does not equip him adequately to empathise with the attitudes and beliefs of cultural minorities, and it is being increasingly felt that white therapists should have some practicum and internship experience in black communities (Butts, 1972; Vontress, 1974). This is tenable *mutatis mutandis* with regard to any ethnic minority anywhere, and in developing countries, many of the strategies and techniques of Western psychiatry, especially those of psychotherapy, do not seem to be relevant to their belief systems (Pande, 1968).

Witchcraft-ideation 'stands out' prominently among such beliefs. A Western-trained psychiatrist experiences much difficulty in dealing with it, and therefore tends to dismiss it as 'one of those primitive superstitions'. By doing so, he may block the opportunity to discover the dynamics that might be causing, shaping, elaborating, or repressing symptoms. In therapeutic work, we have often encountered witchcraft-ideation, and believe that if one tried to unravel its dynamic significance, new therapeutic possibilities might emerge.

The authors are a multi-national team, engaged in psychotherapy with the Bantu population. Three (NN, GK and JGH) are native Tanzanians; JSN is an Indian psychiatrist who has worked in Africa for nearly six years; BJ a French national who has been a priest cum psychologist for 20 years in Tanzania;

and GD a German clinical psychologist, working in Tanzania for over two years. We, therefore, bring together a variety of cultural perspectives—Western, Eastern, and African. By virtue of cultural contrast the non-Africans among us can more easily identify culturally significant phenomena in their Bantu patients, and offer hypotheses about them; their local counterparts provide the corrective feed-back from their indigenous vision.

What is Witchcraft?

Reviews of witchcraft are already available (e.g. Evans-Prichard, 1937; Robins, 1959; Parinder, 1963), so that we will only try to highlight those aspects which seem most relevant to our discussion. There are some differences of opinion about the definition: while most lexicographers (e.g. Little, 1956) do not distinguish it from 'sorcery', many anthropologists in fact do so. Evans-Prichard (1937) and, following him, Middleton & Winter (1963), Beattie (1964), Harwood (1970) and Mair (1974) among others, (especially those who have worked in Africa), make a distinction between the two terms. Witchcraft is defined as a 'mystical and innate power which is used by its possessor to harm people'; while sorcery is defined as 'evil magic against others' employing herbs, medicines, charms, etc. Yet there are other anthropologists, e.g. Raum (1967) who use the two terms interchangeably; in fact, these have been employed in a thoroughly chaotic manner, even within a single symposium

(Middleton & Winter, 1963). Many African tribes make a linguistic distinction between the two concepts: Kiswahili, for example, has the expression *ulozi* for witchcraft and *uchawi* for sorcery, while the same distinction is made by the Akan, Ashanti, Azande, etc. However, we propose to subsume both these terms under the rubric 'witchcraft', because both represent culturally appropriate responses, which function as alternatives in the same kind of situation (Farke, 1962), co-exist frequently in the same society (Harwood, 1970), and provide habitual explanations for misfortune (Lewis, 1976).

Witchcraft is a theory of causation: it does not deny natural or empirical causes, but seeks supernatural ones behind them. Two questions can be asked in the context of every misfortune: 'how' did it happen?, and 'why' did it occur at all?. The 'how' is answered by empirical observation; the 'why', *inter alia*, by witchcraft. Even if our scientific understanding of the 'how' increases, it will still not be able to dispose of the 'why' of a misfortune. Hence, the two beliefs may easily co-exist.

Figure 1 illustrates the place of witchcraft in the system of causality. Witchcraft shares this hierarchical position with sorcery, cursing and the evil eye. A witch-dominated world is essentially a closed world, where persons and human relationship and more important than material good, where everybody is somebody, and where moral identity prevails and group values receive emphasis. Two kinds of person threaten this closed world—the deviant and the alien—who are the sources of evil, since they may well be witches.

A witch is believed to carry out her art in secret, especially at night. The practice is socially proscribed in almost all African tribes, and anyone discovered practising the black art, is punished, ostracised, or even killed.

As a system, witchcraft can absorb empirical causality, and can also absorb and explain many of its own failures by virtue of what Evans-Prichard (1937) called 'secondary elaboration on belief'—successes will be remembered and failures explained away. An analogous situation can be discerned in psychoanalysis—if you accept an 'interpretation', well and good; if you don't, 'denial' is presumed to be at work. "Direct assault on a closed system of ideas is not easy, since the system absorbs the attacks and converts them to strengthen itself" (Gluckman, 1966).

Witches and anti-witches

The African witch is quite distinct from the European version in having no truck with the Devil

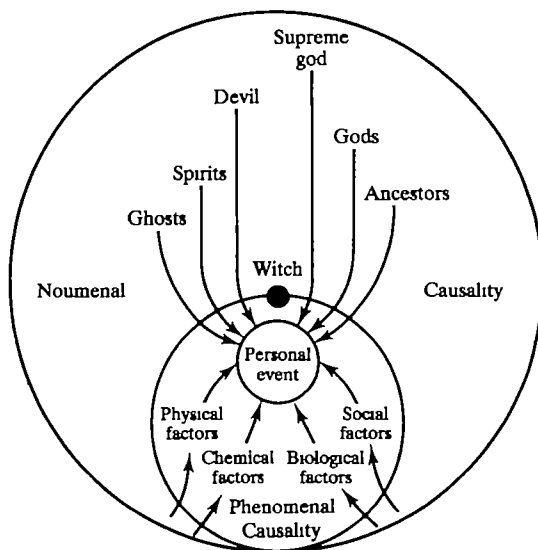


FIG. 1 The place of witchcraft in the system of causality. The witch sits on the borderlands between the two, and is human in form (thus subject to intutional causality) but imbued with supernatural power (and hence wields noumenal causality).

and not holding Sabbath assemblies. In certain tribes, however, there is something like a Sabbath assembly, where the 'witch' is believed to ride snakes and hyenas. In fact, an African witch may not even be aware that he/she is one, until beginning to bring misfortune to those wished ill. It is the use of witchcraft, and not its mere possession which is despised by African tribes.

Three types of witches have often been distinguished: (i) *Stereotyped* or 'night' witch, with a cosmic dimension of evil, flouting all the rules governing property, sex, law, and decency. (ii) *Presumed* known about and feared by everyone, but somehow left alone. These include unpopular, asocial, arrogant people, as well as those with a staring gaze, with abnormal and ugly features, of mixed descent, and very advanced age (who have lived so long 'by virtue of witchcraft'). (iii) *Accused* indicted to have caused a misfortune; generally an avowed enemy or rival of the accuser.

In fact, these are not three different types of witches, but three stages of the process of witch-naming. Since witchcraft is not necessarily a voluntary act, and can even be exercised in sleep, without the person knowing it, no account is taken of the accused's denial of guilt or of any defence. Advantage always lies with the accuser.

The antiwitches or witchdoctors are benevolent witches, whose powers are superior to those of the malevolent, whom they can contain and control; through the power of oracle or divination, they can identify the malevolent witch responsible for a given act. They can also exorcise an evil spirit through their mystical power, ritual, or medication. They may be able to forewarn through foretelling oracles, and even intercede between the mortals and the spirit world; they have a medley of procedures at hand—rituals, spells, charms, potions, and even jugglery.

There is no clear distinction between a witch and an antiwitch, except intentionality. However, the witch-doctor is generally an intelligent person, competent in ritual knowledge, and convinced of the power of witchcraft and of his own powers. As he can bring harm to a man who is doing a moral wrong, he is held in great awe. In most of Africa, witchcraft is believed in not only by the uneducated and semi-educated, but also by well educated people—even doctors and scientists.

Emergence into Therapy

With the African, witchcraft ideation is a pervasive background belief which turns into a cognitive reality in times of stress. It may well be present at the very outset of therapy, having been generated prior to it, or may be unleashed by the therapeutic process itself. It may, at times, even be occasioned by the therapist who, on account of his appearance and demeanour, gets identified with the witch-doctor, or it may be part of the symptomatology of mental illness itself.

It generally emerges in one of the following ways:

(i) *As an explanation for all kinds of misfortunes* e.g. frustrations of life, failure in love, lack of success in business or examinations, or losses or deprivations of any kind. It may be spontaneously verbalised or brought out by encouragement. Sexual difficulties such as impotence are especially prone to be explained as caused by witchcraft. Prince (1966) reported that Yoruba males often dream of an assault against their genitals; alternatively, a witch is believed to have used an individual's penis in order to consort with his wife or with another woman, and then returned it in an altered and functionless form. Women consider witchcraft to be the prime cause for drying up of their breasts, for frigidity toward their husbands, and for sterility.

Witchcraft may also be a preferred explanation for bodily sensations such as the paraesthesiae of peripheral neuropathy and the phantom limb phenomenon after an amputation. It may also be the presumed

cause for physical or psychic illness, ruptured social relations, physical disaster, and natural catastrophe. (ii) *As a cause of the presenting symptomatology itself.* Actual or presumed bewitchment often generates intense anxiety and apprehensive foreboding. The physical and psychic symptoms that it produces only tend to confirm the suspicion and power of witchcraft, and further strengthen apprehension: a vicious circle sets in, and apprehension spirals up. Bewitchment anxiety, if unrelieved by traditional countermeasures, or if such measures are unavailable, may develop into "malignant anxiety" (Lambo, 1962), which may even lead to death, in a way that can be likened to 'voodoo death' (Canon, 1957). Occasionally, there is 'boomerang witchcraft': that practised on someone else having failed, the attempter begins to believe that the other person has superior powers, and might play counter-witchcraft. Patients with anxiety generated by suspected or boomerang witchcraft often look pale, sickly, and terrified, and are often perplexed or even confused. They verbalise their terror characteristically as 'a ghost has hit my heart'; 'a dragon is choking my breath', 'my life is being sapped away', etc. The onset is often traced back to some 'unusual' event, e.g. the skin of a snake lying outside one's door, or seeing a dead body in a dream.

(iii) *A symptom of illness.* The content of delusions is known to be subject to pathoplastic cultural influences and amongst people who believe in witchcraft, delusions of witchery are commonplace. The mentally ill, as much as the healthy express themselves in the idiom of their culture. A paranoid patient in the West may talk of people harming him through radio-waves, microwaves, or laser beams, while one from a witchcraft-believing culture will complain in witchcraft terms. In the latter, the complaint of witchcraft as such may not be clinically significant, but the embedded complaint of being persecuted is (Leighton, 1982). Basher, a Sudanese psychiatrist, said that to diagnose delusional witchcraft ideation, you have to "look for the knight's move" (Savage *et al*, 1965); in other words, there is some oddity which other members of the patient's cultural group sense in the patient, and do not consider to be identical with familiar witchcraft ideas. An astute clinician is generally able to distinguish between culturally prevailing non-delusional fixed beliefs and the delusional symptomatology of a psychosis, expressed in witchcraft terms. The delusional ideation of witchcraft is often mistaken as superstition, but a distinction has to be made between simply 'superstitious' and 'basically delusional' beliefs. Table I presents the distinguishing features of the two.

TABLE I
Differences between delusional and non-delusional witchcraft ideation

Non-delusional Witchcraft Ideation	Delusional Witchcraft Ideation
1. Little accompanying symptomatology – at most, symptoms of anxiety and apprehension, which seldom assume psychotic dimensions.	Symptoms of a psychotic disorder almost always accompany. These often include hallucinations and loss of contact with reality.
2. Symptoms generally subside after a diagnostic divination and prescribed corrective measures.	Symptoms remain unabated, even after a diagnostic divination and observance of prescribed corrective measures.
3. Accompanying behaviour defensive and guarded; often considerable reticence in expression witchcraft ideas.	Accompanying behaviour may openly be vindictive and hostile; e.g. acting on hostile impulse and assaulting the person who is suspected to have effected witchcraft.
4. Considerable acceptance of the patient's ideation by his social milieu.	Only a partial and often sceptical acceptance of his ideas by the patients' family and others around him.
5. Ideas seldom, if ever, get generalised and systematised, but may become vague and dissipated.	The ideas often tend to get generalised as well as systematised.
6. Ideas of witchcraft seldom appear suddenly. 'Evidence' accumulates over a period of time before crystallising into definitive witchcraft ideation.	Witchcraft ideas may suddenly descend 'like a bolt from the blue', thereafter showing secondary elaboration.
7. Ego-syntonicity of the ideas comparatively less rigid.	Ego-syntonicity is firm and rigid, and unamenable to influence.
8. Patient usually shows considerable concern about having been bewitched.	Patient may show a weird unconcern with his witchcraft-delusions, much like 'double orientation'.
9. Social withdrawal is circumscribed, i.e. only from the suspected person.	May be much more generalised social withdrawal than from the suspected person alone.

(iv) *As feigned or malingered.* The patient is consciously aware that no witchcraft has actually been practised, but feigns it, and imputes it with clandestine intention to some specific person; i.e. there is no real victim of witchcraft, but only a victim of imputed witchery. Such behaviour is indulged in generally for the purpose of 'distancing' from a person whom the patient finds emotionally offensive, but socially inescapable.

Sociodynamics

(a) *Instrument of moral suasion*

Witchcraft belief is a potent instrument of moral influences and checks members of the community from transgressing its moral codes. The fear that a transgression, even an inadvertent one, can lead to

punishment through witchcraft keeps people from infringing taboos and moral sanctions.

It is not unlikely that 'guilt', 'shame', and 'fear of witchcraft' are prototypes of one another, in different cultural settings. In cultures with 'internalised' moral controls, *guilt* feelings are unleashed by moral transgression. In other cultures, where social controls are, by and large, external, *shame* is generated. In yet others, where such controls are supposedly exercised by 'supernatural' forces, *fear of witchcraft* and allied phenomena (hex, sorcery, etc.) tends to appear.

(b) *Control of aggression and ambition*

It is believed that one who attains any 'glaring success' comes 'into the people's eyes'; others

become jealous of him, and may try to undermine his success and achievement. Therefore, people must try to keep their ambitions under check, and not outbid their neighbours, or it will evoke jealousy, which, in turn, evokes witchcraft—'jealously comes first, witchcraft later'. A person who is exceedingly successful in life is suspected of possessing witch-power himself, while other aggressive tendencies are also believed to evoke counter-measures in the form of witchcraft.

The witch as a scapegoat also becomes an outlet for repressed aggressiveness of the group. In general, Bantu society represses aggression, permitting this, at best, to be symbolically expressed in joking relationships, and dances. However, it can occasionally gain a real hold upon a socially acceptable target: a thief, if caught, is often beaten to death on the spot, and so is a person accused of witchcraft.

(c) Solving relationship difficulties

Interpersonal relationships, especially within the extended family, are fraught with tensions of all kinds, which may exist between relatives who cannot be informally distanced. Imputation of witchcraft brings about a formal distancing, where this had not been possible—generally within the family—but it may also be employed for distancing real or suspected 'enemies'.

(d) Guide for action in disaster

In times of disaster, an oracle or divination is resorted to, which not merely serves to diagnose the presence (or otherwise) of witchcraft as the cause of disaster, but may also prescribe counter-measures to be taken. It thus provides guidance for action and thereby mitigates suffering.

Psychodynamics

(a) Ego-defence mechanism

Witchcraft is a socially accepted explanation, not simply for personal misfortune, but also for failures and weaknesses: a student failing an examination, a businessman incurring losses from indiscreet investment, a drunk driver causing an accident, a hunter unable to shoot any game may all impute their 'bad-luck' to witchcraft. Goldschmidt (1977) gives an instance of a Sebei young girl who 'cried the knife' when she was being circumcised for her initiation, and refused to let the operation be completed until she was held down forcibly; he had expected that she

would have remorse over her behaviour and go into 'deep depression and self-hatred'. However, nothing of that sort happened, for the culture had provided her with an institutionalised ego defence: she knew who had made her 'cry the knife'—it was a man whose advances she had rebuffed, and who had performed witchcraft. Witchcraft ideation, as a coping mechanism, enables one to shelve responsibility for personal failings, and is a reason why feelings of guilt are so rare among believers in witchcraft.

It may also involve the dynamic of scapegoating. If witchcraft imputations are readily accepted by one's social milieu as a semi-serious alibi for personal failings, then witchcraft acts as a buffer against social sanction. However, if the imputation is taken seriously, and leads to a witch-hunt, it can end in catastrophic scapegoating.

(b) Institutionalised distrust and jealousy

Behind the ideation of witchcraft often lie distrust and jealousy, both of which appear to prevail in a veiled way throughout the social substratum, but during times of stress, these become unveiled in the form of bewitchment imputations. Alternatively, an individual presumes himself to be the victim of witchcraft because he disturbed the social harmony, by getting out of step with the others through high ambition or extraordinary success. In either case, witchcraft ideation serves to repress jealousy and to preserve the adjustment of individuals to their social milieu. In another way, it actually institutionalises distrust. Goldschmidt (1977) found validity for this conclusion among the Kamba of Kenya, who are highly *gynophobic* men, hating and distrusting their wives with institutionalised fervour. Since it is considered bad for the man to give sexual satisfaction to his wife, the result, deriving no doubt from 'affect hunger' on both sides of the bed, is a great deal of clandestine extramarital sexual activity. The men who do not give their wives satisfaction seek and satisfy other men's wives, yet beat their own wives for being involved with other men. No wonder, they are all afraid of witchcraft from their own wives.

Much of the imputation of witchcraft is focussed on members of one's own family or immediate neighbourhood: co-wives out of envy, neighbours out of malice, and spouses out of spite, are easy targets. In fact, it is always 'the enemy within the gates', hiding behind the mask of kinship and friendship, that becomes the easiest target; it is the person with whom the ego's relationship is incorporative and not just transactional (Harwood, 1970).

(c) Externalised super-ego

Belief in witchcraft makes for externalisation of super-ego: children are sensitised to react to others. There is a double-pronged sensitisation—to those whom you trust and to those whom you do not: the former will put you to shame if you are found transgressing the social norms, while the latter will make you the victim of witchcraft. Whether it is shame about ridicule or fear of witchcraft, both these modes of functioning of the super-ego are external; there is no internal referent—no feelings of guilt or sin. Many African languages have no word for 'sin' or 'guilt'—though they have for 'right' and 'wrong', 'good' and 'bad'. "This is not due to poverty of languages, but to the fact that the concepts expressed by the European words—'sin' and 'guilt'—do not exist in African thinking" (Castle, 1967).

With the African, "An act is not sinful until it is discovered by another person; there are no secret sins in African concepts of sin. It is not sinful for a girl to sleep with a man so long as they are not discovered by someone else... sometimes, however, 'sin' will manifest itself through sickness, psychical disturbance or other form of misfortune and people begin to trace the effect to its 'cause'" (Castle, 1967). This 'cause' may be a breach of customary behaviour, and if the interests of a third party have been injured in the process, fear of witchcraft will ensue.

(d) Relieving fear of the unknown

Misfortunes, if unexplained, cause tremendous anxiety because it is not possible then to take any remedial or preventive measures. However, through oracles and divination, one can know the cause: it may be witchcraft, an evil spirit, or a displeased ancestor, once the cause has become known, suitable measures can be taken.

(e) Denial

If one has really indulged in witchcraft, one is bound to deny it, out of fear of the consequences, but even in the case of a false imputation, one may be approached by the 'victim' to remove the spell. One may not deny having indulged in the practice then, for fear that one would not be believed, but might acquiesce in doing what is requested, perhaps, entering into a blood-contract with the 'victim' that he will keep the whole affair a secret. Thus, the imputed person, may be led into believing that he is really in possession of the power of witchcraft.

Management in Psychotherapy

Witchcraft ideation is so 'other-worldly' to Western psychotherapy that little room can be found for it in the latter's conceptual framework and techniques. Unfamiliarity with the nature of witchcraft concepts, their dynamic significance, and their social function, coupled with his own inability to manage them may force a Western therapist to retreat and perhaps refer the patient to a witch-doctor (Jilek & Todd, 1974). However, this is not always in the patient's best interests: the anti-witchcraft ceremonial remedies prescribed by the witch-doctor are often exactly expensive, and in any case, referral is undesirable on moral grounds. Witch-doctors generally identify and 'name' a witch; this stigmatising fosters "labelling, mistrust, prejudice, ostracism and killing of persons who are guiltless of the crimes attributed to them" (Leighton, 1982). Also, there may well be a psychotic or neurotic condition behind the facade of witchcraft-ideation, which is outside the competence of the witchdoctor. The therapist-psychiatrist, therefore, should not shirk his responsibility, but rather enlarge his therapeutic competence to deal with witchcraft ideas, should these emerge in therapy.

Most Western therapies are essentially hide-bound within Western culture, and conditioned to specific theoretical frameworks. "More than a phenomenon, it (Western psychotherapy) is a cultural institution of this (Western) society, deriving its roots from it and, in turn, affecting and transforming the soil and era in which it thrives" (Pande, 1968). It is therefore important for a Western therapist working in non-Western cultures to be aware of the ethnocentricity of the techniques he has learnt. What is required is a pliable, strategic therapy, able to function in diverse cultural moulds. The basic requirement is a therapist who should have the ability to work with diverse world-views, keeping his own under control, and not letting it intrude unnecessarily into the patient's.

This can be called *strategic therapy*: it has often to be carried out expeditiously, but at the same time cautiously, to be launched in the hostile territory of mistrust; and must focus on the here and now of the patient's problems, with neither the time nor the inclination to resurrect presumed causation in the dead past. It has to adopt realistic goals and avoid quixotic confrontation with the culture it faces; it chooses to be 'directional' or 'non-directional', as therapeutic exigency demands. It recruits allies, e.g. the patient's family, to further its objectives. The keynote of its theoretical orientation is relevance, with responsiveness to the patient's needs and

problems—the immediate ones first. Its operational mode is flexibility—maintaining an orientation compatible with the patient's and employing techniques with which he/she is not altogether unfamiliar.

Management of mistrust

Discussing the dynamics of therapeutic relationship in its cultural perspective (Neki *et al.*, 1985a), we have observed that a patient tends to clutch at the most relevant of the 'floating transferences' in his social milieu. Thus, an African patient who believes in witchcraft tends to identify the therapist with the witchdoctor, to whom he seems to have an ambivalent attitude. "There is always a certain ambiguity regarding the status of the witch-finder, which is evident in the ambivalence with which Africans perceive them... Witchfinder and the witch share the same shadow world" (Sow, 1980) Paulme (1954) reports that the Kissi of the Guinea forest, where the witchfinder is identified with the witch, ask "how... would the witch-finder recognize witches and how would he combat them if he himself were not initiated into their secrets, if he himself were not a Kuino, a killer and eater of human flesh?" Thus, the African fears the witch-doctor as much he trusts him, and this ambivalence is easily transferred on to the therapist, who is also 'powerful and mysterious'. To this, is added the patient's distrust of hospitals in general and his lack of motivation to see a therapist. The therapist's foremost task, then, is to conquer the patient's fears and strengthen his trust. This has to be achieved rapidly, but any lack of tact undermines trust and reinforces suspicion; the therapist has to be cautious, yet natural. The patient's transference may swing to the extremes of both positive and negative, which needs deft handling.

In the West, it is customary for therapist and patient to sit in close proximity with one another—side by side, or face to face—with nothing intervening between them. However, most African patients would panic if the therapist sat next to them; it is important to let the patient sit opposite the therapist, across a table; even then, he will often draw his chair further back, to increase the distance. Careful spatial moves by the therapist, empathically monitored to patient-readiness, can bridge this distance and eventually bring about the desired proximity.

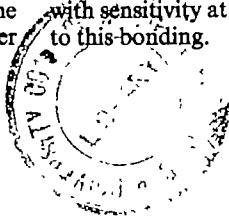
Notwithstanding the views of Sommer (1969), who considers this to be characteristic of competitive confrontation, we prefer an initial across-the-table meeting with the patient. Gardin *et al.* (1973) maintained that this arrangement increases the chances of eye-contact, and signals liking, rather

than competitive confrontation; the table provides a buffer of security to the suspicious, hostile, mistrusting or even ambivalent patient. Since there are great individual differences in patients' responses, each response should be mentally evaluated on a hypothetical scale of 'immediacy behaviour', i.e. interpersonal distance, amount of eye-contact, forward lean, and direction of shoulder orientation (Mehrabian, 1972). Exline & Fehr (1978) have experimentally confirmed that subjects reciprocate the immediate behaviour of interviewers, and further spatial dynamics must rest on mutual reciprocity.

One set of such moves are described here, as an example: although the therapist sat face-to-face with the patient, the latter, who was suspicious and distrustful, pulled his chair back and moved it to one side, to avoid direct confrontation. The therapist waited for a while before making his next move, trying to allay the patient's fears. After a short while, the patient was less ill at ease; he drew his chair closer to the table. Watching this, the therapist moved his chair sideways, so as to be face-to-face with the patient once again. As the interview continued, the patient began to lean forward on the table; then, the therapist, and following him, the patient, began to put their hands on the table. A little later, the therapist shifted his chair to bring it by the side of the table, sitting diagonally rather than in front of the patient; he thus created greater proximity with the patient, but left the corner of the table jutting in between the two of them so as not to reactivate the patient's much allayed suspicion and mistrust. As the interview further warmed up, and the therapist sensed further closeness, he chose an appropriate occasion to let reciprocal (and culturally acceptable) body contact be established, by stroking his hand over that of the patient.

In these dynamics, each step was taken after an emphatic assurance that trust had been sufficiently strengthened and fear sufficiently assuaged to make the move acceptable. The developing relationship can be injured if the steps taken are too hasty, but if, on the other hand, they are too slow, the patient—still ambivalent—might close his mind to any further therapeutic process. These moves were also performed in a natural and contextually appropriate way.

Such a step-by-step, carefully monitored spatial strategy in some way parallels Tinbergen & Tinbergen's ethological approach with autistic children (1972). In developing spatial (or 'proxemic') dynamics, interactive patterns that create effective affiliative bonding with the patient must be observed, with sensitivity at the same time to any stress caused to this bonding.



Should a patient enter therapy with negative attitudes and inappropriate expectations, the burden of responsibility to reverse these handicaps essentially lies with the therapist. The patient should be enabled to see him as an ally in this battle, which according to the patient's world-view, is against 'witchcraft' but according to the therapist's is against an 'illness'. The adversary is essentially identical, but is conceptualised in different vocabularies.

(i) Avoiding quixotic confrontation with the system

Since witchcraft ideation is a closed system, it is advisable to avoid confrontation with it: we may not accept it ourselves; but must neither decry it, nor try prematurely to educate the patient out of it. This can be frustrating, because witchcraft is a belief deeply and firmly rooted in the patient's entire cultural environment. Any attempt to dislodge it only leaves the patient confused, helpless, and more anxious. Therefore, ideological confrontation must be avoided, and though patients occasionally ask "Doctor, do you believe in witchcraft?", it is best to return a cautious answer, such as, "I am aware of this belief, held by thousands of people, but I also know that many people have suffered a great deal on account of the witchcraft they believe has been done to them. We have helped quite a number of such people". Non-confrontation means going along with the patient to the extent of 'understanding' his beliefs, but no more than that. Thus, the system can be utilised strategically, without entering it and without reinforcing it. Emphatic understanding is the best guide to avoid treading on the patient's sensitivities.

At the same time, it is important to explore the genesis and elaboration of the patient's witchcraft ideation, and to enquire what treatment he has already received or been recommended, and with what results. One should also enquire about his aetiological beliefs, and perhaps indicate one's own, though without entering into an argument. If an underlying psychotic or neurotic illness is suspected, an attempt should be made to disentangle it from the elaboration of witchcraft ideation.

The therapist may have to use active therapeutic techniques—directive, confrontative, and persuasive—for certain problems—but his approach should always be functional, never ignoring the most pressing issues of the moment. During the early phases, some medication may have to be prescribed, even if only a placebo, as well as such active intervention as assurance, advice, and environmental manipulation. The content of therapy has to be linked closely to the patient's current experience.

These strategies have, in part, also been proposed by Harper & Stone (1974), Grier & Cobbs (1968), and by Gordon (1968) in therapy with black patients in the United States.

Setting Realistic Goals

The most realistic and least controversial goal of psychotherapy, is **relief of symptoms**; this has to be achieved expeditiously, even in the first session as much as possible. It serves to strengthen the patient's trust in the therapist's 'power', and to assure him that the power of witchcraft has been stalled. Symptoms most amenable to relief need to be picked up first, and the patient given a firm assurance of relief—"I am going to take away your pain or sleeplessness, or . . . (whatever it is)". If the pain has a physical cause, an analgesic should be given; if it is due to tension, relaxation exercises. For insomnia, a (placebo) pill can be given sealed in an envelope, and the patient asked to keep it under his pillow; should he not fall asleep within half an hour, he may swallow it. Relatively few patients really need an hypnotic, which may be prescribed for a night or two to break the vicious circle. Judicious use of neuroleptics for psychosis, anti-depressants for depression, anxiolytics for acute anxiety states, and vitamins, minerals or other nutrients for deficiency disorders not only relieve major symptoms, but also serve in this way to mollify the fear of witchcraft.

Commonly associated with fear of witchcraft are conversion symptoms: aphonia, astasia-abasia, paralyses, weakness, anaesthesia, and hyperventilation are commonly seen. One of us has employed modules of single-session therapy for a medley of such symptoms (Neki, 1985b); the dramatic relief experienced appears to the patient to be nothing short of 'magical', and this convinces him of the therapist's power against presumed 'witchcraft'.

Thus, the early phases of therapy are marked by active and energetic intervention, which may consist of direct assurance, advice, or persuasion, active or placebo medication, environmental manipulation, or crisis intervention. Crisis therapy, being brief, limited in breadth and depth of exploration, and focussing on the here and now, is readily accepted. However, if the therapist seeks to alter the patient's outlook, his socio-cultural reality has to be kept in mind. "Any changes sought contrary to the dictates of belief systems surrounding him are bound to prove self-defeating" (Thomas & Sillen, 1972). Such resistance to change may, in fact, be a sign of health, and since African patients generally resist reducing their illness to a psychological level (Morakinyo,

1985), providing insight as a goal is unpragmatic and elusive. Prince (1962) found that insight therapy was not possible with Yoruba patients.

Since the patient feels so 'powerless' and bereft of strength, due to witchcraft, a second goal of psychotherapy with such patients should be helping them to gain a sense of power. To achieve this, the patient must be helped to learn to feel needed, loved, and wanted, as well as useful to himself. This can be done by assigning him carefully graduated useful tasks, which can be easily carried out; doing them engenders feelings of satisfaction, and relieves the patient of his negative self-image, as well as his non-commitment.

It is advisable to focus on pragmatic life strategies, and persuade the patient to try them out. Patients soon realise then that they have more control of their destiny; they have generally been unaware of the many options of action open to them, but making them aware of these, and helping them to exercise a suitable choice enables self-confidence to be regained.

Appropriating the functional dynamics of the witchcraft system

Whilst witchcraft should be neither confronted nor strengthened the functional dynamics of this system can be appropriated into psychotherapy. The witch-doctor 'understands'; he has no need to enquire, but informs. So our enquiries must be kept to the minimum, at least in the beginning, but through empathic understanding, the therapist tries to know what is happening to the patient, and to make him feel understood. What the patient may have only haltingly and inadequately expressed may be reformulated in clearer terms: 'mirroring' back to him his own thoughts, formulated more clearly, makes him feel understood. Thus, the therapist tries to make the patient feel understood, and clinical experience enables him to guess more than he has been told; communicating what one has thus guessed back to the patient is also reassuring.

Relevance, which is always the key note, has to be also in the patient's eyes, and not just in those of the therapist. Hence, detailed enquiry, especially about the patient's past, which is not seen by him to be immediately pertinent, is avoided. Later on in therapy, it is generally possible to delve into the requisite details of patients' lives.

The language employed by the therapist should be syntactical to that of the patient; this term is used in a broader sense than usual, and includes concepts, myths, ideas, etc. In other words, the 'idiom' of the therapist should be congruent with that of the patient. For instance, a patient said, "Doctor, I

know a baby doctor, a bone doctor, but I never know psychia—what do you call it?. Yeah, psychiatrist. What kind of doctor are you?". The psychiatrist, who knew that the patient's culture is characterised by physical expression and human relationship rather than abstract thinking and intellectual discussion, answered, "I am a heart doctor. I help people with worry in the heart". The patient was then sure that she was seeing the 'right kind of doctor' for, as she explained, "my heart has lots of worry" (Tseng & McDermot, 1981). A useful form of explanation is—"I am a doctor who helps patients get rid of their . . . (here mention is made of the main symptom(s) of the patient), whether or not caused by witchcraft". All along, it is important to be open and direct, and to provide unequivocal feedback to the patient about what the therapist understands of his problems, and the instructions he wants to give him.

Since a witch-doctor generally prescribes ritualistic remedies, the patient in therapy also expects to be involved in a ritual of some kind. Most cultures have their own remedial rituals—of handling grief, separation from the dead, restoring harmony with family or group, or combating fears—natural and supernatural. The patient may be encouraged to make use of these as a parallel to psychotherapy, without supplanting it, and some ritualistic activities may be incorporated into therapy. One of us (BJ) employs relaxation therapy for this purpose, while role-playing and modelling may also be utilised; these not only employ a physical, action-orientated style, but also stimulate introspection. Patients may even be given 'home-tasks'; one of us (JSN) asks his patients to record or remember their dreams, and to bring them to subsequent therapeutic sessions. Apart from the 'window' they provide into the patient's dynamics, dreams also have an esoteric meaning, so that the therapist who makes use of them makes the patient feel doubly assured of his prowess. The content of dreams can be used to lift a patient's morale; one had felt that he had been bewitched since he dreamt of having eaten at the hands of a stranger. Later, he had another dream, in which he found his house being cleaned and white-washed; this was interpreted to counter the significance of the first.

Recruiting allies

The patient's family can be great allies in therapy. Whether they have to be involved directly (family therapy), in the patient's 'home-work', to supervise his compliance, or just be kept in touch regarding progress is a decision which has to be taken in each

individual case. Family therapy, for example, was dictated by the needs of the following case:

A 25 year-old girl, suffering from pulmonary tuberculosis, came to the hospital, and the physician had her chest X-rayed; when she went to collect her report, the film could not be found. The same happened with repeat X-rays, taken twice more. She then gave up the chase, saying, "I know these X-rays are not going to be found. I have been bewitched, that is why!". On further enquiry, she averred having married a man against the wishes of her family; she had in fact become pregnant by him, even before marriage, and her grandmother had never been reconciled to this. The patient now felt that it was the grandmother who had bewitched her. Family therapy, involving the grandmother herself, led to the resolution of the conflicts and tensions.

In rallying allies for the patient, one has to scan his social network, and identify who can be the best source of support—father, mother, a particular sibling, employer, friend, priest, or traditional healer. Home visits provide a good avenue for studying the family dynamics.

For the sake of his patient, the therapist may have to plead with housing authorities, welfare agencies, employers, schools or courts of law. The witch-doctor has his extensions into the community, and if the therapist also has such contacts, his effectiveness becomes greater.

Studying the dynamics

The therapist studies the patients dynamics not with the objective of providing interpretations or insight, but to inform himself so as to be able to help the patient more effectively. To start with, the therapist must evaluate the patient's attitudinal dynamics: is he of a mistrusting or a dependent kind? Is he approaching therapy with interest, doubt, or

apathy? The findings will determine what strategy the therapist must adopt.

It is also important to find out what sort of feelings lurk behind the expressed fear of witchcraft. Is it aggression, fear of punishment due to moral transgression, jealousy or fear of presumed jealousy, the desire to escape responsibility for personal failure, or the irksomeness of some relationship? Different feelings warrant different management strategies. There is a need to identify defensive denial of inner feelings of weakness, fear of being discovered, reliance on maintaining a false image, or malingering. All these require handling in a way that does not cause loss of face to the patient.

Reality factors often intrude into psychotherapy: conflict, jealousy, frustration, suspiciousness, or confusion may easily arise if the patient's aspirations collide with reality, and all can rekindle witchcraft ideation. The patient must be helped to appreciate alternative possibilities, and to fortify his adaptive skills; he may also have to be helped to 'accept' the world, in existential terms.

The therapist is often faced with the dilemma of whether to lay emphasis on autonomy and self-actualisation, or on group solidarity and conformity. It is essential then to find out the sheet-anchor of the patient's personal values and to provide him with the means of making restitution toward that system of values. Empathy must not be with the patient alone, but also with his culture, and cultural empathy cannot be gained merely from academic information about any group. This has to be done by sharing actual life-experiences with people—visiting their homes, joining them in their ceremonies, breaking bread with them, and observing them in both formal and informal social occasions. It is only through such cultural empathy that proper understanding of culturally rooted problems can arise.

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Behaviour Problems in Retarded Children with Special Reference to Down's Syndrome

ANN GATH and DIANNE GUMLEY

Data were collected, using interviews with parents and teachers and rating scales, concerning the behaviour of two groups of mentally retarded children: 193 children with Down's Syndrome (DS) and 154 children with a similar degree of handicap were identified in the same schools. The proportions of children who had high scores on the Rutter or an additional behaviour check list, were similar in the two groups. Deviant behaviour was markedly more common in both sets of retarded children than in their siblings next in age; 31% of the children with DS and 29% of controls were judged to be well adjusted, while 38% of the DS children and 49% of the controls had significant behaviour disorder. Conduct disorders were most common in the children with DS. 'Psychosis' was the most common diagnostic label in the control group, but was also found in the children with DS.

Behaviour problems are a common and distressing complication of mental retardation: indeed it has been argued (Webster, 1970) that psychiatric disorder is an almost inevitable accompaniment: he could find no child who was 'simply' retarded in a group of Boston children aged 3-6 years. Webster described the "primary psychopathology" of retardation, the characteristics of which were "repetitiveness, inflexibility and simplicity of emotional life". However, Chess & Hassibi (1970) in their study of 52 children living at home, aged between five and twelve years and with mental ages of between four and six years found that 40% showed behaviour that could be accounted for by the fact of retardation alone, without psychiatric disorder. The features described by Webster as signs of 'primary psychopathology' were considered by Chess (1977) to indicate merely lack of ideas, which is an integral part of mental retardation.

A very high rate of psychiatric disorder in retarded children was noted by Philips & Williams (1975) who found only 13% free of it among clinic attenders, but there was likely to be a referral bias in this group towards children with problems. In another clinic for retarded children, 24% were considered to have a primary emotional disorder and 30% emotional problems secondary to other developmental difficulties (Szymanski, 1980). Kamimer *et al* (1984) found a 75% rate of behavioural or emotional problems among 167 children evaluated at a clinic: their criteria for 'no problems' were that the child was compliant to adult requests, interacted with appropriate affect, had no deviant behaviours

noted or reported, and showed a level of attentiveness and activity appropriate for his mental age level, and that the parent did not report management problems. These are stiff criteria, and the findings were not validated by comparison with a control group of children in the normal range of intelligence, judged by the same yardsticks.

The evidence from epidemiological studies indicates that psychiatric disorder becomes more common with decreasing levels of intellectual functioning (Rutter, 1971; Corbett, 1977). Rutter *et al* (1970) had defined psychiatric disorder as a disturbance of behaviour or emotions sufficiently severe and prolonged to cause suffering to the child, to the family, or to the community. By this definition, the incidence of behaviour disorders in severely retarded children in the Isle of Wight study was 50%, compared with 6.6% among in the normal range of intelligence. This figure for retarded children is similar to those found in retarded children in Scotland (Birch *et al*, 1970; Koller *et al*, 1983) and in Camberwell (Corbett, 1977), but somewhat lower figures were reported by Stein & Susser (1975) in severely retarded children aged 5 to 14, 20% of whom had behaviour problems, and by Groden *et al* (1982), who found 25% out of 1114 retarded children in Rhode Island had behavioural and/or emotional problems.

Some investigators have attempted to determine what sort of disorders are seen in retarded children: 38 of the 100 children described by Philips & Williams (1975) were classified as psychotic, while, of the rest, 26 had behaviour disorders, five had

neuroses, two had transient situational disorders and 16 personality disorders. The making of specific psychiatric diagnoses is particularly difficult in retarded children, but the recent trend to multi-axial classification in I.C.D.9 (World Health Organisation, 1978) and D.S.M.III (American Psychiatric Association, 1980) seems to have made the task a little less daunting. 'Psychosis specific to childhood' was the most common diagnosis made in the Camberwell survey (Corbett, 1977), being found in 17% of children, with 6% showing adjustment reactions and emotional disorders; conduct disorders and hyperkinetic syndrome were each found in 4% of his sample while a relatively large group, 12%, had to be classified under 'Special symptoms or syndromes not elsewhere classified'. Reid (1980), found conduct disorders to be most common, being diagnosed in 27 out of 60 clinic attenders. Of the remaining 33 children, 13 showed neurotic disorders, four had adjustment reactions, nine had the hyperkinetic syndrome, five had childhood psychosis, and one was manic-depressive leaving one girl unclassified.

There are references in the literature to subjects with Down's syndrome (DS) being less prone than other retarded people to the severe complications of mental retardation of which behaviour disorder is one (Kushlick, 1975). The personality stereotype of someone with DS is adaptable, humorous, and affectionate, with skills in mimicry (Smith & Berg, 1976), and recent studies appear to confirm that DS children are easy to manage (Gibbs & Thorpe, 1983; Gunn & Berry, 1985). In a review of the personality characteristics associated with DS, Gibson (1978) concluded that age, IQ, and ability to communicate are important whereas older children, particularly post-pubertal boys, are more likely to show aggression, hostility, or sullenness. However, in a longitudinal study, Carr & Hewett (1982) noted that 22% of the mothers of 11-year-old children with DS said they had worries about their children's behaviour, but that five years later, three quarters of the children were described as 'easy' and the rest as 'fairly easy'.

There are conflicting reports of the incidence of psychiatric disorder in DS children. Webster (1970) considered such children to have only minor degrees of emotional disorder, but on the other hand, Menolascino (1970), found 37% of a sample of 54 boys and 41 girls with an average age of 13.2 years to have a psychiatric disorder. The present authors followed a small group of children first seen in infancy (Gath & Gumley, 1984) and found that eight out of 14 boys and three out of eight girls had behaviour problems, as shown by deviant scores on standardised behavioural rating scales, but that nine

controls (normal children born at the same time to similar families) also had deviant scores on the same scales.

Method

Children with DS were identified, using the School Health Service records, in one Health Region in England. Each was matched, in the same school, with another child of the same sex and age and with a degree of physical and mental handicap as close as possible to the index child. The general practitioner was contacted before any approach was made to the parents of any child. Data were collected during the period 1979-1982: a member of the research team carried out semi-structured interviews with the parents in their homes. The parents were asked to complete the Rutter behavioural scale A2 for the identified child and for the sibling next in age who was also attending school; they also completed Part 1 of the Adaptive Behaviour Scale (Nihira *et al.*, 1974) and an Additional Behaviour checklist, consisting of items of behaviour found to concern parents of children with DS in a previous study (Gath & Gumley, 1984). With the permission of the parents, the clinical psychologist visited the school attended by the Child with DS and interviewed the teacher; the control child was selected at this meeting. Teachers completed the Rutter B2 scale, the Adaptive Behaviour Scale, and the Additional Behaviour checklist for both the children.

Results

Subjects

One hundred and ninety three children with DS, 94 boys and 99 girls, were enrolled: ages ranged from six to 17 years. The control group consisted of 154 children, 76 boys and 78 girls, attending the same schools and with a similar degree of verbal and motor handicap: 87% of the children with DS and 89% of the controls lived at home with at least one natural parent while seven children with DS and four controls were in long-stay hospitals. General practitioners had refused consent to the participation of one family with a DS child and of four families of the control group; 18 families with DS children and 16 families in the control group had refused to take part in the study. The refusal rates were thus 8.96% for the DS group and 11.5% for the control group, and 90% of the families approached in fact took part in the study.

Teachers completed the Rutter B2 rating scale and the Additional Behaviour checklist for 170 of the DS children and for 136 of the controls—a response rate of 88% for both groups.

Adaptive Behaviour Scale Scores: The range of raw scores on the Adaptive Behaviour scales were similar in the two groups (Figures 1 and 2). The means for each age group were similar in the Down's syndrome group and the controls.

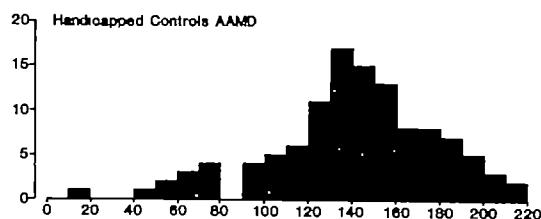


FIG 1 AAMD Adaptive Behaviour Raw Scores.

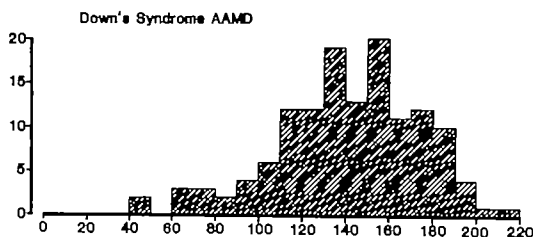


FIG 2. AAMD Adaptive Behaviour Raw Scores.

TABLE I
Down's syndrome children and similarly handicapped controls: deviant scores on Rutter behavioural rating scales

Scale used	DS children	Controls
	<i>n</i> = 193	<i>n</i> = 154
Rutter A2 scale		
(score 13 or more)	66 (34%)	63 (41%)
mean	11.23	12.84
	$(\chi^2 = 1.045, d.f. = 1 \text{ NS})$	
Rutter B2 scale	<i>n</i> = 170	<i>n</i> = 136
(score 9 or more)	53 (31%)	58 (42.6%)
mean	7.55	9.2
	$(\chi^2 = 3.8184, d.f. = 1, P < 0.05)$	

Rutter Behavioural Rating Scales: There were no significant differences in the percentages of children who had deviant scores, defined as 13 or more on the Rutter A2 scale, completed by the parents. The control children, however, were more frequently found to have deviant scores of nine or more on the Rutter B2 scale as completed by teachers (Table I). Parents reported more neurotic symptoms in the control than in the DS group. Both parents and teachers found significantly more hyperactive behaviour in the control than in the DS children. Anti-social sub-scores were similar in the two groups.

Additional Behaviour Checklists: Seventy three (38%) of DS children and seventy one (46%) of controls had scores

of ten or more on the Additional Behaviour checklists as completed by parents and 30% of both groups as completed by teachers. Individual items of behaviour on the checklists also showed few differences between the groups. The most frequent item reported was that the child became very distressed if corrected, which appeared to inhibit the parents from disciplining him or her. Aggressive gestures, rebellious behaviour, attention-seeking and interference with the persons or property of others were common in both groups; the least common behaviours were head banging, inappropriate use of the toilet (e.g. by drinking from it or stuffing it with paper), and fire setting. Behaviour associated with psychotic disorders in childhood, such as stereotypies, use of objects as twiddles or unresponsiveness were as frequently found among DS children as among the controls. Running away was reported in one in four of DS children (24.5%), but in only one in eight (12.5%) of the controls ($\chi^2 = 3.999, d.f. = 1, P < 0.05$). Shouting or screaming was reported in 33.5% of the controls, but in 19.4% of the DS children ($\chi^2 = 4.427, d.f. = 1, P < 0.05$).

Comparison with siblings next in age Ninety nine children, 56 boys and 43 girls, with DS and 92, 50 boys and 42 girls, among the controls had a sibling who was still attending school and within five years' age difference. In the DS families, there were 51 sisters and 48 brothers, and in the control families 32 sisters and 60 brothers who fulfilled the criteria. In the DS group, 58 were older and 39 were younger than the retarded child, while in the control group, 40 were older and 49 were younger. There were two sets of twins in the DS group and three sets in the control group. (None of the twin siblings were retarded.)

A2 behavioural rating scales were completed by the same parent for each paired retarded child and sibling: deviant scores were found in 30 DS children and 11 siblings ($\chi^2 = 9.996, d.f. = 1, P < 0.01$) and in 33 controls and 11 siblings ($\chi^2 = 13.17, d.f. = 1, P < 0.001$).

High neurotic, anti-social, and hyperactive sub-scores were each found in between 3 to 5% of the siblings—much less frequently than in the retarded children.

Children with no behaviour problems: Fifty nine children with DS (31%) and 46 controls (29%) had no deviant scores on either scales or on the Additional Behaviour checklists. These children had no more behaviour or emotional problems than children in the average range of intelligence and could certainly be regarded as normal or well adjusted.

Children showing severe behaviour problems: Seventy four (38.3%) of the 193 children in the DS group had high scores on at least one of the Rutter scales with a score of ten or more on the Additional Behaviour checklist, as rated either by parents at home or teachers at school, or by both. In the 154 controls, 74 (49%) also had significant behaviour problems or were definitely deviant by the same definition. Serious behaviour problems were more

TABLE II

Deviant behaviour in retarded children by age-groups DS

	Age-group (years)				Total
	I	II	III	IV	
	6-8	9-11	12-15	16+	
Sex					
Boys					
Deviant	13	14	9	8	44
%	50	51.8	37.5	47	47
Borderline	5	7	8	4	24
Non-deviant	8	6	7	5	26
Total	26	27	24	17	94
Girls					
Deviant	10	6	8	6	30
%	43.4	32	25	24	30
Borderline	8	8	10	9	35
Non-deviant	5	6	13	10	34
Total	23	20	31	25	99
Both sexes					
Deviant	23	20	17	14	74
%	47	42.5	32	32	
Borderline	13	15	18	14	59
Non-deviant	13	12	18	15	60
Total	49	48	55	43	193

TABLE III

Deviant behaviour in the retarded child by age-group in controls

	Age-group (years)				Total
	I	II	III	IV	
	6-8	9-11	12-15	16+	
Sex					
Boys					
Deviant	13	14	9	3	39
%	50.5	66	45	25	
Borderline	5	1	5	5	16
Non-deviant	5	6	6	4	21
Total	23	21	20	12	76
Girls					
Deviant	9	10	12	4	35
%	50	55.5	46	25	
Borderline	3	4	7	4	18
Non-deviant	6	4	7	8	25
Total	18	18	26	16	78
Both sexes					
Deviant	22	24	21	7	74
%	53.6	61.5	45.6	25	
Borderline	8	5	12	9	34
Non-deviant	11	10	13	12	46
Total	41	39	46	28	154

Down's syndrome children v. Handicapped controls

Boys	$\chi^2 = 0.314$, d.f. = 2	NS	
Girls	$\chi^2 = 5.819$, d.f. = 2	$P < 0.1$	NS
Both sexes	$\chi^2 = 4.474$, d.f. = 2	NS	

common in the children with low scores on the adaptive behaviour scales. There were 17 children in the DS group with adaptive behaviour scale scores of below 100 both at home and at school—well below the mean for any age-group; 14 (82%) had severe behaviour disorders and the other three were in the borderline group ($\chi^2 = 20.54$ d.f. = 4, $P < 0.001$). Eleven children with DS had very low adaptive behaviour scores in one situation only, either at home or at school, and seven of these also had severe behaviour disorders which tended to be situational—associated with low expectations or achievement.

Similarly, 11 (78%) of the controls with very low adaptive behaviour scale scores had severe behaviour disorders, as did all 7 children with low adaptive behaviour scale scores in one situation only ($\chi^2 = 15$, d.f. = 4, $P < 0.001$). Behaviour disorders tended to be less common in the most competent children in both groups.

Possible behaviour disorders: Sixty children with DS and 34 in the control group were regarded as on the behavioural borderline (Tables II and III). The difference between the two groups fell just short of significance at the 5% level ($\chi^2 = 5.7569$). More girls in the control group were deviant than in the DS group, the difference being very close to significance ($\chi^2 = 5.819$ d.f. = 2). Similar proportions of boys in the two groups had many behaviour problems.

Psychiatric Diagnoses: The majority of the children with severe behaviour problems could be classified, using Axis I of ICD 9, as shown in Table IV. DSM-III diagnoses agreed with those under ICD 9 except for 299.8 under ICD 9 'Psychosis with origin specific to childhood' where the DSM-III diagnosis of Pervasive Developmental Disorder was considered more appropriate but for its stipulation that the disorder had to have developed before the age of 30 months. Multiple diagnoses are possible on the DSM-III system so that, for example, hyperactivity and conduct disorder were both coded.

Two children with DS and three of the controls fitted the criteria for infantile autism. More boys than girls in both groups were diagnosed as having a 'psychotic' disorder, including autism, but this difference is not significant with the small numbers concerned. More girls than boys in the handicapped control group were judged as having an unsocialised conduct disorder, corresponding to 'unsocialised aggressive disorder' in D.S.M. III., in contrast to the DS group, where this diagnosis tended to be

TABLE IV
Psychiatric diagnoses of retarded children with deviant behaviour

<i>Diagnosis (ICD-9)</i>	<i>(1) DS</i>		
	<i>Boys</i>	<i>Girls</i>	<i>Total</i>
Infantile autism	2	0	2
Psychosis with origin specific to childhood	9	8	17
Unsocialised disturbance of conduct	13	8	21
Mixed disturbance of conduct and emotions	6	3	9
Disturbance of emotions specific to childhood and adolescence—other or mixed	3	3	6
Hyperkinetic syndrome of childhood—with developmental delay	2	2	4
Hyperkinetic conduct disorder	9	5	14
Undiagnosed	0	1	1
Total	44	30	74
<i>Diagnosis (ICD-9)</i>	<i>(2) Controls</i>		
	<i>Boys</i>	<i>Girls</i>	<i>Total</i>
Infantile autism	2	1	3
Psychosis with origin specific to childhood	14	10	24
Unsocialised disturbance of conduct	4	12	16
Mixed disturbance of conduct and emotions	7	4	11
Disturbance of emotions specific to childhood and adolescence—with anxiety and fearfulness	2	2	4
Hyperkinetic syndrome of childhood—with developmental delay	6	3	9
Hyperkinetic conduct disorder	1	1	2
Undiagnosed	3	2	5
Total	39	35	74

more common in boys. Hyperactive conduct disorder was a diagnosis which tended to be more commonly made in the DS group, but there were somewhat more controls who did not fit into any diagnostic category. Conduct disorders and psychoses were found in all age-groups, in both DS children and the controls, but hyperkinetic disorders tended to be found more, though not exclusively, in the younger children. Psychoses were found in the more able children with higher adaptive behaviour scores but were

more numerous in the children with lower adaptive behaviour scores.

Discussion

The results of this study reveal that a significant proportion of children show problem behaviour, which causes suffering to them and to their families. In this, they are little different from a group of children, whose retardation arises from many other causes.

When a measure standardised for use with children within the normal range of intelligence is used, many more of the children with DS have scores that are judged as deviant than do their siblings closest in age. The DS children are, in this respect, as different from their siblings as the controls are from their brothers and sisters. The two groups of retarded children also show a number of behaviour and emotional problems in addition to those rated on the Rutter scales. A similar proportion (30%) of children in the two groups of retarded children, had no behavioural or emotional problems on any of the measures used in our study. This finding is less, though not significantly so, than that of Chess & Hassibi, but is certainly larger than that found in most of the studies of clinic populations. The proportion of children with definite disorder is less than that found in most epidemiological studies of mixed groups of retarded children. However, in this study, a borderline group is included, since no evidence could be found for a clear cut off point between normal, well adjusted children and those with behavioural or emotional disorders. A continuum of deviancy is a more accurate concept than one of health and disease. Behavioural problems are much more common in those children who have been unable to learn appropriate behaviour, as measured by the adaptive behaviour scales, on which scores were found to be related to measured intelligence quotients in a group of children with DS who had been followed from infancy. It is therefore likely that severity of intellectual handicap is an important factor in the development of the disorders.

As in most other studies, the most common diagnostic group in our controls was that of psychosis (17.5% including autism)—very similar to the proportion found in Camberwell but less than in most clinic samples. The proportion of DS children with severe psychiatric morbidity was not markedly different at 9%, certainly higher than might be anticipated from most of the literature on DS. However, this label of 'psychosis' is unsatisfactory, not reflecting the flavour of the problems the children showed; Tanguay (1984) discussed the diagnosis of severe

psychopathology in children in a developmental context. The next revision of the two systems of disease classification may contain categories with less pejorative and more accurate labels. Conduct disorders form the most common diagnostic group for children with DS (11%) and are almost as frequently found in the controls (10%). Hyperkinetic conduct disorder was more often found in the DS group (which appears to contradict the finding that hyperactive behaviour was significantly more often found in the controls). The psychotic children, often with histories of brain damage, in the control group had very high hyperactive scores.

Ineichen (1984) has questioned the findings of some studies on the prevalence of what has been termed 'mental illness' in retarded patients, particularly in hospitals. It is suggested that some at least of such 'illness' may be attributed to the poor environment or to failure to teach appropriate skills. Only a very small proportion of the children in our study were living in institutions; most of the children enjoyed family life as did their siblings. However, many of their disorders, particularly those grouped

rather unhappily as 'psychosis with origin specific to childhood: other', did appear to be related to the failure to communicate and to learn more acceptable ways of attracting attention or even passing time. Psychiatrists themselves have suggested that many children who are retarded do not need the attention of psychiatrists (Tarjan, 1977; Wortis, 1977), and these would include nearly one-third of all the subjects in this study. Nevertheless, a sizeable proportion of retarded children, just under 40% of those with DS but nearly half of the mixed group of similarly retarded controls, do have severe and distressing problems that require a broad range of psychiatric skills and understanding in order to make a valid diagnosis.

Behavioural and emotional problems are a serious secondary handicap for between four and five out of ten children with mental retardation and children with DS have nearly as many problems of this sort as other children similarly handicapped. There is, however, clearly a role, though not an exclusive one, for the psychiatrist in investigating and treating the problems described.

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Family Background of Children with Down's Syndrome and of Children with a Similar Degree of Mental Retardation

ANN GATH and DIANNE GUMLEY

The families of Down's Syndrome children and another group with a similar degree of retardation were compared using data collected at interview. Differences in social class distribution were explained by the maternal age effect in the Down's group. Divorce was more common in the control group but the quality of marriage in those parents still together and the health of parents were similar. No close associations were found between behaviour disorders in the children and family factors, except that behaviour disorders were likely to be associated with similar disturbance in siblings next in age and to be more common in the less happy marriages.

The literature concerning families of mentally retarded children is mostly concerned with the effect upon parents and siblings of the additional burden that bringing up a handicapped child imposes. Evidence from earlier studies was used to support the case for long-term hospital care; even families who

wanted to bring up the retarded child at home often felt themselves driven to request such care for their children because they could not cope with the demands upon them, (Oster & van den Tempel, 1975). The emphasis thereafter moved strongly to "community" care, and with more positive attitudes

to retarded children, including provision of education for all children, and some social support such as occasional relief care, more encouraging findings from research on families has appeared. One long-term study (Carr & Hewett, 1982) followed infants with Down's Syndrome (DS), originally investigated by Cowie (1970), to the age of 16, and found that a number of families were coping well and enjoying a good life in many ways. Similar positive findings were also noted in the follow-up of another group of children with DS first seen in infancy (Gath & Gumley, 1984).

There are, however, indications that some adverse effects on families occur quite early on, before the care of the child itself becomes very different from that of a normal child (Gath, 1977, 1978). These early effects are concerned with the 'value crisis' (Wolfensberger, 1968) which follows the initial shock, and occurs when the parents are forced to revalue many of their previous beliefs. Some marriages did not survive this revaluation, and others continued with deep rifts. Later effects, such as increased psychiatric morbidity in parents, shown when the children were aged eight or nine, are more related to what Wolfensberger called the 'reality crisis' and Tizard & Grad (1961) the 'burden of care'.

Recent research on families of mentally retarded children has moved from the negative approach of measuring what was assumed to be the inevitable stress upon other family members to looking at positive and negative effects in both directions. Strengths as well as weaknesses of parents and siblings are noted, particularly in relation to their ability to cope with not only the added work of caring for a more dependent child, but also the considerable demands of treatment. Objective measures of the way families actually live with a retarded child have now been developed. Bringing up such a child is extremely hard work, especially for the mother (Dupont, 1980; Wilkin, 1981; Pahl & Quine, 1984). Parents are now expected to take an active part in early education as well as in any physiotherapy or behavioural therapy which may be prescribed for the common additional problems. Even when these secondary handicaps become very severe, it is no longer common practice to admit the child for intensive therapy away from the family as the improvement often fails to generalise on discharge. Instead, the other family members are enrolled as co-therapists from the start and must learn and then continue the treatment programmes devised by professionals.

Our previous paper (Gath & Gumley, 1985), describes the behaviour problems in two groups of

retarded children, one with DS, which makes up the single biggest diagnostic group in special schools, and the other of children chosen by the teachers as having comparable verbal and physical abilities. Behaviour disorders were common in both groups, but slightly less so in the DS group. The present paper aims to compare the socio-demographic backgrounds of the two groups, and to look for factors particularly concerned with the presence of behaviour disorders in retarded children.

Method

Children with DS were identified in schools in one Health region and were matched where possible with a similarly retarded child in the same class. The parents were interviewed in their homes using a semi-structured interview which was recorded on tape and coded by an independent rater. Part of the interview consisted of an assessment of the quality of the parents' marriage based on that devised by Brown & Rutter (1966) and used in previous studies of the families of retarded children (Gath, 1977, Gath & Gumley, 1984). A judgement was made of the quality of the marriage, particularly of the warmth and/or hostility evident throughout the interview. A further judgement was made of the state of mental health in both parents; this was based on the General Health Questionnaire (Goldberg, 1972) and on answers to questions concerning symptoms in the interview. Degree of incapacity was determined from the ability to work or to cope with home chores and the impression from the interview as a whole. This second judgement was agreed upon by the interviewer, the rater, and the first investigator.

Specific questions were asked about the sibling closest in age to the retarded child. Parents completed the Rutter A behavioural rating scale and, with their permission, teachers completed the Rutter B behavioural rating scale and a school report form.

As described by Gath & Gumley (1986), all the retarded children had been assessed on the American Association for Mental Deficiency Adaptive Behaviour scale (Nihira *et al.*, 1974). They had been divided into three groups—those with definite serious behaviour disorders, those with no behaviour disorder and a borderline group.

Results

Family interviews had been carried out in the homes of 192 of the children with DS and 147 of the children with a similar degree of mental retardation.

Age. The mean age of the children with DS was 11.77 years with a range of 6–17. The mean age of the control children was 11.82 years

Home and parents: One hundred and sixty-three (85%) of the children with DS lived at home with both natural parents. Thirty-one children with DS and 12 controls were

not living full-time with at least one natural parent. Three DS children were in boarding schools out of the region, but returned home for holidays and half terms. All the other children went to schools in the region, as did all the controls. Ten DS children and 3 controls were in the hostels run by the local authority, but usually went home for holidays and often for weekends as well. Comparable children were found as controls for four of the seven children with DS who were in long-stay hospitals for mentally handicapped patients. No suitable matches could be found for those in boarding schools. Three children with DS were adopted, as were two of the control children. Four families had made the adoption in the full knowledge of the child's retardation and associated medical diagnosis. The exception was one couple whose originally normal baby, adopted at three weeks, had suffered severe brain damage following encephalitis at the age of four. Eight DS children and 3 controls were in long-term foster care.

Parents' understanding of the handicap: Since DS is apparent from birth, there were marked differences between the two groups of parents as to when they first understood that their child had a handicap: 42% of the parents of the DS children said they knew immediately after the birth, and a further 30% said they knew during in the first week, compared with only 9% of the parents of the controls who had learnt by the end of the first week. Only 3% of the DS group felt they had not clearly understood about the handicap until after the first birthday, while 41% of the parents of the controls had learnt about it between the first and fifth birthdays, and in 9% of cases, the child had already reached compulsory school age before they knew.

All the parents of the DS group knew that their child had the syndrome or 'was a Mongol'. In 13 of the controls, there was no information about the parents' understanding of the child's handicap. Fifty (34%) thought the child had brain damage, 13 (9%) mentioned a specific syndrome, e.g. San Filippo disease, 53 (36%) had been told that there was no medical disorder but that their child was 'just generally backward' and 18 (12%) believed there was an underlying medical condition, as yet undiagnosed.

Ethnic origin: The majority (180, 93%) of the children with DS were Caucasian, with parents and grandparents born in the British Isles. Four were children of Caucasians from other European countries, five were children of Asian immigrants and five had parents born in the West Indies. The ethnic background of the control group was very similar, with four from other European countries, three from Asia and one from the West Indies.

Family size: The families in the Down's were larger than those in the control group, but the difference was not great. The mean sibship size in the Down's families was 3.44, with a range from one to ten, while that of the other families was 2.9, with a range of one to eight. Mean sibship size dropped from 4.0 in the families of the 16 year old children to 3.07 in families of the children aged 6-8 in the DS

group, but remained much the same in each age-group in the families of the controls. Fourteen DS children and 11 of the controls were only children, i.e. 11% of both groups.

Slightly fewer, 37 (22%) of the DS group, were first-born, compared with 53 (39%) of the control children. The mean ordinal position of the retarded child was 2.9 in the Down's group and 2.3 in the controls. Just over half the children with DS were the last born in the families, excluding only children, as were 63 or 46% of the controls.

Social class: There was a shift towards social classes I and II in the Down's as compared with the control group, ($\chi^2 = 7.257$, d.f. = 2 $P < 0.05$). The social class difference was insignificant in the children aged 12 or more, but was most marked in the youngest group, aged between six and eight at the time of assessment. The social class of 14 families in the DS group and of five families in the control group was not ascertained, as the fathers were either long dead or divorced, and their job status was unknown. Family size was related to social class in the control group only (Table I). Large families were common in all social classes in the DS group. There was a marked social class difference related to the parents' understanding of the nature of the child's developmental difficulties, with over-representation of social classes IV and V in the group who believed that there was no medical explanation for their child's retardation. The social class distribution of the DS group more closely resembled that of the families of the brain-damaged children or of those with specific syndromes.

Maternal age: Mothers of the children with DS were older at the time of the birth than were those of the controls, the mean ages at the time of the birth being 33.53 and 28.03. The age gap was greatest in the small group where the social class of the father was unknown. As expected, mothers who had been aged 40 or more at the birth of the retarded child were much more common in the DS group, numbering 58 (28%) as against only seven (5%) in the control group ($\chi^2 = 29.3$ d.f. = 1, $P < 0.001$). By contrast, there were 13 (9%) young mothers aged 20 or less at the birth of the child in the control group, and six (3%) in the Down's group, ($\chi^2 = 4.14$, d.f. = 1, $P < 0.05$). The mean age of mothers and the total family size tended to be less in the younger groups of children. Mothers of DS children were more likely to have done well at school and to have had further education: 26 mothers of DS children had had professional training, particularly as nurses or teachers, and ten had a university degree, compared with ten and two respectively of the control mothers, ($\chi^2 = 15.37$, d.f. = 3, $P < 0.001$).

Marital relationship of the parents: The natural parents were no longer together in 32 families in the DS group and in 35 families in the control group. There were nine widows and one widower in the Down's group and six widows in the control group. Twenty-three marriages were broken by divorce in the control group, but only ten in the DS group,

TABLE I
Family size and social class

	Down's syndrome				
			<i>Social class</i>		
<i>Family size</i>	I & II	III	IV & V	<i>Not known</i>	<i>Total</i>
1	6	5	2	1	14
2 or 3	46	42	20	1	110
4 or more	16	28	14	11	68
Total	68	75	36	13	192
$(\chi^2 = 4.943, \text{d f.} = 4, \text{NS})$					

	Controls				
			<i>Social class</i>		
<i>Family size</i>	I & II	III	IV & V	<i>Not known</i>	<i>Total</i>
1	3	6	2	0	11
2 or 3	27	46	22	3	98
4 or more	3	17	16	2	38
Total	33	69	40	5	147
$(\chi^2 = 9.3328, \text{d.f.} = 4, P < 0.02)$					

$(\chi^2 = 7.3, \text{d.f.} = 1, P < 0.05)$. There was no significant difference between the quality of the marital relationship in those families where the natural parents were still living together. In four of the DS group and one of the controls, no information was available on the natural parents. Good marriages were evenly distributed throughout the social classes in both groups, but very poor marriages tended to be more frequently found in social classes IV and V.

The health of the parents: The physical health of the mothers was considered to be a problem in 17% of the families of DS children and 13% of the families of controls. A similar degree of concern about the fathers' health was expressed by 15% of families of DS children and by 12% of controls. Many mothers described themselves as suffering from 'nerves', but there were slightly more of these in the control group—42% as opposed to 39%. Fathers were less commonly affected by 'nerves'—22% of the control group and 14% of the Down's group. Scores of ten or more on the General Health Questionnaire were obtained from seven mothers and two fathers in the Down's group and six mothers and seven fathers in the control group.

Severe mental illness (incapacitating, necessitating admission to a psychiatric hospital and/or inability to cope for at least a month in the past year) affected 2.8% of the whole group of parents and was evenly distributed among mothers and fathers in both groups. There was one case of manic-depressive psychosis in the DS group and one case of schizophrenia in the control group. The remainder of the affected parents suffered from depression (Table II). Minor disorder, causing distress and some impairment of

TABLE II
Mental health status of parents of Down's syndrome children and controls

	Well	Minor disorder	Severe	Total
<i>Mothers</i>				
Downs syndrome	130	41	5	176
Controls	105	29	4	138
Total	235	70	9	314
<i>Fathers</i>				
Downs syndrome	125	17	3	145
Controls	93	17	4	114
Total	235	34	7	259

Down's Syndrome v controls.

Mothers: $\chi^2 = 0.2325, \text{d.f.} = 2, \text{NS}$

Fathers: $\chi^2 = 1.1461, \text{d.f.} = 2, \text{NS}$

Mothers v. Fathers.

Down's syndrome: $\chi^2 = 7.606, \text{d.f.} = 2, P < 0.05$

Controls: $\chi^2 = 1.585, \text{d.f.} = 2, \text{NS}$

the capacity to cope, was found in twice as many mothers as fathers in both groups. There were few differences between the groups, but despite more mothers of the controls reporting problems with 'nerves', a clinical psychiatric disorder was judged to be present slightly more often in the mothers of the DS group. A mixture of anxiety and depressive symptoms was reported in most cases. In all, 46 mothers and 20 fathers in the DS families were judged as

TABLE III
Psychiatric disorder in parents and quality of parents' marriage

	Marriage rating				
	Good	Moderate	Poor	Widowed	Divorced
<i>Down's syndrome</i>					
Parents with psychiatric disorder	9	25	10	2	7
Both parents well	82	32	1	8	5
Total	91	57	11	10	12
<i>Controls</i>					
Parents with psychiatric disorder	13	14	9	2	8
Both parents well	46	35	0	4	15
Total	55	49	9	6	23

having a psychiatric disorder. In 12 families, both parents were affected; 33 mothers and 21 fathers in the control group had a psychiatric disorder, with both parents affected in eight families. Psychiatric disorder in the parents of both groups of children was closely correlated with the quality of the parents' marriage (Table III).

At least one parent, but in many cases both, had a psychiatric disorder in ten of the eleven most disharmonious marriages in the Down's group and in all the very poor marriages in the control group. Widowed and divorced mothers, by contrast, were not particularly vulnerable in this respect. Psychiatric disorder showed a gradual increase with social class. In class I, 19% of the children with DS and 20% of the controls had a parent with psychiatric disorder. The percentages of affected families in social classes IV and V rose to 30% for DS and 35% for the controls. Psychiatric disorders was not more common in older mothers.

Brothers and sisters There were 99 siblings in the DS families and 92 in the families of the control children who were still at school and aged within five years of the retarded child. Eleven (11%) of the siblings of the DS children were regarded by their parents as having significant problems and 12 (13%) of the control siblings. The ratings made by the parents were very similar to those on the Rutter A scale where five boys and six girls in the DS group and seven boys and four girls in the control families had high scores. More siblings, 15 boys and eight girls, in the families of the controls had high scores on the Rutter B behavioural scale, as rated by the teachers, compared with nine brothers and eight sisters of the DS children. There were no significant differences between brothers and sisters or older or younger siblings in either group. Behaviour disorder in the siblings was closely related to that of the retarded children in the families of DS children but not in the families of the controls, (Table IV)

TABLE IV
Disorder in the retarded child and disorder in siblings

	Normal siblings		
	With behaviour problem	No behaviour problem	Total
<i>DS children</i>			
With behaviour problem	18	22	40
No behaviour problem	6	53	59
Total	24	75	99
	$\chi^2 = 13.9075$, d.f. = 1, $P < 0.001$		
<i>Control children</i>			
With behaviour problem	16	27	43
No behaviour problem	15	34	49
Total	31	61	92
	NS		

Practical aspects of family life: Very few families employed paid help. The eight who did have paid help all came from the DS group; five were from social class I and three from class II. More families in the control group depended upon a grandmother of the child for help: 35 (24%) of the controls and 27 (14.5%) of DS children had grandmothers who were a regular and important source of help ($\chi^2 = 4.087$ d.f. = 1, $P < 0.05$). Sixty-one (33%) of the families of the DS children were helped regularly by a neighbour or friend, compared with 33 (22%) of the families of the controls ($\chi^2 = 3.84$, d.f. = 1 $P < 0.05$).

Slightly fewer in the control group of families (48%) had regular annual holidays than did the families in the

DS group (58%). Similar numbers went on family trips altogether or the parents went out as a couple. Only 8% of each group felt they had major problems making baby-sitting arrangements.

Support services: During the pre-school years, the family doctor and health visitor provided most support, and despite the more regular contact of the health visitor, parents were more appreciative of the help offered by the doctor. A significant minority, 29%, in both groups, could name no reliable source of help or advice when their children were small. Once the child had gone to school, more (21%) of the parents in the DS group still felt they had no one to turn to, compared with 12% of the control parents. The doctor's involvement did not lessen although in 15% of cases the school staff were rated as equally important. The teachers were consulted about problems almost as frequently as doctors, and a fifth of the parents regarded them as the main source of help with the retarded child. Social workers were seen as closely involved in less than 3% of all the families.

The majority of parents, more than 80% in each group, felt that the school concerned was the right one for their child's particular needs. Relief care was available for the families. 44% of both groups of families used hostels, of which there was at least one in each county. Slightly more, (25, 13%), of the DS children went to a hospital for relief care than did the controls (12, 8%). Half of the parents were very appreciative of the relief care available, finding it provided a useful break for them and often a good experience for the child. However, 30% of both groups of parents were clearly dissatisfied with the services provided for their children.

Pre-school education. The pre-school educational opportunities for the children were different in the groups: 89 DS children and 37 controls had been to either a play group specifically for handicapped children or a nursery in a special school ($\chi^2 = 15.35$, d.f. = 1, $P < 0.001$). Conversely, 70 of the controls and 55 DS children had been to an ordinary play group, catering for normal children ($\chi^2 = 10.67$, d.f. = 1, $P < 0.01$). However, an additional 17 DS children and four controls had had the advantage of both sorts of pre-school provision.

Early intervention for handicapped children had only just become available when these children were small. 11 of the DS children and eight of the controls had been visited regularly by a teacher or counsellor in the first five years. None of the DS children who had had some early intervention had adaptive behaviour scores significantly below the average for the age-group but four had behaviour disorders and a further four were in the borderline group. The children in the control group given early intervention included three multi-handicapped children with very low adaptive behaviour scores and much deviant behaviour; only one child in this group had no behaviour problems.

Adaptive behaviour and parental psychiatric illness: Parents with psychiatric disorder were not more commonly found

in those families where the child had very low AAMD Adaptive Behaviour scores. Neither was additional physical disability, indicated by a low score on the physical development sub-scale, nor a low language score associated with a higher incidence of parental psychiatric illness.

Parents' perception of behaviour problems in the retarded child: The parents thought that 29 (15%) of the DS children and 37 (25%) of the controls had significant behaviour problems. These figures are much lower than the numbers of children judged as having serious behaviour disorder from the rating scales and behavioural check-lists. Hyperkinetic or bizarre behaviour were the most likely types to have been seen as problems by the parents, but they were not especially likely to see the children with very low adaptive behaviour as also having behaviour problems. Mothers with psychiatric disorder themselves were more likely to perceive their DS children as having significant behaviour problems, but this was not so in the control group. In the borderline group, there were four DS children identified by parents as having significant problems and two others in the group judged as normal. Three judged as 'borderline' and two as 'no disorder' in the control group were considered by their parents to have behaviour problems.

Factors associated with behaviour disorders: Behaviour disorder in the retarded child, as defined in the previous paper, were not related to family size, to age or educational achievements of the mother, to the physical health of either parent, or to relationships with members of the extended family. Neither was there evidence of association with social class, since the frequency of definite disorder was not significantly different in any social class in either group.

Three of the seven Down's children and three out of the four control children in the long-stay hospitals were found to have serious behaviour disorders. Disturbance was common also among those in local authority hostels, in five out nine DS children and in all three controls in hostels. By contrast, only one in eight of the fostered and one of the three adopted DS children had serious behaviour disorder. The adopted and fostered control children had more behaviour disorder, affecting all but one fostered child.

Behaviour disorders in the children and psychiatric disorder and quality of marital relationship of parents: Behaviour disorders in the controls were more frequent in the children whose parents' marriage was rated as moderate or poor than in those where it was rated as good (Table V). The same trend, to a lesser degree, could be seen in the DS families. There were fewest behaviour disorders in DS children of widowed or divorced mothers, with a clear difference between them and the children where the marriages were rated as moderate or poor, ($\chi^2 = 4.63$, d.f. = 1, $P < 0.05$). In the control group, the highest rate of disorder was found among children of widows, the rate in the

TABLE V
Disorder in the retarded child and quality of the marital relationship between parents

	Good	Quality of marriage Moderate	Poor	Total
<i>DS Children</i>				
Definite disorder	33 (36%)	26 (46%)	6 (55%)	65
Borderline/no disorder	58	31	5	94
Total	91	57	11	159
	$\chi^2 = 2.18$, d.f. = 2, NS			
<i>Control children</i>				
Definite disorder	19 (35%)	24 (49%)	7 (77%)	50
Borderline/no disorder	36	25	2	63
Total	55	49	9	113
	$\chi^2 = 6.645$, d.f. = 2, $P < 0.05$			

TABLE VI
Disorder in the retarded child and psychiatric disorder in the parents

	Parents' mental health		Total
	One or both parents with severe disorder	Parents well mild disorder	
<i>Down's group</i>			
Disorder in retarded child	4	16	54
Borderline or no disorder in retarded child	4	28	86
Total	8	44	140
<i>Control group</i>			
Disorder in retarded child	6	17	48
Borderline or no disorder in retarded child	21	54	76
Total	7	38	102

(Down's $\chi^2 = 0.5316$, d.f. = 2, NS)

(Controls $\chi^2 = 4.1802$, d.f. = 2, NS)

divorced group being very similar to that found in association with poor marriages. Parental psychiatric disorder was most common in social classes IV and V, affecting 30% of families so classified in the DS group and 35% of families in the control group. Behaviour disorders in the retarded children in these social classes occurred just as commonly in families with well parents as in families with a psychiatrically ill parent. Psychiatric illness in the parents was likely to be associated with a poor marriage relationship. In the DS families with a good marital relationship,

parents with psychiatric disorder were more likely to have a retarded child who had a behaviour disorder, although the numbers in this group were small. In the context of a moderate or poor marriage, there was no demonstrable association between parental psychiatric disorder and behaviour disorders in the DS children themselves. Similarly, there was no strong association of parental psychiatric illness with behaviour disorders in the control group, except where there was severe mental disorder in the mother or father (Table VI).

Discussion

There was greater maternal age and a higher level of maternal education, with a bias towards upper social class, in the families of the children with DS. In this study, the older mothers included a significant proportion of women with degrees or professional training which had necessitated postponement of child bearing. Younger children with DS tended to come from smaller families, unlike those formerly typical—usually in social classes IV and V—where a child with DS would be born at the end of a long sibship. The social class distribution of our controls did show an opposite bias towards social classes IV and V, as has been reported in other recent studies of the family background of retarded children (Cooper & Lackus, 1984).

We found no evidence that older mothers were more vulnerable than younger ones, or that they were less effective in rearing these difficult children. There was less support from grandmothers in the DS group, which can be explained on grounds of age, as these older mothers did not themselves have mothers capable of offering them support.

The comparatively easy recognition of DS influences decisions made not just by the families themselves but by the community on how the child is reared, as is seen in the preschool period. The children are set off on a different pathway early in life, long before any objective measure can be made of the extent of intellectual impairment.

There is little evidence from this study to support the idea that bringing up a child with DS is markedly less stressful than rearing one with mental retardation due to other causes, although indications of other social problems were seen in the control families. No large differences in morbidity emerged among the parents in the two groups. Divorces were more common in the control group, although the quality of those marriages where husband and wife remained together differed little.

The incidence of psychiatric disorder in both groups of fathers was not markedly higher than that found in the general population, of whom 10.9% in surveys using the PSE have sufficient symptoms to be regarded as 'cases' (Wing *et al.* 1981). The higher incidence in the mothers can be interpreted as supporting the theory that they carry most of the burden and show most ill-effects. However, even the finding of 24% is not dissimilar to those of maternal disorder in families bringing up normal children under difficult circumstances (Brown & Harris, 1978; Richman *et al.* 1982).

Unhappy marriages are clearly associated with psychiatric disorder in parents in this group, and

both adverse effects occurred most frequently in the socially deprived families, in social classes IV and V. A mentally retarded child and an unhappy marriage in poor social circumstances add up to a high level of adversity (Pahl and Quine, 1984). There is a stronger association between behaviour disorders in the retarded child and marital discord in the control group than in the DS group.

The two groups of siblings of handicapped children had very similar rates of disturbance at home, but there was a difference, although not a large one, at school where the siblings of the control group were more often rated as deviant. An earlier finding, (Gath, 1974) that older sisters were particularly vulnerable has not been replicated in this study, ten years later. At that earlier time, their vulnerability was thought to be possibly due to the excessive burden of domestic responsibility. Improvements in provision for mentally retarded children may have contributed to the disappearance of sisters as a particularly vulnerable group, but in the 1974 study of siblings, 14 out of 89 sisters (16%) and 22 out of 85 brothers (26%) of children with DS were rated as deviant, using the same behavioural rating scales. In the present study, deviant scores were obtained by 25% of sisters and 23% of brothers. An alternative explanation might be that over the years, deviancy among all girls, including the sisters of DS children, has caught up with that previously found only in boys.

The behaviour disorders in the DS children showed a close association with psychiatric disorder in the siblings, as defined by deviant scores on either of the two Rutter scales. However, no such association was noted in children and siblings in the control group. A more detailed comparison of the retarded child/sibling pairs will be the subject of a further paper.

Parents appeared to show a remarkable degree of tolerance towards abnormal behaviour in their retarded children. Using objective measures, many more children were found with serious behaviour disorders than had been identified as such by their parents. Many parents regarded behaviour difficulties as an integral part of the handicap, and their attitude was one of resignation rather than tolerance. Psychiatrically disordered mothers of DS children did tend to perceive their children as having behaviour problems, whether they actually had them or not.

In child psychiatric literature, the association between parental psychopathology and childhood psychiatric disorder is well known. Various explanations have been suggested—genetic, based on family studies (Crowe 1983) and adoption studies

(Bohman, 1978) and environmental, the latter with particular emphasis on the effects of marital discord which often accompanies parental psychopathology (Quinton & Rutter, 1985). Most published work on families of handicapped children, particularly those who are mentally retarded, looks at the problem from the opposite point of view—that the child's disturbing behaviour is the cause of 'stress' to the parent, and hence of the symptoms, both physical and mental, which may result. Stress in our DS families is shown by the mother's perception of the child's behaviour as a problem.

Behaviour disorder in the DS child in this study was as common in social class II as in IV and V. This certainly does not fit with the findings on children in the normal range of intelligence, and is more in keeping with the hypothesis that in families with a retarded child the development of a behaviour disorder in that child is related to factors within the child rather than to environmental influences, especially within the family. A retarded child with a behaviour disorder in the home is certainly stressful, but psychiatric disorders in the parents studied by us, particularly the mothers, bore no simple relationship to the presence of behaviour disorder in the child, any more than they did to other measures of handicap. Other social problems compound this issue in the most deprived families, where the greatest ill-health and marital misery is found, but different factors must be operating in classes I and II.

The longitudinal study (Gath, 1977) showed that effects on a marriage occurred very soon after the birth of a child with DS and our follow-up of the families of children first seen in infancy indicates that behaviour disorders are likely to develop in families which have long-standing problems. In the present study, families where both parents were well and had a good harmonious marriage were in the minority in both DS and control groups, but where they occurred, they did so irrespectively of the ages and sexes of the affected children.

However, family influences alone can by no means explain the development of behaviour disorder either in children with DS or in controls. In the longitudinal study, communication problems with marked speech delay were found to be closely related to behaviour disorders, as also was general severe retardation. Both groups of children in this present study have been assessed using Adaptive behaviour scales and more detailed psychological testing of the DS children has also been carried out. These findings will be further analysed in relation to behaviour disorders in a later paper.

Conclusion

The family backgrounds of children with Down's syndrome have somewhat different socio-demographic features from those of other children with a similar degree of intellectual retardation. These differences can be summed up as a bias towards relative social advantage and easy parental recognition of the handicap in contrast to an opposite bias towards social deprivation and parental uncertainty in the control group whose retardation resulted from various known and unknown causes.

In our previous paper, the myth of the child with DS being an easy child was found not to be valid, so the apparent advantage of early diagnosis and the higher chance of being born to more educated parents does not spare the majority of families from the initial grief or long years of very hard work involved in rearing them. Marital discord is common and is associated with behaviour problems in the retarded child. This was even more true in the families of our control children than in families of those with DS. The finding that psychiatric morbidity is higher than in the general population in mothers, but not in fathers, in both groups suggests that the carer, usually the mother, has a heavy burden and is at risk of developing psychiatric disorder. That the risk is much the same for other women rearing children in difficult circumstances does not detract from the importance of this finding, since the others are likely to improve as their children grow up, whilst mothers of mentally handicapped children will face an even greater problem as their children leave school but not home. The options are least for those children who are doubly handicapped by difficult behaviour as well as mental retardation. Families who would be able to cope adequately with the demands of ordinary family life are now being asked to take on an exceptional role with no time off for an indefinite period. Even the most stable and healthy families require more help from outside than is easily available at present. Those with additional problems will need the services even more desperately but may be less able to obtain the help to which they are entitled. Services, therefore, must be sufficiently flexible to be able to respond to individual needs but this must not be at the expense of either the self-respect of the family or the well-being of the child.

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Psychiatric Disorder in the General Hospital

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There have been many reports of psychiatric disorder in medical populations, but few have used standard methods on representative patient groups. Even so, there is consistent evidence for considerable psychiatric morbidity in in-patient, out-patient and casualty department populations, much of which is unrecognised by hospital doctors. We require a better classification of psychiatric disorder in the general hospital, improved research measures, and more evidence about the nature and course of the many different types of problem so that we can provide precise advice for their management of routine clinical practice.

Psychiatric problems in general hospital patients are important, both because they are often easily treated and because they can also affect the course of medical conditions. However, it is uncertain which disorders benefit from extra treatment and how common these are in the various types of medical population. Most psychiatric research in the general hospital has been concerned with specific types of physical disorder, but we also need to know the overall prevalence of psychological problems in general hospital units as a basis for planning services, including liaison psychiatry, to meet clinical needs. In this review we examine the various forms of psychiatric disorder seen in the general hospital, and discuss their prevalence, clinical significance and the implications for management. We do not cover findings from hospital-based primary care, both because there is a large separate literature (see Goldberg & Huxley, 1980; Cavanaugh & Wettstein, 1984) and because the issues involved are somewhat different.

Research methodology in general hospital surveys

To obtain a full picture of psychiatric disorders in the general hospital, we need to know: the extent and nature of psychiatric morbidity; its relationship to the different types of physical illness; the degree to which it reflects a long-term history of psychiatric disorder or current social problems, or is more specifically related to the physical illness or hospital admission; and the medical, psychological and social outcomes. Unfortunately, there have been relatively few studies comparable to the extensive research examining these same issues in primary care (Goldberg & Huxley, 1980). Research in the general hospital has largely been of poor quality, failing

to describe adequately the medical populations surveyed or to use an adequate nomenclature or standard measures (Cavanaugh & Wettstein, 1984).

The characteristics of the medical population

Geographical, social and medical factors mean that there are considerable differences between hospitals in the nature of referrals and admissions. Few reports have adequately described the populations studied, in terms of age, sex, social class, types of medical problem and source of referrals. Some studies provide point or period prevalence, while others are surveys of admissions or new attenders. Not only are there differences between hospitals, but the pattern of medical care within hospitals is changing rapidly. For example, between 1972 and 1983 the proportions of emergency and elderly patients admitted to a medical unit in Oxford have greatly increased and the length of stay decreased (Maguire *et al*, 1974; Feldman *et al*, 1986).

In most studies it is not clear what efforts have been made to obtain information on patients who refused or were too ill to be interviewed, or whether they have been included in the analyses. Exclusion of such patients causes an under-estimate of psychiatric morbidity, since they are likely to have a relatively high prevalence of disorder—especially organic mental states (Feldman *et al*, 1986).

Nomenclature and case definition

A fundamental problem is that psychiatric classification and concepts are almost entirely based on research with psychiatric in-patients, and there has been little attempt to adapt them for use in general populations, primary care or the general hospital

(Williams *et al*, 1980; Goldberg, 1981; WHO, 1983). It is particularly difficult to distinguish between 'appropriate distress' and affective disorder, especially in the presence of medical symptoms. There are no generally accepted criteria for organic disorders and substance abuse. It is also common to see other patients who are not psychiatrically ill but who require extra help because their personality or behaviour is thought to be maladaptive—non-compliance, for instance, and some cases of deliberate self-harm. New methods of clinical classification are required if we are to define and sub-classify problems that need extra help.

Further difficulties arise because it is uncertain whether measures developed in specialist psychiatric populations can be used in the same way with patients who are physically ill. Even if simple screening instruments can be successfully adapted for epidemiological research, they may be less useful in clinical 'prescriptive' screening, which should be accurate enough to identify all those who may require further intervention (Skuse & Williams, 1984).

Affective disorder

Standardised measures

Most surveys have relied on simple screening methods, but a minority used two-stage procedures in which all screened 'cases' were interviewed. Earlier studies used unstandardised interviews or scales (see Cavanaugh & Wettstein, 1984), whereas most recent reports are based on established psychiatric procedures. Unfortunately, none have been adequately validated for use with the physically ill (Williams *et al*, 1980) and research in general practice has shown that there are considerable difficulties in identifying affective disorder in the presence of physical symptoms or illness (Goldberg & Huxley, 1980). On the one hand, standard procedures fail to detect 'masked' depression and anxiety in patients who 'deny' their emotional distress; on the other hand, they over-estimate disorder, because many patients have physical causes for fatigue and other somatic symptoms which are prominent items in all instruments used to detect emotional disorder. The recently described Hospital Anxiety and Depression scale (HAD) is the first to be designed specifically to avoid these problems (Zigmond & Snaith, 1983).

Subclassification into the specific diagnostic categories is often inappropriate, and it would be more useful to use a clinical classification similar to that proposed for primary care by Goldberg (1981):

- (a) major psychiatric illness—a small group which broadly corresponds to psychotic illness, and responds to physical treatments

- (b) psychological distress syndromes not requiring intensive intervention; some of these are transient reactions to events such as physical illness. For these, simple exploration, discussion and advice may be helpful

- (c) psychological distress syndromes which may require some form of more active intervention, such as drug treatment or social or psychological help.

These same groups are also apparent among patients with psychiatric morbidity in the general hospital, although there has so far been little attempt to distinguish between transient and persistent distress. It is to be hoped that eventually these difficulties of case definition will be resolved by the introduction of a multiaxial classification which will enable psychological syndromes, physical illness, social problems, and perhaps personality, to be coded on separate axes (Goldberg, 1981; Williams *et al*, 1980; WHO, 1983).

We consider below examples of three types of standard measure—self-report screening questionnaires, standardised clinical interviews, and symptom rating scales.

General Health Questionnaire (GHQ). This is by far the most popular screening instrument, in its full 60 item, or abbreviated 30 and 28 item versions (Goldberg, 1972; Goldberg, 1978). The 30 item version (GHQ-30) has often been preferred as being the least dependent on somatic symptoms. Since the GHQ is based on recent change in an individual's psychological state, it does not identify all patients with chronic disorders, and Goldberg (1983) now recommends two extra questions about use of psychotropic medication and history of nervous problems. A practical problem is that many elderly, confused and seriously ill patients find it difficult to complete the questionnaire, although this can often be overcome by reading out the questions.

Goldberg has always emphasised that calculated specificity and sensitivity depend on the nature of the screened population and that optimal discrimination depends on the correct choice of threshold (Goldberg, 1985). It is apparent that in medical populations both sensitivity and specificity are low, especially in the most severely physically ill groups (Table I). The high proportions of false positive cases are usually attributed to a number of items which are commonly answered by those who have physical symptoms or disabilities. It is possible to improve specificity by raising the cut-off threshold, but this reduces sensitivity. In most studies, GHQ negative subjects were not further assessed; the

TABLE I
 Characteristics of GHQ when used on general hospital patients

Form of GHQ	Reference	Population	No. of cases screened	Criterion	No. of interviews	Threshold	Sensitivity: %	Specificity: %
GHQ-60	Goldberg (1970)	Gastrointestinal out-patients	91	CIS	91	11/12	80	93
	Maguire <i>et al</i> (1974)	Medical admissions	230	CIS	77	11/12	90	58
	Kirk & Saunders (1979)	New neurology out-patients	342	CA ¹	342	11/12	66	59
	Byrne (1984)	New gynaecology out-patients	211	PSE	35	26/27 4/5	40 67	85 86
GHQ-28	Rabins & Brooks (1981)	Multiple sclerosis out-patients	150	PSE	25	4/5	92	92
	Bridges & Goldberg (1984)	Neurology admissions	100	CIS	70	4/5	100	57
	Vazquez-Barquero <i>et al</i> (1985a)	Cardiology out-patients	194	CIS	194	11/12	80	80
	Mann (1977)	Hypertension screening	222	CIS	222	16/17 4/5	81 96 73	78 64 89
GHQ-30	Nott & Cutts (1982)	8-14/52 post-partum in community	200	CIS	200	4/5	92	66
	Feldman <i>et al</i> (1985)	Medical admissions	382	PSE	200	6/7 4/5	89 93	80 63
	Tyrer (1986)	Pain clinic	97	PSE	97	4/5	87	27

1 Clinical assessment

figures for false negatives depend only on review of medical notes and must be under-estimates.

We conclude that although the GHQ is the best available screening instrument for affective disorder, it is of limited value in the general hospital because it cannot by itself provide an estimate of prevalence until it has been validated for the particular medical population. Furthermore, it must be accepted that when used in a two-stage procedure there will be many false positives. In 'prescriptive screening' there is the additional problem that the sensitivity is too low to detect all those in need of further care (Skuse & Williams, 1984).

Other general screening tests. Goldberg (1985) has reviewed several other tests, some providing general scores and some subscores for specific symptoms. Only the Hospital Anxiety and Depression (HAD) scale has been derived from clinical experience to detect significant psychiatric disorder among medical patients (Zigmond & Snaith, 1983). Further research is required to determine the applicability of these tests.

Clinical Interview Schedule (CIS). This interview, developed for general practice research (Goldberg *et al.*, 1970), is sensitive to the psychiatric symptoms seen in general hospitals and acceptable to patients. Unfortunately there is no single accepted procedure for case definition, which can be based on an arbitrary total score, or on psychiatric judgement, or on a combination of both methods. It lacks the standardisation, computer program and comparability of the Present State Examination, but is more sensitive to less severe symptoms.

Present State Examination (PSE). World-wide use and standardisation are the major advantages of this instrument (Wing *et al.*, 1974), allowing comparison with general populations (Bebbington *et al.*, 1981; Henderson *et al.*, 1979) and with other psychiatric and medical groups. There are detailed rules for rating neurotic and psychotic symptoms, but no more than token questions for cognitive impairment and substance abuse. It can be a lengthy interview, requires skilled experienced interviewers, and does not allow rating of lesser degrees of emotional distress, since symptoms are only rated if 'clinically fairly severe'.

Some of the questions are awkwardly worded for use with the physically ill. Particular problems arise when rating 'understandable' distress apparently related to the physical disorder, since the instructions

state that 'appropriate' distress should not be rated. For general hospital use it is probably best to make explicit amendments to the PSE instructions. The CATEGO programme enables classification into diagnostic categories, and case definition is based on the total symptom score or, more satisfactorily, on the computed Index of Definitions grading of severity. The PSE case definition is more stringent than many other procedures. For example, in an Edinburgh community samples, Dean *et al.* (1983) found 8.7% of the sample were psychiatric cases with the PSE and 13.7% when Research Diagnostic Criteria (Spitzer, 1978) were used.

DSM-III. Although DSM-III was not designed to be an epidemiological instrument, it has been used in a number of general hospital surveys. However, the diagnostic rules are insufficiently precise for acceptable reliability. Furthermore, despite the general advantages of multiaxial classification, DSM-III categories have not proved entirely satisfactory for clinical use in the general hospital (Mackenzie *et al.*, 1983; Leigh *et al.*, 1982).

Screening tests for depression. Few of these procedures have been validated for use with the physically ill. The Beck Depression Inventory (BDI) has been the most widely used scale, and has proved acceptable in general hospitals but has not been standardised for this use (Beck *et al.*, 1961). It contains some somatic items, and this means that with the normal cut-off it identifies a rather high proportion of subjects as being mildly depressed (Moffic & Paykel, 1975; Clark *et al.*, 1983). The non-somatic items appear to discriminate well between depressed and non-depressed medical patients (Clark *et al.*, 1983). However, there are disadvantages in using the Beck and other mood scales to rate a single dimension of affect in populations in which undifferentiated emotional syndromes are more common than more specific forms of anxiety or depression.

There is no simple screening procedure suitable for routine clinical use by general hospital staff. However, there are several symptoms included in routine history taking which suggest possible affective disorder (e.g. lack of energy, insomnia, poor appetite, weight loss, memory disturbances, poor concentration, non-specific aches and pains, and bowel disturbances). Careful observation to detect emotional withdrawal and retardation or agitation, together with a few additional questions about mood, attitude to the future, interest in social and domestic life, libido, previous history of treatment

TABLE II
Studies of affective disorder in general hospital in-patients

<i>Reference</i>	<i>Population</i>	<i>No. of patients</i>	<i>Case definition</i>	<i>Cases: %</i>
Maguire <i>et al</i> (1974)	Medical admissions	230	GHQ-60, CIS, and casenote review	23
Bergman & Eastham (1974)	Admissions aged 65 or over to acute medical ward	100	Clinical assessment: functional psychosyndrome	19
Moffic & Paykel (1975)	Medical in-patients	150	BDI (13/14)	24
Knights & Folstein (1977)	Medical admissions	57	GHQ-30 (4/5)	46
De Paulo <i>et al</i> (1980)	Neurology admissions	126	GHQ-30 (4/5)	50
Cavanaugh (1983)	Medical in-patients	335	GHQ-30 (4/5)	61
			BDI (13/14)	32
Bridges & Goldberg (1984)	Neurology admissions	121	GHQ-28 and CIS	38.9
Johnston <i>et al</i> (1985)	Patients aged 65 or over	204	GHQ-28 and psychiatric assessment	13
Feldman <i>et al</i> (1986)	Medical admissions	453	GHQ-30 and PSE	14.6

TABLE III
Studies of affective disorder in referrals to general hospital out-patient clinics

<i>Reference</i>	<i>Population</i>	<i>No. of patients</i>	<i>Case definition</i>	<i>Cases: %</i>
Culpan & Davies (1960)	Medicine	100	Psychiatric interview and	51
	Surgery	100	Cornell Medical Index	51
Kenyon (1962)	Dermatology	100	Maudsley Personality Inventory and interview	14
Mayou (1975)	Venereal disease	100	CIS	20
Ballinger (1977)	Gynaecology (aged 40-45)	217	GHQ-60 (11/12)	52
Worsley <i>et al</i> (1977)	Gynaecology	97	GHQ-60 (11/12)	49
Kirk & Saunders (1979)	Neurology	342	GHQ-60 and interview	27
MacDonald & Bouchier (1980)	Gastroenterology and general medicine	100	GHQ-60 (9/10) and CIS	26
Hughes <i>et al</i> (1983)	Dermatology	196	GHQ-30 (4/5) and Wakefield (13/14)	30
				15
Byrne (1984)	Gynaecology	230	GHQ-60 (11/12) and PSE on sample	46
Vazquez-Barquero <i>et al</i> (1985b)	Cardiology	234	CIS	29
Tyrer <i>et al</i> (1986)	Pain clinic	97	PSE	45
				32

TABLE IV
Factors associated with affective disorder in general hospital patients

<i>Factor</i>	<i>Association and references</i>
Age	More common in younger patients ^{1,3}
Sex	More common in women. ^{1,4} No relationship ^{2,3}
Social class	Associated with lower social class in women ¹
Marital status	Associated with dissatisfaction with living alone ¹
Severity of physical illness	Associated with severe illness. ² Trend for association with severe, acute or chronic ¹
Absence of physical diagnosis	No clear relationship to physical diagnosis ^{1,2} Associated in out-patient series ⁵⁻⁸
History of psychological problems	Associated with previous psychiatric history, ^{1,2} psychotropic drugs and admission ^{1,8}
Social problems	Associated with social problems reported both by patients and by their general practitioner ¹ Associated with unemployment in men, problems in housing, finance, social life ¹

1. Feldman *et al* (1986); 2. Moffic & Paykel (1975); 3. Cavanaugh (1983); 4. Bridges & Goldberg (1984); 5. Mayou (1973); 6. Vazquez-Barquero *et al* (1985b); 7. Kirk & Saunders (1979); 8. MacDonald & Bouchier (1980)

for emotional problems and any major social difficulties, will allow detection of the majority of patients with significant mood disorders. Information from relatives, general practitioners and other informants and nursing observations is helpful.

Prevalence of affective disorder

Cavanaugh & Wettstein (1984) have reviewed the earlier literature and Table II and III summarise more recent reports. Surveys which used the GHQ alone report high figures, whereas those based on two-stage procedures report lower prevalences. Emotional disorder is mainly of moderate severity (Moffic & Paykel, 1975; Cavanaugh, 1983; Feldman *et al*, 1986), but both psychiatric disorder and sub-clinical distress are considerably more common than in the general population (Feldman *et al*, 1986). Some reports have considered anxiety and depression separately (see Cavanaugh, 1983) but we believe that, as in primary care (Goldberg & Huxley, 1980; WHO, 1983), the commonest pattern of affective disorder is an undifferentiated neurotic syndrome, which would be classified as adjustment disorder in DSM-III (Derogatis *et al*, 1983), and that only a small, but important, minority suffer from more specific psychiatric disorders. Moffic & Paykel (1975) compared the depressive symptoms of medical patients with those in psychiatric populations and found more pessimism and hopelessness but less

suicidal ideation. The limited evidence on associations with demographic, type of medical illness or other variables (Table IV) suggests that these are similar to the associations described for general populations.

Little is known of the prognosis of psychiatric disorder identified in the general hospital. Hawton (1981) found that psychiatric disturbance often persisted, especially in patients with a previous history of psychiatric disorder. The only study in which patients with psychiatric disorders were interviewed again after discharge found that most patients were improved at four month follow-up, but that a chronic course was associated with evidence of a previous history (Feldman *et al*, 1986). There is some evidence that psychiatric disorder is associated with poor prognosis for the medical condition (Querido, 1959; Moffic & Paykel, 1975; Hawton, 1981), although it is unclear whether this is because the psychiatric condition hinders recovery or because psychiatric disorder is associated with more severe conditions. The findings of Moffic & Paykel suggest the latter interpretation.

We conclude that although the methods used have often over-estimated the prevalence of affective disorder in the general hospital, disorders of moderate severity are considerably more common than in the general population, especially in younger women. They are often associated with evidence of long-standing psychological or social difficulties, but are

usually of good prognosis. These conclusions are supported by studies of patients with particular problems, e.g. myocardial infarction (Lloyd & Cawley, 1983), chronic intestinal disorders (Goldberg, 1970), admission for hysterectomy (Gath *et al*, 1982), chest pain (Bass & Wade, 1984) and cancer (Greer, 1985).

What are the clinical implications of these disorders? Some are distressing yet undiagnosed major psychiatric conditions, likely to respond to appropriate treatment. Some will severely complicate the management of physical illness. However, others (perhaps the majority) are transient states secondary to hospital admission or the physical condition, and may be expected to resolve soon after discharge. It remains uncertain which types of affective disorder benefit from recognition and treatment. When the condition appears severe, or where there are diagnostic or management difficulties, a psychiatric opinion can be helpful. The majority of patients probably require no more than support and simple help, although it is also sensible to inform the general practitioner so that he can reassess the psychological state.

Organic disorders

Standardised measures

There are no precise criteria for acute and chronic organic conditions, and it is difficult to distinguish mild impairment from normal functioning, especially in the elderly, the educationally and socially disadvantaged, and the distressed physically ill (WHO, 1983; Cooper & Bickel, 1984; Henderson & Huppert, 1984). There are three main approaches to screening for organic disorders: psychometric scales, brief clinical rating scales, and behavioural rating scales for use by nursing staff (see Cooper & Bickel, 1984; Henderson and Huppert, 1984).

Psychometric assessments, by means of tests such as the Weschler Adult Intelligence Scale (WAIS) (Weschler, 1955) or specific measures of memory, are useful in individual cases when close scrutiny is required in order to make a specific diagnosis. However, they are not suitable in general hospital surveys as they are time-consuming, require special skills to administer, and may prove unacceptable, especially to some cognitively intact subjects (Cooper & Bickel, 1984).

Mini-Mental State (MMS). This is the best known brief rating scale, consisting of thirty items which

assess orientation, registration, attention and calculation, recall, and language, and is based on answers to standard questions and performance of simple tasks (Folstein *et al*, 1975). It is brief, easy to use, and acceptable to most patients. Validity assessed against the WAIS appears to be good, but the ability of the MMS to detect mild dementia is unclear (Folstein *et al*, 1975). Using a psychiatrist's standardised clinical assessment, Anthony *et al* (1982) found reasonable sensitivity (87%) and specificity (82%) in the detection of cognitive impairment. False positives were more likely among poorly educated individuals and older subjects, so that while the specificity below the age of 60 was 92%, above 60 it was only 65%.

The MMS is a clinically useful instrument for the detection of poor cognitive function, but does not enable distinction between dementia, delirium, low intelligence and poor education. It is too lengthy for use with every patient.

Cognitive Assessment Schedule (CAS). This scale, a sub-scale of the Clifton Assessment Procedures for the Elderly (Pattie & Gilleard, 1979; McPherson *et al*, 1985), is similar to the MMS in that it includes specific orientation and information questions and performance items. It is brief, consisting of only 12 items, appears to have been satisfactory in general hospital surveys (Johnston *et al*, 1986; Feldman *et al*, 1986) and could easily be used in everyday clinical practice. When both the MMS and CAS were evaluated against psychiatrists judgements, the CAS was found to yield far fewer false positives (Johnston *et al*, 1986).

Standardised interviews. A number of standardised interviews have been developed to provide a more accurate and detailed psychiatric assessment, the best known being the Geriatric Mental State (Copeland *et al*, 1976) and the Comprehensive Assessment and Referral Evaluation (CARE) developed by Gurland *et al* (1977). Both are lengthy procedures and neither has yet been adequately assessed for reliability and validity in medical or other populations. The Geriatric Mental State is a standardised, semi-structured interview based on items from the PSE and the Psychiatric Status Schedule (Spitzer *et al*, 1970). There is a computerised diagnostic system (AGECAT).

In conclusion, several standardised measures are now available for the detection and assessment of organic disorders (Nelson *et al*, 1986). For large-scale surveys of general hospital patients either the MMS or the CAS can be recommended, although neither provides specific diagnostic information.

TABLE V
Studies of cognitive disorder in the general hospital

<i>Reference</i>	<i>Population</i>	<i>No. of patients</i>	<i>Case definition</i>	<i>Cases. %</i>
Bergman & Eastham (1974)	Admissions aged 65 or over to acute medical ward	100	Clinical assessment and memory tests	Dementia 7
Knights & Folstein (1977)	Medical ward admissions (mean age 55)	57	MMS (<23)	Delirium 10 33
De Paulo <i>et al</i> (1980)	Neurology admissions (mean age 50)	126	MMS (24)	30
Anthony <i>et al</i> (1982)	Medical ward admissions (29% aged >60)	97	MMS (<24) Clinical interview	33 Dementia 13 Delirium 9 Delirium + dementia 1
Cavanaugh (1983)	Medical in-patients survey (37% aged >65)	335	MMS (<23)	28
Bridges & Goldberg (1984)	Neurology admissions (mean age 45)	121	Clinical assessment and cognitive tests	2
Roca <i>et al</i> (1984)	Medical admissions (46% aged over 65)	380	MMS and psychiatric assessment	Dementia 15
Feldman <i>et al</i> (1986)	Medical admissions	453	CAS and review of notes	<70 years 2 >70 years 27

They require a second-stage method of assessment, preferably using an objective observer-rated method, or clinical assessment, in order to establish specific diagnoses and severity. In clinical practice, screening for organic disorders can be restricted to patients in the older age groups. Unless the routine history suggests difficulties, the CAS scale appears to provide a reasonably reliable method of screening, and can be recommended for this purpose. The MMS is valuable for more detailed assessment.

Prevalence of organic disorders

Table V summarises recent reports on the prevalence of organic disorders in general hospital in-patients, the majority of which have used a single screening instrument rather than a two-stage procedure. Unfortunately, most published reports have excluded a number of patients who were too ill for interview, many of whom were probably cognitively impaired (Feldman *et al*, 1986). Surveys using the MMS of patients of all ages admitted to general medical wards have yielded surprisingly consistent findings, with between a quarter and a third showing intellectual impairment. Few surveys have

examined the association of prevalence with age, but not surprisingly, organic mental disorders are especially common among older subjects (Bergman & Eastham, 1974; Feldman *et al*, 1986; Johnston *et al*, 1986). In neurology patients, abnormal MMS results are usually found in those with cerebral rather than peripheral disorders (De Paulo & Folstein, 1978).

In a study of 100 referrals to a consultation service, Cutting (1980) found it difficult to establish the specific cause of cerebral dysfunction in the majority of the 74 patients with acute organic psychiatric disorder. However, Hodkinson (1973), in a large multi-centre study of elderly patients, concluded that factors predisposing to delirium were dementia, defective hearing and vision, parkinsonism, and advanced age; precipitating factors were pneumonias, cardiac failure, urinary infection, carcinomatosis, and hypocalcaemia in particular, and severe illness in general.

There is little evidence concerning the prevalence in the general hospital of the various organic syndromes: delirium, dementia and delirium plus dementia. The importance of such distinctions was emphasised by Rabins & Folstein (1982), who found significantly higher death rates for delirious patients

compared with those with dementia, during hospital admission and at one year follow-up. Several accounts of referral to consultation-liaison services have also described the high mortality of those with delirium (Ruskin, 1985; Rabins *et al*, 1983; Popkin *et al*, 1984).

Recognition of organic mental disorders has major practical implications. Mistakes in the differential diagnosis of organic and affective disorder (Hoffman, 1982; Dubin *et al*, 1983) and misdiagnosis of dementia in the unimpaired elderly and the poorly educated (Roca *et al*, 1984) may result in inappropriate treatment and referral. The early identification of delirium is extremely important, because it may be life-threatening and require immediate treatment of the underlying physical disorder. Furthermore, delirious patients are often distressed and can be difficult to manage, yet appropriate psychiatric treatment is usually rapidly effective (Lipowski, 1983). Dementia has implications for continuing care. Johnston *et al* (1986) found that cognitively impaired patients stayed significantly longer in hospital than the unimpaired, an important reason being lack of suitable accommodation. Cognitive impairment limits compliance with medication, because of failure to remember dose schedules, especially when more than one drug is prescribed.

Alcohol

Screening procedures

The lack of any satisfactory classification of problem drinking and its complications (WHO, 1983, 1981) has limited study of epidemiology in the general hospital (McIntosh, 1982). Apart from detailed questioning about consumption (Midanik, 1982), there are two principal approaches: screening interviews and laboratory tests (Mayfield & Johnston, 1980; Holt *et al*, 1981; Bernadt *et al*, 1982). The first, and probably still the best known screening interview, is the Michigan Alcoholism Screening Test (MAST), which is available in 13 and 25 item versions (Selzer, 1971; Pokorny *et al*, 1972). A simple and attractive alternative is the four question CAGE (Ewing, 1984). These procedures have been shown to identify the majority of known alcoholics in a variety of psychiatric and other settings. However, specificity of screening is relatively low (Lancet, 1980; Bernadt *et al*, 1982; Holt *et al*, 1981), and none of the methods have been adequately validated in general hospital populations. There is no doubt that many patients who are known by physicians to have major alcohol problems are not detected, and also that screening questionnaires are not sensitive to early, and perhaps more treatable, alcohol problems.

Laboratory tests include blood alcohol, liver enzymes (aspartate aminotransferase; alanine aminotransferase, gamma glutamyl transpeptidase) and haematology (macrocytosis without anaemia). Individual tests and combinations can be helpful in identifying many patients who abuse alcohol (Lancet, 1980; Bernadt *et al*, 1982; Holt *et al*, 1981), although they are sometimes difficult to interpret and are insensitive to early drinking problems (Lloyd *et al*, 1982).

There are considerable advantages in using a procedure which combines screening interviews such as the CAGE and MAST with laboratory tests (Holt *et al*, 1981; Mayfield & Johnston, 1980). A promising research alternative is a detailed structured interview procedure covering the psychological, social and medical aspects of alcoholism, administered by a specially trained research worker (Lloyd *et al*, 1982; Chick *et al*, 1985).

Prevalence of alcohol problems

It is generally accepted that alcohol dependence and its complications are particularly common in medical populations. However, the evidence is confusing and unsatisfactory (McIntosh, 1982). There are considerable problems in interpreting published findings: the use of many different criteria for alcohol dependence; difficulties in defining alcohol-related disease, since all forms also occur in non-drinkers; failure to distinguish between active problems and those in remission; it is frequently unclear as to whether findings refer to alcoholism as the primary cause of the admission or as an entirely incidental finding; and few accounts state to what extent the findings in the hospital reflect particular characteristics of the local population.

Most surveys refer to general medical patients, and only a small proportion deal with surgical, orthopaedic, gynaecological or other medical groups (see McIntosh, 1982 and Table VI). In his very thorough review, McIntosh pessimistically concludes that: "It is still not possible to say, either precisely or even within what limits, what proportion of general hospital patients have alcohol-related disorders which are either causal or incidental to admission". It is probable that drinking problems are particularly common in emergency departments (Holt *et al*, 1980), accident and orthopaedic wards, and general medical units (Barrison *et al*, 1982), especially those specialising in gastroenterology.

Recognition of problem drinking is important both in immediate management and because of the greatly increased risk of future physical and mental ill-health, as well as social and domestic problems,

TABLE VI
Studies of alcohol problems in general hospital patients

Reference	Population	No of cases screened	Case identification	Problems: %
Barrison <i>et al</i> (1982)	Consecutive hospital admissions aged 18-65	520	Consumption and	16
		219	CAGE	23
Beresford <i>et al</i> (1982)	Consecutive acute injury admission to orthopaedic ward	87	CAGE and interview	14
Jariwalla <i>et al</i> (1979)	Admission to medical ward	545	Consumption and medical assessment	(alcohol related illness) 27
Holt <i>et al</i> (1980)	Evening attenders at an emergency department	702	Clinical assessment and blood alcohol	32 (blood alcohol > 80 mg/100 ml)
Lloyd <i>et al</i> (1982)	General medical admissions aged 18-65	275	Standard interview for current problems or previous treatment	Women 11 Men 27
Martin <i>et al</i> (1983)	Medical admissions	648	MAST, blood alcohol, medical assessment	20
Feldman <i>et al</i> (1986)	Medical admissions	453	Consumption, CAGE	Women 4 Men 18

in those who continue to abuse alcohol. While the results of treatment for alcoholism continue to be modest, sufficient numbers of individuals can be helped to make detection worthwhile. Intervention may be particularly effective in those whose alcohol consumption is excessive but not yet out of control (Skinner *et al*, 1981; Chick *et al*, 1985). In clinical care, there are no entirely satisfactory and simple means for detecting patients with problems in the use of alcohol. However, the four questions in the CAGE questionnaire, supplemented by simple questions about levels of alcohol consumption, awareness of the physical conditions commonly related to alcohol abuse (e.g. peptic ulcer, oesophagitis, head injury) and the blood test findings likely in alcoholics, will help in the detection of the majority of patients with serious drinking problems. It is sensible to concentrate on more intensive screening of high risk groups such as those attending emergency departments and liver clinics and those with predisposing occupations.

Other psychiatric problems

Several other types of psychiatric problems are more common among general hospital patients than in the general population.

Drug abuse

Although medical and psychological complications

of drug abuse are encountered in all parts of larger hospitals, studies in North America and Great Britain have concentrated on the prevalence of drug-related problems in accident and emergency departments. In a survey of seven London casualty departments (Ghodse, 1981) the rate of drug-related attendances in the casualty population was estimated to be 18.3/1000. They were predominantly young women, and at least 32% of individuals were dependent on drugs; 95% of attendances were for overdoses and 5% for reasons such as the complications of self-injection. Similar findings have been reported from the United States and Canada (Russe & Wells, 1980; Sellers *et al*, 1981; Maslansky, 1974). Drug dependence has been less well described in other medical populations, but narcotic and other drug dependence is particularly common in pain clinic attenders (Evans, 1981; Maruta *et al*, 1979).

Sleep disorders

There appears to have been no studies of sleep disorders in general hospital patients, but use of hypnotic drugs has received much attention. Reported rates of prescribing of hypnotics have varied from between less than a quarter of patients (Magni *et al*, 1984) to the majority (Perry & Wu, 1984). Prescriptions are usually on an 'as required' basis, with far fewer patients actually receiving them. Hypnotics are prescribed more often for the elderly

(Salzman & Van der Kolk, 1980; Mallings & Moon, 1983), for women, and for patients with relatively long admissions (Magni *et al*, 1984). Mallings & Moon reported greater use of hypnotics in medical than surgical patients, while Perry & Wu reported very much the reverse. Hypnotics are used particularly often in patients with malignant disease, those undergoing cardio-thoracic surgery (Mallings & Moon), and those recovering from surgery (Perry & Wu). Sleep disorders are likely to be associated with pain, and with anxiety and depression (Feldman *et al*, 1986), but noise and other environmental factors may have an important role (Berlin, 1984). Medication and drug withdrawal (especially alcohol) are also relevant.

A disturbing report from the USA by Perry & Wu (1984) suggested that the extent of hypnotic use provides little indication of sleep disorders in general hospital patients. None of the case-notes recorded the indications for hypnotics, and there was no correlation between nurses' notes of sleep patterns and prescription. They concluded that the "rationale for hypnotic agents in the general hospital is not clear".

The prevalence and causes of sleep disorders in general hospital patients, and the best means of managing them, is obviously an area for further investigation.

Sexual problems

The very common associations between sexual problems and medical and surgical disorders have been extensively reviewed elsewhere (Hawton, 1982; Bancroft, 1983). However, few systematic studies of specific conditions have been carried out, the main exception being diabetes. Approximately half of all men with diabetes will suffer erectile failure, due to either neuropathy of autonomic nerves or vascular disease, the proportion increasing rapidly with age (McCulloch *et al*, 1980; Goldstein *et al*, 1983). Sexual problems are also commonly associated with heart disease, renal failure, and peripheral neurological disorders. Numerous drugs used to treat medical conditions can have sexual side-effects, with anti-hypertensives being the worst culprits (Lancet, 1981). In contrast to the considerable information about the effects of physical illness and drug treatments on male sexuality, far less is known about their effects in women.

Although medical disorders and surgical procedures may cause sexual problems because of direct interference with anatomical or physiological mechanisms, psychological responses to illness are also important. Unfortunately, the latter have

undergone little systematic investigation, although numerous reports point to them as major factors.

Eating disorders

Similarly, there is virtually no information about the prevalence of eating disorders in general hospital patients. Although there have been some investigations of obesity and malnutrition, there is little indication of any associations with psychiatric morbidity. Obesity is common in medical patients, whether measured objectively (Abiin *et al*, 1982) or identified retrospectively from scrutiny of case-notes (Berger *et al*, 1977). Recent figures for the incidence have varied between 9% (Abiin *et al*) and 24% (Berger *et al*), although such variation is likely to be related to cultural factors, as well as methods of assessment. Malnutrition has been identified in at least one in five patients (Bistrian *et al*, 1976; Abiin *et al*, 1982), being particularly common in the elderly.

We are not aware of any studies of the prevalence of either anorexia nervosa or bulimia nervosa in general hospital patients, although they are described as reasons for referral to psychiatric consultation services. Case reports and surveys (Rodin *et al*, 1985) suggest that eating disorders may be especially common in diabetic clinics. Apart from severe weight loss and consequent malnutrition, anorexia nervosa may cause electrolyte disturbance, especially if associated with laxative abuse or vomiting. Similarly, there are important medical complications of bulimia nervosa, including acute gastric dilatation, oesophagitis, pancreatitis, and electrolyte disturbances (Harris, 1983).

Attempted suicide

Attempted suicide, especially deliberate self-poisoning, is the most common reason for acute medical admission in women, and in men it is second only to ischaemic heart disease (Hawton & Catalan, 1982). Following a rapid rise in such admissions during the late 1960s and early 1970s (Weissman, 1974; Wexler *et al*, 1978), the rates have recently levelled off and possibly declined, especially in women, although deliberate self-poisoning continues to be a major problem among young people (Hawton & Goldacre, 1982).

Widely varying prevalence figures of psychiatric disorders among attempted suicide patients have been reported, reflecting differences in diagnostic criteria. Two studies in which the PSE was used have helped to clarify the picture. Although psychiatric symptoms during the month preceding attempts are found in the majority of patients, Newson-Smith &

TABLE VII
Absence of physical diagnosis in new referrals to out-patient clinics

	Setting	No. of patients	% without physical diagnosis
Culpan & Davies (1960)	Medicine	100	38
	Surgery	100	5
Mayou (1975)	Venereal disease	100	34
Kirk & Sanders (1979)	Neurology	342	27
MacDonald & Bouchier (1980)	Gastroenterology	100	45
Hughes <i>et al</i> (1983)	Dermatology	100	1
Pearson (1985)	Allergy clinic	250	13

Hirsch (1979) identified 'definite' psychiatric disorder in only 31% using the PSE, with a further 29% being in the borderline or 'threshold' category. Almost all were suffering from depression. Similar findings were reported by Urwin & Gibbons (1979), although they also identified patients with alcoholism, anxiety states and schizophrenia. Other studies have found alcoholism to be particularly common in male attempters (Morgan *et al*, 1975; Holding *et al*, 1977).

There is a rapid decline in the prevalence of psychiatric disorder during the first few months after attempts (Newson-Smith & Hirsch, 1979), although it is unlikely that there is a significant change in alcohol problems.

Psychiatric disorder presenting with physical symptoms

Somatic symptoms without significant physical cause are regularly seen in general hospital wards, and are especially common in many out-patient clinics (Table VII) and in emergency departments. Psychiatric accounts of hypochondriasis are based on highly selected clinical experience, and there is no adequate classification (Barsky & Klerman, 1983; Lloyd, 1983). Atypical abdominal pain (Gomez & Dally, 1977; Drossman, 1982), chest pain (Mayou, 1973; Bass & Wade, 1984) and pain clinic attenders (Tyrer *et al*, 1986) have been well described.

Most patients are easily reassured, but a minority suffer persistent symptoms with psychiatric and social disability. Descriptions of referrals to psychiatric services (Katon *et al*, 1984; Slavney & Teitelbaum, 1985) show that most are suffering from primary affective disorder, although it is not uncommon for the more characteristic psychological symptoms to be 'masked'. Others suffer from what are classified in DSM-III (American Psychiatric Association, 1980) as somatoform, dissociative and

factitious disorders, and in a further small group there is no convincing psychological or social explanation. It is important to be aware that a proportion of such patients are eventually diagnosed as suffering from major medical illness.

Hysteria

Hysterical symptoms are undoubtedly common, though usually referred to as 'functional overlay'. The main evidence for the prevalence of syndromes of hysteria (conversion, dissociative states and Briquet's syndrome) comes from surveys of psychiatric consultations (see Ziegler *et al*, 1960; McKegney, 1967; Cavanaugh & Wettstein, 1984; Folks *et al*, 1984) in which up to 13% of patients referred to consultation services were diagnosed as having hysteria. Trimble (1981) reported that 1% of admissions to a specialist neurological hospital are given the diagnosis of hysteria.

Factitious disorder

The dramatic clinical picture of the Munchausen syndrome is uncommon. Blackwell (1968) identified ten cases in a London teaching hospital in one year. Patients with other forms of artefactual or factitious disorder are more common, and are more willing to accept psychiatric assessment and treatment (Carney & Brown, 1983; Reich & Gottfried, 1983; Bayliss, 1984). Reich and Gottfried described 41 cases, of whom 39 were women. Most had experience of occupations related to medicine, and there were four main clinical groups: self-induced infections, simulated specific illnesses with no actual disorder, chronic wounds, and self-medication. Reports of other patients with specific artefactual symptoms, such as dermatitis artefacta (Sneddon, 1983), pyrexia of unknown origin (Aduan *et al*, 1979), bruising

disorders (Ratnoff, 1980) and diabetes (Schade *et al*, 1985) suggest that factitious disorder is much more common than is normally recognised (see Bayliss, 1984).

Recognition of psychiatric disorder by medical staff

It has frequently been observed that hospital doctors and nurses seriously underestimate and misdiagnose all forms of psychiatric disorder (Table VIII). Although we do not doubt the general conclusion, there are serious flaws in some of the evidence. Most reports assume that the other screening instruments provide a satisfactory criterion for the 'true' prevalence of psychiatric disorder, but we have already seen that the GHQ has a low specificity in the general hospital and substantially over-estimates the prevalence. On the other hand, it is unreasonable to assume that case notes are a reliable guide to physicians' assessment.

The diagnosis of affective disorder is often difficult in the presence of physical illness. Goldberg & Blackwell (1970) found this was the main cause of 'hidden psychiatric morbidity' in a general practice study in which the general practitioner was an

experienced and qualified psychiatrist. Hospital doctors are no more likely to diagnose severe problems than mild ones, and when they do recognise psychiatric disorder they are unlikely to code it in case summaries (Feldman *et al*, 1985). It is expected that systematic enquiries about the non-somatic features of anxiety and depression would enable much higher rates of detection.

Cognitive disorder is often obvious but may be misdiagnosed as affective disorder (Hoffman *et al*, 1982; Dubin *et al*, 1983) or missed altogether. Roca *et al* (1984) compared the judgements of medical interns with psychiatrists' standardised diagnosis of dementia and found a sensitivity of 79% and specificity of 80%. The interns were just as likely to mis-diagnose dementia in unimpaired subjects as to fail to make a correct diagnosis. Use of systematic screening tests (Goldberg, 1985) and proper attention to age, educational status and evidence of previous function could be expected to improve recognition.

Surveys of alcohol problems have commented that doctors often fail to record the clinical history (Barrison *et al*, 1980) and that they underestimate both problems and consumption (Barrison *et al*, 1980; Lloyd *et al*, 1982). Westermeyer *et al* (1978) also

TABLE VIII
Recognition of psychiatric disorder by medical and nursing staff

<i>Reference</i>	<i>Case definition</i>	<i>Hospital staff assessment</i>	<i>Undetected. %</i>
<i>Affective disorders</i>			
Maguire <i>et al</i> (1974)	GHQ-30 + SPI	Medical notes	49
Knights & Folstein	GHQ-30 (4/5)	Interview with doctors and nurses	Doctors 35 Nurses 70
Brody (1980)	GHQ-30 (4/5)	General medical clinic, regular attenders	34
Lucas & Folstein (1980)	GHQ-30 (4/5)	Nursing assessment	41
Cavanaugh (1983)	GHQ-30 (C/5)	Interview with residents	47
Bridges & Goldberg (1983)	GHQ-28 and SPI	Neurology casenotes	72
Feldman <i>et al</i> (1985)	GHQ-30 and PSE	6-point rating scale	56
<i>Cognitive disorders</i>			
Knights & Folstein (1977)	MMS (<23)	Interview with doctors and nurses	Doctors 37 Nurses 55
De Paulo & Folstein (1978)	MMS (<24)	Rating by doctors	30
Lucas & Folstein (1980)	MMS (<25)	Nursing assessment	43
Cavanaugh (1983)	MMS (<24)	Interview with residents	63
Roca <i>et al</i> (1984)	MMS (<24) and psychiatric assessment for dementia	Rating by doctors	20

noted that physicians who were aware of problem drinking did not regard this as a medical concern, and did not see themselves as having responsibility for initiating treatment. Effective detection of alcohol problems requires a high degree of suspicion of high risk groups and the use of a combination of screening measures (Skinner *et al*, 1981).

Psychiatric disorder and medical settings

There are, undoubtedly, wide differences in the prevalence of psychiatric disorder in the many types of in-patient and out-patient units within the general hospital, but there have been few systematic studies and most epidemiological evidence derives from acute medical units. It is important that areas with a particularly high prevalence of psychiatric disorder be detected, since these must be the priorities for the provision of liaison psychiatry and other psychiatric and social services.

In-patients

Acute and chronic organic disorders are especially common in patients aged over 65 years (Bergman & Eastham, 1974; Lipowski, 1983), although they are much less commonly referred for psychiatric opinions than younger medical patients (Rabins *et al*, 1983; Popkin *et al*, 1984). Emotional disorder is more frequent in younger patients (Feldman *et al*, 1986) and is usually either of long standing or a reaction to acute illness and hospital admission. A small proportion of in-patients present with somatic symptoms due to primary psychiatric disorder.

Little is known of prevalence in other than medical and neurology wards. Psychiatric morbidity is probably particularly high in accident wards and in those specialising in the care of severe illnesses, often involving distressing treatment, for example cancer units (Derogatis *et al*, 1983; Bukberg *et al*, 1984), intensive care, radiotherapy (Forester & Kornfeld, 1978) and spinal injury wards.

Out-patients

This review does not cover hospital-based primary care as practised in North America and elsewhere (see Cavanaugh & Wettstein, 1984) and refers only to secondary care as is usual in Britain (Table II). In an early study, Culpan & Davis (1960) found considerable morbidity in patients attending general medical clinics and rather less in surgical out-patients. Short-term prognosis was relatively good, but 18% of medical patients and 5% of surgical patients were thought to require more psychiatric care than was

possible in a general out-patient clinic. Particularly high prevalence of psychological and social problems has been described in some specialist out-patient clinics, including cardiac out-patients (Vazquez-Barquero *et al*, 1985a,b), pain clinics (Tyner *et al*, 1985; Merskey & Spear, 1967), and clinics caring for severe chronic disorders such as renal failure (Czackes & De Nour, 1978), rheumatoid arthritis (Rogers *et al*, 1982) and diabetes (Tattersall, 1981).

We have already seen that patients without a significant physical cause of somatic symptoms are common in many out-patient clinics and that some of these patients may suffer from primary psychiatric disorder. These disorders are often easily treated, and it is unfortunate that they are frequently unrecognised and cause chronic disability. Research on specific patient groups suggest that current and previous psychiatric state are good predictors of psychosocial and possibly medical outcome in these patients.

Emergency departments

Affective and organic psychiatric disorder, substance abuse and other psychiatric problems are common in emergency departments, both as presenting problems and as incidental findings in those with physical diagnoses. Almost all detailed surveys have been restricted to the small minority of attenders seen by psychiatrists, or recorded by the consulting doctor as having psychiatric disorder.

In Britain, casualty doctors see all attenders and refer a proportion of those to duty psychiatrists (about 2% of all attenders). Bassuk *et al* (1983) reported that casualty doctors at the Bristol Royal Infirmary made a principal diagnosis of overt psychiatric disorder in 0.98% of attenders, of self-injury in 2.5%, and of physical symptoms with no specific physical cause in 2.5%. They made no comment on psychiatric disorder in those who had physical problems. In North America, preliminary assessment by nursing staff results in 3–5% of attenders being seen by the psychiatric emergency clinic. American reviewers have noted that over the past 10–20 years emergency departments have taken on increasing responsibilities for the management of chronic mental illness (Gerson & Bassuk, 1980; Bristol *et al*, 1981; Jones & Yoda, 1982). The nature of the psychiatric morbidity depends greatly on the area served (Jacobsen & Howell, 1978).

The only survey of consecutive emergency department attenders is that of Summers *et al* (1979), who assessed samples of 100 daytime attenders and 100 night-time attenders in an Ohio general hospital. Psychiatric disorder was identified in 26% of the

former and 65% of the latter. The most frequent diagnoses were alcohol problems and anti-social personality (29% and 17% respectively at night), with Briquet's syndrome, drug and alcohol dependence all being common. Salkovskis *et al* (1986) surveyed 149 accident and emergency department attenders in Leeds, having excluded deliberate self-harm, overt psychiatric disorder and the severely ill, and found that 36% scored above threshold on the GHQ or HAD scales.

Concluding comments

There is ample evidence that psychiatric morbidity is common in the general hospital. There are also hospital patients who do not have major psychological symptoms but who present management difficulties or are distressed by social problems, and who may benefit from psychological or social

intervention. There is a clear need to develop both a better classification of psychiatric disorder in the general hospital and appropriate research measures so that prevalence, nature and outcome of the various forms of disorder can be accurately described. Such basic epidemiological research should be accompanied by the evaluation of treatments leading to provision of better care for those who need it.

Sophisticated research measures are unlikely to be useful to busy general hospital staff. We have made suggestions about how they can detect the majority of patients who have significant psychiatric disorder. In particular, there is a need for greater awareness of the disorders that we have reviewed, together with systematic history-taking which has both psychiatric as well as physical diagnoses in mind. We believe that general hospital staff can detect their patients' more serious psychiatric problems and provide simple care without excessive extra demands on their time.

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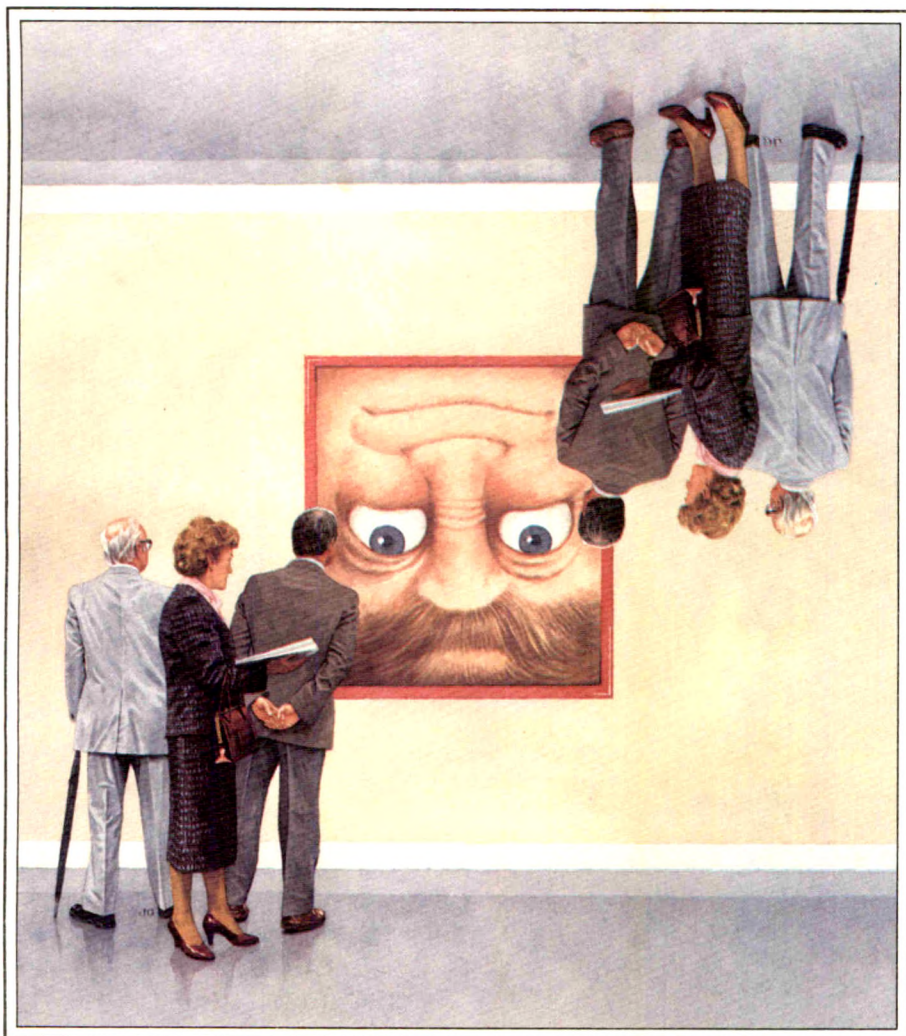
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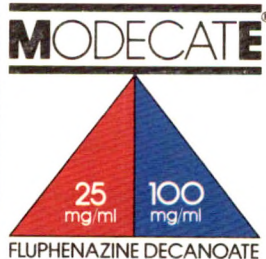
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Conditioning and Sensitisation in the Longitudinal Course of Affective Illness

ROBERT M. POST, DAVID R. RUBINOW and JAMES C. BALLENGER

Few biological theories of manic-depressive illness have focused on the longitudinal course of affective dysfunction and the mechanisms underlying its often recurrent and progressive course. The authors discuss two models for the development of progressive behavioural dysfunction—behavioural sensitisation and electrophysiological kindling—as they provide clues to important clinical and biological variables relevant to sensitisation in affective illness. The role of environmental context and conditioning in mediating behavioural and biochemical aspects of this sensitisation is emphasised. The sensitisation models provide a conceptual approach to previously inexplicable clinical phenomena in the longitudinal course of affective illness and may provide a bridge between psycho-analytic/psychosocial and neurobiological formulations of manic-depressive illness.

Many of the current biological theories of manic-depressive illness have focused on the acute changes in affective states and on their possible underlying biological determinants. There has been less attention paid to the mechanisms underlying the longitudinal course of affective illness. We examined two different sensitisation models, behavioural and electrophysiological, which might help to conceptualise the progression of symptoms in recurrent affective illness. However, we do not propose that these models fulfil all the usual requirements for an animal model of mania or depression *per se* (McKinney *et al*, 1984).

Several aspects of the course of affective illness deserve further emphasis. First, both bipolar and unipolar affective illness tend to be recurrent in the majority of patients (Perris, 1966; Angst, 1978; Kraepelin, 1921; Grof *et al*, 1974; Zis & Goodwin, 1979). Secondly, the frequency of recurrence and rapid cycling increases not only with age, but as a function of the number of prior episodes (Kraepelin, 1921, Grof *et al*, 1974; Zis & Goodwin, 1979; Cutler & Post, 1982). Thirdly, similar symptoms tend to be reproduced in each episode with new ones added in sequential or stepwise fashion. Affective episodes later in the course of the illness may be more severe and have more precipitous onsets (Post *et al*, 1981a) than earlier episodes.

We hypothesise that a subgroup of patients becomes more vulnerable, or 'sensitized', to recurrences of affective episodes. We suggest that these patients show characteristics of a sensitisation or kindling-like process in which the biochemical and physiological processes involved in the illness

become progressively more easily triggered by the same circumstances or precipitants. Eventually, the episodes may occur spontaneously. Conditioned behavioural and biochemical changes might also occur, as demonstrated in laboratory studies. The models account for several phenomena observed in the course of affective illness; they suggest new avenues of research and stress the importance of preventing episodes with prophylactic treatment to inhibit sensitisation.

While there is still debate about the aetiological role of psychosocial stress in the onset of affective episodes, there is substantial evidence that for some patients environmental precipitants are associated with the onset of illness (Paykel, 1979a, b; Dunner & Hall, 1980; Brown *et al*, 1975; Amelas, 1979; Lloyd, 1980). Behavioural sensitisation could provide a model for the large subgroup of patients who develop more severe and rapid recurrences, in apparent response to repeated stresses of equal or reduced magnitude. Moreover, as patients develop increasing rapidity of cycling, the illness appears to evolve with its own rhythmicity and spontaneity, independent of ongoing life events. The mechanisms underlying such recurrent illness may be similar to kindling and behavioural sensitisation, as described below. Some patients have only a single affective episode. Genetic and environmental influences may be important in determining this pattern of non-recurrent illness; we will not address this subgroup further.

Kindling

Electrical kindling, as originally described by

Goddard *et al* (1969), refers to the development of major motor seizures in response to repeated intermittent electrical stimulation of the brain with insufficient current to produce overt behavioural effects (Racine, 1978). Recent data suggest that repeated application of some pharmacological agents, either parenterally or directly into the brain, may also produce a progressive increase in neural excitability, similar to kindling, eventually producing major motor seizures in response to a previously subthreshold dose of drug (Post & Kopanda, 1976; Post *et al*, 1982).

The intermittency of the stimulation appears to be critical for the development of amygdala-kindled seizures (Goddard *et al*, 1969; Racine, 1978; Post, 1980). Continuous amygdala stimulation or stimulation every 2–5 min is not associated with kindling; rather, the animal habituates and does not show major motor seizures. In contrast, intermittent stimulation at intervals longer than once every 2 hours (optimally, once every 24 hours or even once a week) is associated with the relatively rapid development of a convulsive response to the original subthreshold kindling stimulation. This temporal pattern of intermittent stimulation in the development of either tolerance or sensitisation may have parallel implications for the development of pathological behaviour in response to other intermittent stimuli, including stress (Post, 1980).

Kindling is a long-lasting, possibly permanent, change in neural excitability. Animals kindled in youth retain convulsive susceptibility in adulthood. As one ascends the phylogenetic scale, particularly in rhesus and human primates, kindling to a convulsive end-point appears to be increasingly difficult to achieve. Therefore, kindled seizures are only one marker of the development of the sensitisation process; many of the important characteristics of the kindling paradigm, including change in threshold and associated changes in behaviour (Adamec, 1975; Post, 1981; Post *et al*, 1984), can be observed in the absence of seizures.

After many repetitions of kindled seizures an animal may exhibit 'spontaneity', i.e. develop seizures in the absence of external stimulation (Pinel, 1981; Wada *et al*, 1974). The phenomenon of spontaneity has obvious importance for establishing kindling as a model of epilepsy. We suggest that kindling may help us understand how stress-induced mood alterations may become so sensitized that they also occur spontaneously; i.e. the rapidly or continuously cycling pattern of manic-depressive illness. It is pertinent that approximately one-third of kindled animals show cycling in their seizure patterns (Post, 1981).

Behavioural sensitisation

Repeated, intermittent application of psychomotor stimulants and dopaminergic agonists produces an increasing behavioural responsivity to selected end-points, such as motor hyperactivity or stereotypes. When sensitisation occurs, the behavioural changes develop with faster onset, increased magnitude and longer duration (Martres *et al*, 1977; Shuster *et al*, 1977; Segal & Mandell, 1974; Kilbey & Ellinwood, 1977). While behavioural sensitisation and electrophysiological kindling show similar progressive time courses, the mechanisms underlying these two phenomena appear to involve different neurotransmitter pathways (Post *et al*, 1982, 1985). The similarities and differences between kindling and behavioural sensitisation may help us understand the mechanisms underlying the progressive phenomenology of affective illness.

Conditioning appears to play a role in the development of behavioural sensitisation. For example, environmental cues associated with repeated cocaine exposure are important determinants of the degree of response to cocaine (Post *et al*, 1981b; 1985). Hinson and Poulos (1981) also found that the increased behavioural response to cocaine could be desensitized with saline injections. Behavioural sensitisation is modulated by peptide hormones, is more robust in females compared to males, shows cross-sensitisation to stress, and may involve a dopaminergic substrate (Post *et al*, 1984). A dopaminergic mechanism in behavioural sensitisation and its potential cross-sensitisation to stress (Antelman *et al*, 1980; Antelman & Eichler, 1979) suggest a link to the dopaminergic mechanisms in affective illness (Post *et al*, 1978; Jimerson & Post, 1984). The findings of Pickar *et al* (1984) also suggest that our model of drug-induced behavioural sensitisation may be extended to include the development of manic syndromes in man. Depressed patients who became manic during their first course of treatment with a monoamine oxidase inhibitor (MAOI) consistently became manic earlier in the course of a second treatment with an MAOI than on the first occasion. We postulate that a similar sensitisation may occur in non-drug-induced episodes.

Stresses of particular type, intensity, and intermittency may produce sensitisation in a fashion similar to the behavioural sensitisation described above. Sklar & Harris (1985) reported that intermittent or temporary loss, rather than continuous loss of a parent, was associated with greater depressive and other types of psychopathology in subjects who came from large families. Repeated exposure to

inescapable shock has been discussed as a model of stress-induced analgesia, 'learned helplessness', or depression (Anisman, 1984; Seligman, 1975; Drugan *et al*, 1981). The quality, intensity, periodicity, and degree of control, as well as environmental context and conditioning, which are important variables for behavioural sensitisation, also appear important to the behavioural and biochemical consequences of this stress model.

Conditioned biochemical changes

There is a growing belief in the literature that drug-induced and environmental stress-induced changes in brain biochemistry may be conditioned (Deutch *et al*, 1985; Post *et al*, 1984; Schiff *et al*, 1981; Cassens *et al*, 1981; Perez-Cruet, 1976; Anisman & Sklar, 1979; Herman *et al*, 1982). For example, Schiff *et al* (1981) have demonstrated that amphetamine-induced alterations in homovanillic acid (HVA) could be conditioned. They observed that the significant increases in mesolimbic HVA areas following amphetamine could be conditioned to a loud tone that was originally paired with amphetamine administration.

Herman *et al* (1982) have observed conditioned biochemical effects on re-exposure to stimuli previously associated with inescapable shock. Rats given inescapable shock showed increases in plasma corticosterone and brain dihydroxyphenylacetic acid (DOPAC) in frontal cortex, olfactory tubercle, nucleus accumbens, and amygdala. Re-exposure of

rats to the environment where they had been previously shocked 24 hours earlier induced elevations in plasma corticosterone and brain DOPAC in the frontal cortex only. Deutch *et al* (1985) reported parallel conditioned changes in DOPAC in prefrontal cortex and in the ventral tegmental (A 10) cell body area. Cassens *et al* (1981) also demonstrated conditioned biochemical responses in a model of learned helplessness. They demonstrated that the 3-methoxy-4-hydroxyphenylglycol (MHPG) increases, which were ordinarily elicited during the learned helplessness induction procedure, could be associated merely with the placement of the animal in that apparatus. In this regard, the selective changes in brain MHPG in animals with and without control of the environmental stress deserve particular comment.

Control over shock and environmental contingencies determines whether 'helplessness' behaviour emerges (Jackson *et al*, 1979) and the direction of the biochemical changes involved. Miller & Weiss (1969) have demonstrated increases in MHPG in the brainstem of animals subject to shock which they could terminate themselves; however, decreases in MHPG, the opposite effect, occurred in the brainstem of yoked-control animals that received, but were unable to terminate, the same shock. Vogel (1983) has recently replicated these findings measuring plasma norepinephrine, rather than brain MHPG. The behavioural effects of undemonstrable shock have also been linked to α_2 mechanisms in the locus coeruleus (Weiss & Simson, 1984) and to beta-receptor increases in the hippocampus (Henn, 1984).

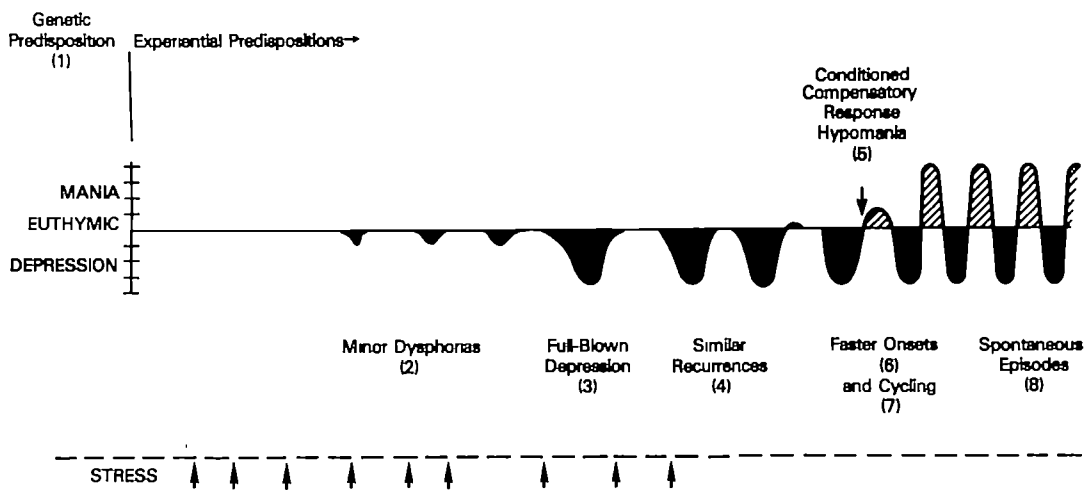


FIG. 1. Schematic illustration of the longitudinal and progressive development of recurrent affective illness.

TABLE I
Parallels in the phenomenology of affective illness to kindling and sensitisation

<i>Characteristic of affective illness*</i>	<i>Characteristic of</i>	
	<i>Kindling</i>	<i>Sensitisation</i>
1. Genetic component	+	+
2. Early experience predisposes	+	+
3. Mild alterations emerge as full blown episodes (threshold effects)	+	+
4. Episodes reach plateau and are similar in content and behaviour over repeated recurrences	+	+
5. Repeated episodes of mania or depression may lead to emergence of opposite phase (conditioned compensatory reactions)	±	+
6. Onset of maximum disturbance occurs earlier in episode with repetitions	+	+
7. Vulnerability to recurrences and faster cycling	+	+
8. Early episodes may be precipitated, later appear "spontaneously"	+	
9. Lithium carbonate effective prophylaxis	-	+
10. Carbamazepine effective prophylaxis	±	-

+ = Observed; ± = Equivocal; - = Not observed.

*Numbers 1-8 also refer to those in Fig 1.

Conditioned emotional responses: possible clinical relevance

The possible contributing role of environmental events, psychosocial stress and loss in the precipitation of affective illness has been discussed extensively elsewhere (Paykel, 1979a,b; Dunner & Hall, 1980; Brown *et al*, 1975; Amelas, 1979; Lloyd, 1980). The sensitisation-conditioning suggests that symbolic components of previous triggers of a depressive response might be learned, or conditioned, such that they become capable of eliciting depression in the absence of the unconditioned stimulus; i.e. a 'real' stress or loss.

We are postulating that anticipated stresses or imagined losses, if sufficiently conditioned, may eventually be capable of producing the behavioural, physiological and biochemical alterations usually associated with an affective episode. Should this concept prove to be valid, it might provide a transitional bridge between psychoanalytical concepts of the aetiological role for real or symbolic losses, and biological-endogenous views of depression.

The kindling and sensitisation models for the way in which repetition of early loss experiences might produce overt psychopathology are schematised in Figure 1 and described in Table I. Both biological and social components could occur separately, but

our framework also considers how stress- or loss-induced reactions could evolve over time, become increasingly facilitated and autonomous, and eventually show a pattern of spontaneity. Both 'reactive' and 'endogenous' (spontaneous) episodes could occur at different times in the same patient. The model might also be relevant when repeated endogenous or exogenous events, such as repeated episodes of premenstrual dysphoria or drug-induced mania, could lead to a sensitized substrate that might then be triggered more easily by subsequent biological or psychosocial stressors. Thus, the biological and psychosocial approaches might not be mutually exclusive, but potentially interactive. This model is consistent with the data of Paykel (1979a), which show that a clear separation of reactive and endogenous depression is often not possible, even after a careful history of stress and life events.

A testable derivative of our schema would be that more clear identification of psychosocial precipitants would be possible earlier, rather than later, in the course of recurrent affective illness. Support for this concept is found in the observations of Amelas (1979), who noted that a high percentage of first manic episodes were preceded by stress. A corollary proposition would be that symbolic losses might be sufficient to trigger affective episodes later in the course of illness.

Mechanisms inherent in conditioning may also offer a conceptual approach to the emergence of apparently opposite behavioural states of depression and mania following similar precipitants (Dunner & Hall, 1980; Amelas, 1979; Cohen *et al.*, 1954; Aleksandrowicz, 1980). Siegel (1979) has reviewed the evidence that behavioural, physiological and biochemical responses opposite to those initially induced might at times appear during conditioning; i.e. conditioned compensatory responses. For example, he suggested that "subjects with a history of morphine administration display morphine compensatory conditioned responses when confronted with the usual administration procedure, but without the drug". We suggest that opposite mood responses could also develop as conditioned compensatory responses following repeated episodes of affective illness. Analogous to Siegel's formulation, we suggest that subjects with a history of depressive responses may display depression-compensatory responses (i.e. mania) when confronted with cues that would ordinarily elicit a depressive reaction, but when the timing, context, or biological substrate is altered.

Within the psychoanalytic framework, theorists have postulated that manic responses may emerge as a defence against depression (Cohen *et al.*, 1954; Aleksandrowicz, 1980; Freud, 1917). Our formulation extends this analytic framework to include the possibility that psychologically- and biologically-triggered compensatory mechanisms may also be involved in this process. Thus, we suggest that the repeated experience of depressive episodes might progressively bring into play biological compensatory mechanisms that not only help terminate the episode, but result in a compensatory manic episode. This process could provide a framework for considering a mechanism by which a large group of apparent unipolar depressed patients experience multiple recurrent depressive episodes prior to the emergence of their first manic episode (Angst & Grof, 1976). In patients with manias at the onset of their illness, compensatory processes might similarly lead to the development of depressive episodes.

One of the primary characteristics of the conditioned response is that, with time, the behavioural response begins to occur earlier in the interval and may even anticipate the conditioned stimulus (Siegel, 1979; Dollard & Miller, 1950). These findings are consistent with the more rapid onset of motor hyperactivity in behavioural sensitisation (Martres *et al.*, 1977; Shuster *et al.*, 1977; Segal & Mandell, 1974; Kilbey & Ellinwood, 1977) and the observation of the more rapid onset of manic episodes in patients with increased numbers of prior

episodes (Post *et al.*, 1981a). With sufficient repetition, compensatory responses might move progressively earlier, so that they occurred within, rather than following, an episode. Such a formulation could provide a mechanism for the evolution of dysphoric manias and activated depressions in bipolar patients. If this explanation is well founded, one predicts that these mixed states would tend to occur later in the course of a patient's affective illness after many recurrences.

Bunney (personal communication) has noted an increased incidence of mania in recovered patients in the period immediately before discharge from the hospital. It is interesting to consider that this occurs during a phase of anticipated psychosocial loss, but occurs in the absence of the unconditioned biochemical stimuli (i.e. the associated depressive substrate has been treated) and thus may be sufficient to trigger the compensatory behavioural response of mania rather than depression, just as Siegel (1979) has postulated the triggering of compensatory responses in the absence of the unconditioned stimuli during the opiate response.

The environmental context-dependency of behavioural sensitisation to cocaine and related drugs may provide another explanation for opposite behavioural effects to the same stimulus. Collins *et al.* (1979) have shown that cocaine may increase or decrease the response, depending on the associative cues. Similarly, Kopa *et al.* (1968) and Belenkov & Shalkovskaya (1980) have shown opposing behavioural responses to the same electrical stimulation of the same brain area (in the thalamus, hypothalamus or amygdala) depending on whether the animal received the stimulation when placed where it had, or had not, previously received a foot shock. Thus, it is possible that similar biochemical changes may be associated with opposing behavioural responses, depending on prior associative contexts and conditional cues.

Similarly, responsivity might be differentially affected by changes in behavioural state following pharmacological manipulations, or the endogenous changes in biochemistry that accompany affective states. Internal biological states can acquire discriminative, or cue, value and become associated with various pathological behaviours. For example, it is possible that an associative link could be established between high levels of circulating glucocorticoids and depressive affect, offering a potential explanation for early morning exacerbation of depression in conjunction with increases in glucocorticoids. The specific relationship between a pathological mood state and a given set of biological variables could thus vary depending on the

association developed through conditioning. Additionally, utilizing the concept of compensatory conditioned responses, one might suggest a mechanism for 48-h cycling. A given phase of a circadian rhythm (e.g. in glucocorticoids, classical or peptide neurotransmitters) might be associated with the switch into depression, while the occurrence of a similar change the next day might evoke the opposing manic response.

These 'theoretical vignettes' are highly condensed and are presented to stimulate discussion about possible biochemical and conditioning mechanisms underlying some clinical phenomena in affective illness. Schou (1982), Dunner and Hall (1980), and Dunner *et al* (1976) have noted that adequate lithium prophylaxis may develop gradually over a period of months in some patients. Schou does not believe this delay in onset is related to pharmacokinetic considerations or the very late onset of

biochemical changes, but to psychological processes in the patient and his family. Based on the conditioning principles discussed above, an alternative formulation could be offered. Time may be required to decondition recurrent or cyclic affective disturbances. Only after repeated experiences of psychological or biological changes, which would previously have been associated with an affective episode, but which now occur in the new substrate of lithium-induced biochemical alterations (i.e. in the absence of the unconditioned stimulus), do affective recurrences begin to subside.

Potential implications for pharmacological and psychotherapeutic treatments

The conditioning and sensitisation models described above would appear to have implications not only for possible mechanisms of action of psychotropic

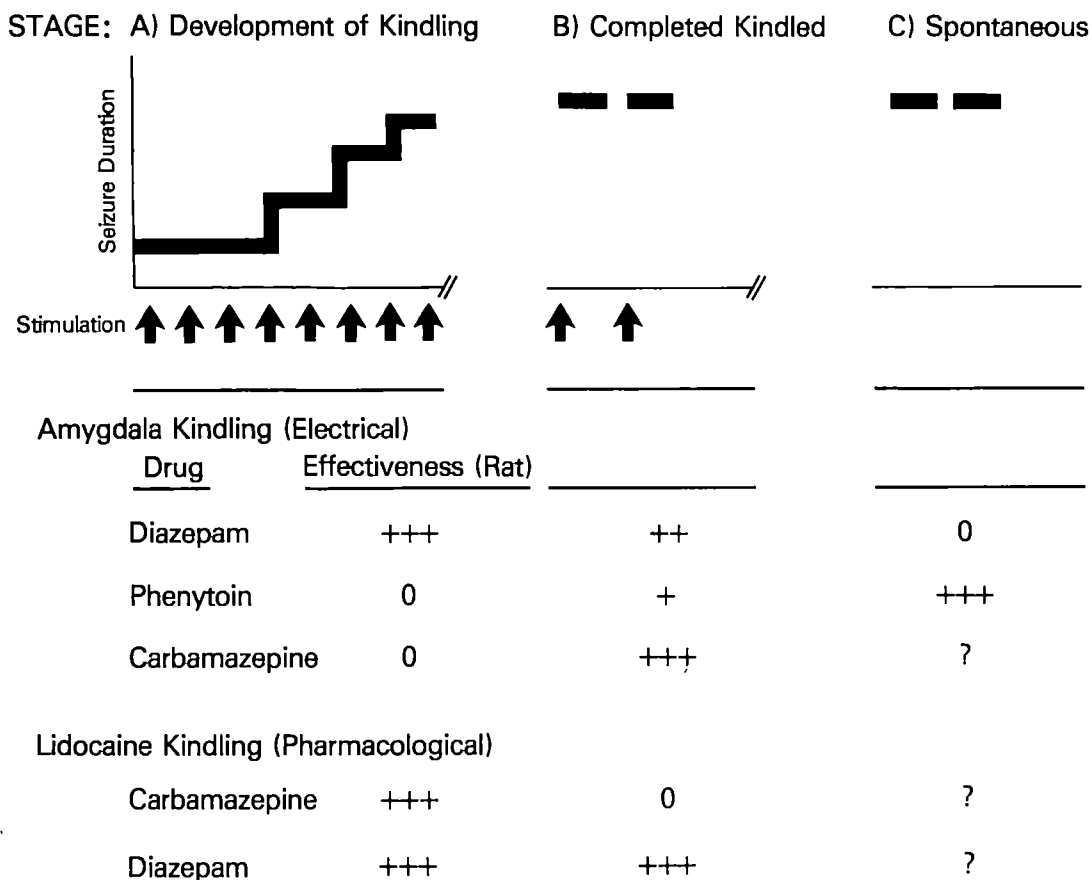


FIG. 2. Pharmacology of kindling as a function of stimulus type and stage of development.

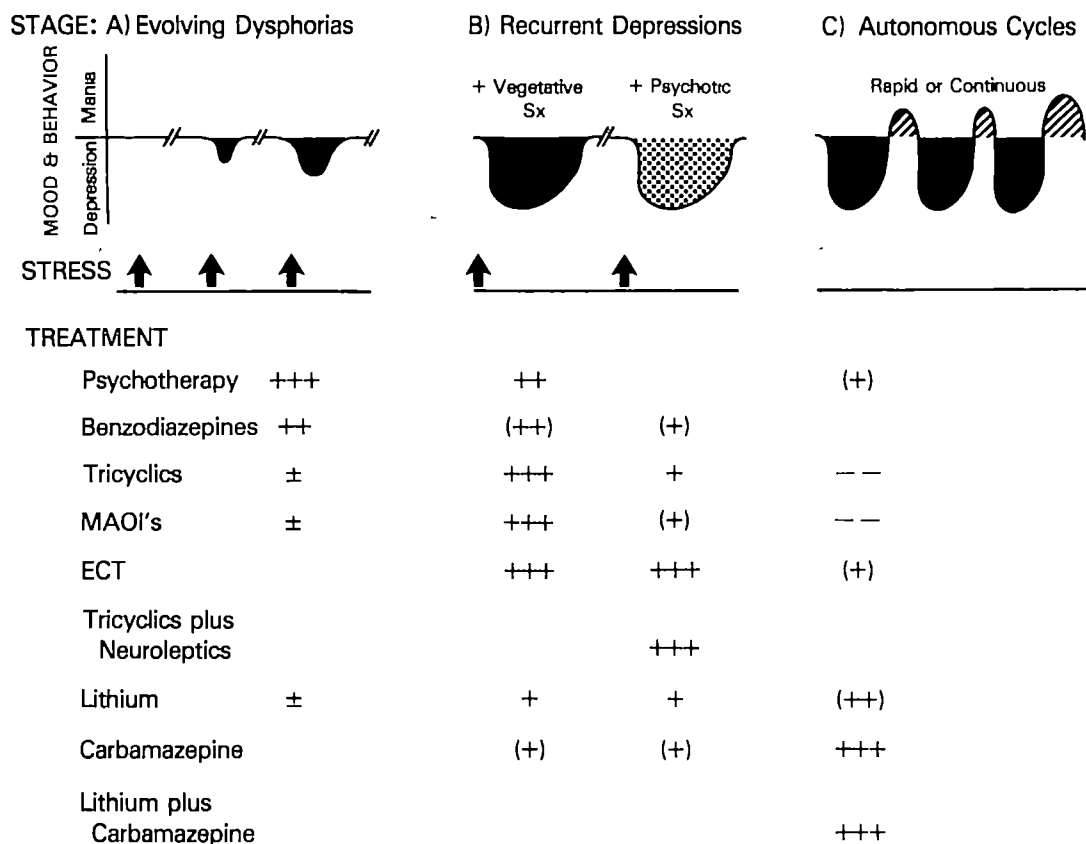


FIG. 3. Hypothetical psychopharmacotherapy of affective illness as a function of type and stage of development.

drugs and psychotherapeutic techniques, but also for suggesting alternative interventions. As reviewed in detail elsewhere (Bunney & Garland, 1984), lithium appears to inhibit a variety of acute and compensatory biochemical and behavioural changes observed following perturbations, particularly of the catecholamine system.

It is possible that other agents, such as carbamazepine, may affect other mechanisms and thus be associated with a different spectrum of clinical efficacy. Carbamazepine appears to be an effective treatment for many lithium-non-responsive, rapidly-cycling patients, both acutely and prophylactically (Post *et al*, 1984, 1985). The kindling and sensitisation models were influential in our initiating clinical trials of this anticonvulsant in affective illness. Carbamazepine inhibits amygdala-kindled seizures (Albright & Burnham, 1980; Wada *et al*, 1976; Babington, 1977), but not cocaine-induced behavioural sensitisation phenomena (Post *et al*, 1984). In

contrast, lithium appears to inhibit behavioural sensitisation, but not amygdala kindling.

As summarised in Figure 2, different stages of kindling and different types of kindling are differentially responsive to anticonvulsant treatment. For example, diazepam blocks the development of kindling, is effective in blocking completed seizures, but is ineffective for spontaneous seizures that develop very late in the kindling process (Pinel, 1983). In contrast, phenytoin is largely ineffective in the first phases, but blocks spontaneous seizures. Carbamazepine does not block the development of electrical kindling, but blocks completed amygdala-kindled seizures (Weiss *et al*, 1986a). In contrast, carbamazepine blocks the development of lidocaine-kindled seizures, but fails to inhibit completed lidocaine seizures (Weiss *et al*, 1986b). Thus, different developmental stages of an apparently unitary phenomenon such as kindling are differentially responsive to treatment.

We suggest that a parallel phenomenon might occur in affective illness, where treatment efficacy may vary as a function of course of illness. As depicted in Figure 3, psychotherapy and benzodiazepine may be effective in early stress-related dysphorias, while traditional antidepressants are effective in recurrent depression, but may exacerbate cycling in some bipolar patients. Lithium appears to be less effective in the treatment of rapidly or continuously cycling patients (Kukopulos *et al*, 1980; Prien, 1984), while these factors may be associated with better response to carbamazepine (Post *et al*, 1985, 1986; Kishimoto & Okuma, 1985). While investigators and clinicians would no doubt argue about the precise categorisations in Figure 3, and the validity of their current supporting evidence, we present the figure in order to raise the question of a differential pharmacology based on the course of illness and to stimulate further research. If valid, this concept might also suggest that underlying neurobiological mechanisms differ as a function of course of illness. Finally, the sensitisation and kindling models would predict that sufficiently early and adequate prophylactic treatment of affective illness should retard the development of later stages of the illness, such as more rapid recurrence and continuous cycling.

Dollard and Miller (1950), in their classic work, 'translated' a variety of psychotherapeutic techniques into the language of learning and conditioning. We have tried to emphasise how the sensitisation and conditioning perspectives might provide additional alternative conceptions of the impact of psychosocial stresses and psychotherapy, not only on psychological conditioning, but on biochemistry and physiology as well. Many current psychotherapeutic techniques could be viewed in relationship to providing effective cognitive restructuring (Beck, 1970: 1976), social support (Weissman, 1979), and potential desensitisation (Wolpe, 1973) or extinction of core conflicts and stress-loss precipitated reactions.

In addition, one might conceive of therapeutic strategies more systematically directed at desensitisation of critical cognitive structures, and symbolic representations of loss or stress, which have acquired cue or precipitant potential in relation to affective illness. We have found that longitudinal charting of affective episodes (Squillace *et al*, 1984; Roy-Byrne *et al*, 1985) is useful, not only in accurately describing the course of affective illness, but

also in helping to more systematically identify critical psychosocial stresses and areas of sensitivity in a given patient, which appear to be temporally related to repeated episodes of affective illness. Work in psychotherapy might also include the construction of a hierarchy of events and ideas that are particularly prone to evoke dysphoric feelings, affects, and the depressive syndrome. These might provide the focus for psychotherapeutic working through and systematic desensitisation. Particularly in patients who have achieved a relatively high state of conditioned emotional reactivity or stress sensitisation, more formal deconditioning techniques might be explored as adjunctive procedures to psychotherapy and pharmacotherapy.

A further utility of the sensitisation-conditioning model might be in the better understanding by the patient of the possible role of both psychological and biological processes in the evolution and/or treatment of his or her illness. With the increasing potential for polarisation of theory and therapy into either biological or psychosocial schools, the patient may be at a loss to understand how psychotherapy and psychosocial influences may be related to the growing evidence for biochemical abnormalities and responses to drug treatment in affective illness. The current exposition highlights the possibility that both psychological and biochemical responses can be associated with conditioning processes, which are themselves amenable to both psychological and pharmacological intervention. This frame of reference adds the longitudinal perspective to the multifactorial considerations of Akiskal and McKinney (1975) and others (Meyersburg & Post, 1979) who have emphasised biological and psychosocial interplay in the development of affective symptomatology.

The kindling and sensitisation paradigms may help focus theoretical and practical clinical attention on the longitudinal course of behavioural and biological change in affective illness, in contrast to the more frequently considered acute characteristics and treatment. We suggest these models not as accurate homologies for the depressive and manic-depressive illness, but as potentially useful constructs in the consideration of a variety of normal and pathological adaptive processes (both tolerance and sensitisation) which may occur upon the repeated presentation of physiological, biochemical or psychosocial stresses.

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Alterations in Cerebral Laterality during Acute Psychotic Illness

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A new dichotic listening test (fused rhymed words) was used to monitor changes in perceptual asymmetry (PA) in patients recovering from acute psychotic episodes; some were also given a dichotic nonsense syllables test, a dichotic tone test, and a single visual field dot localisation test. Contrary to expectation, PA on the word test decreased with recovery, while asymmetry on the tone and dot tests did not change; results cut across diagnosis, could not be related to medication, and were independent of changes in overall performance. More specific hypotheses are needed than those of generalised single hemisphere dysfunction in major psychiatric illness, but existing models and concepts are inadequate to explain the findings.

Numerous studies have examined putative indices of regional brain function (RBF) in psychotic illness, including perceptual asymmetry (PA), spontaneous and evoked electrical activity, and blood flow and metabolism (Flor-Henry, 1979; Gruzelier, 1981; Merrin, 1981, 1982; Newlin, 1981; Riederer, 1981; Wexler, 1980). Most purport to have found evidence of a single localised dysfunction, and suggest that this is characteristically associated with a particular clinical disorder.

However, many have been marked by the methodological shortcomings generally found in first-generation studies: measures of RBF have often been of limited reliability and/or validity; most studies have included patients of only one diagnostic group, making it necessary when attempting to compare different clinical disorders to compare data from different studies using different experimental measures and designs; and many have only compared patients and healthy controls, without assessment of severity of illness or sick/well repeated measures. (Such procedures are essential in differentiating state from trait abnormalities, and are potentially useful in further validating and defining the nature of an association between alteration in RBF and a clinical disorder). Finally, most studies have used only one measure of RBF. Problems resulting from the use of single measures are twofold: the degrees of freedom in drawing inferences from them about possible brain dysfunction are so great that specific conclusions cannot be drawn with any confidence; and the use of single measures biases conclusions toward single localised dysfunctions, since it is then impossible to see enough to assess integrative and interactive aspects of brain physiology (Wexler, 1986).

A new and improved language-related dichotic listening test was used here to assess RBF in acutely psychotic depressed and schizophrenic patients. Subjects were presented with dichotic pairs of single-syllable rhymed words, differing only in the initial consonant. Members of each pair fuse into a single auditory image, and subjects experience and report only one word on each trial. In a multi-centre study of right- and left-handed controls, this test proved to be very reliable and to approximate the validity criteria derived from neurological patients (70% right ear advantages (REA) in sinistrals and 95% in dextrals) as closely as any test currently available (Wexler & Halwes, 1983). Although developed specifically for this study, it is now being used world-wide.

The dichotic word test was used to follow-up an earlier finding of an increase in REA on a dichotic nonsense syllable test, as depressed and schizophrenic patients recovered from acute psychotic episodes (Wexler & Heninger, 1979). The nonsense syllable test was also a fused single response test, in which members of each pair differed in only one consonant and in which the set of such consonants was the same as those distinguishing the rhymed words. To facilitate comparison of the two studies, a sub-set of subjects in the present one also received the nonsense syllables test; sub-sets of patients also received two other PA measures. The four tests together constituted the current state of development of a multi-test battery. The first of these was a dichotic tones test, in which subjects receive a different tone in each ear and indicate which sounds louder; normal subjects show a highly reliable right ear bias (Wexler & Halwes, 1981). The last test was a visual dot localisation test, modelled on one of Levy

& Reid (1978). Dots are flashed briefly in either the right or left visual field, and subjects are required to indicate the position on a response key. Normal subjects typically have a left visual field advantage (LVFA), presumably because specialised final processing of these stimuli takes place in the right hemisphere. All tests were given to subjects twice—shortly after admission, when acutely symptomatic, and again after treatment and major improvement in symptoms; severity of symptoms was assessed at both times with a multidimensional nurses' rating scale (Heninger *et al.*, 1970).

The recovery-associated increase in REA on the nonsense test, noted earlier, was hypothesised to reflect a breakdown in normal interhemisphere inhibitory processes during acute psychosis (Wexler & Heninger, 1979). The present project was designed to replicate the initial finding, and to determine whether results with other measures of PA were consistent with this hypothesis. It was predicted that because of the similarity between the word and nonsense tests, results from them should be comparable. The tone and dot tests, on the other hand, were different enough from the other two for their results to help define the limits of the pathological process reflected in them.

Method

Subjects

Patients admitted consecutively to the Connecticut Mental Health Center, who met research criteria for the diagnosis of schizophrenia or primary major depressive disorder (Spitzer *et al.*, 1975), and were right-handed, participated in the study. One patient's test scores were markedly different from all the others', and he was excluded on this basis. The median age of the remaining 26 was 40 years, range of 19–64, 11 were men and 15 women. Seven were diagnosed as schizophrenic (four men, three women, median age 25 years, range 19–38) and 19 as primary major depressives (seven men, 12 women, median age 50 years, range 23–64). All 26 took the dichotic words test. Sub-sets of 15 took the dichotic nonsense test (three schizophrenic, and 12 major depressive disorder), 19 took the dichotic tones test (three schizophrenic and 16 major depressive disorder) and 16 took the visual dot localisation test (three schizophrenic and 13 major depressive disorder). All patients were without major illness other than their psychiatric disorders and none were taking non-psychiatric medications; none had any history of neurological illness or injury, language or learning disability, or excessive alcohol or drug use.

Control subjects were right-handed and without history of psychiatric, neurological or major medical illness, learning or language difficulties, or excessive drug or alcohol use. None were taking medication with CNS effects. There were 47 controls for the words test (23 men, 24 women, median age 27 years, range 17–56); 31 for the nonsense test (nine

men, 22 women; median age 27 years, range 20–38); 16 for the tones test (eight men, eight women; median age 27 years, range 20–35); and 29 for the dot test (14 men, 15 women, median age 28 years, range 20–56).

Research design

Patients were tested initially seven to 14 days after admission, when actively symptomatic. Each PA test was given twice on the same day, and the results combined. Word and nonsense tests were given on separate days, with about half the subjects receiving the word test first and the other half receiving the nonsense test first. The dot and tone tests were often given on the same day, with a two hour break between tests, and were generally given after the word and nonsense tests. All tests given initially were then repeated 12 to 145 days later (median 50), when clinical staff considered that maximum expected clinical improvement had occurred. Fourteen patients were off all medication at the time of initial testing (median in hospital drug-free period was ten days, range seven to 14), eight of the remaining 12 were on the same or the same class of medication (e.g. antidepressant) on initial testing as they were on retesting. The remaining four patients discontinued or had a change in class of medication between the initial and final testing. The depressed patients were treated with TCAs and/or lithium carbonate, all within usual therapeutic dose-ranges. The schizophrenics were treated with haloperidol or chlorpromazine, within usual dose-ranges. All 23 controls for the nonsense test took the test repeatedly, 11 with a nine-week inter-test interval, and the remaining 12 with less than a one-week interval; 18 word test controls, all 16 tone test controls, and ten dot test controls were retested within one week of original testing.

Behavioural measures

Once during the 7 am–3 pm shift and once during the 3–11 pm shift, patients were rated by nursing staff on a 13-item clinical rating scale, identical to one described by Heninger *et al.* (1970), save for the elimination of an expressed anger rating. Ratings on the day prior to a PA test, the day of the test, and the day after the test (six ratings in all) were averaged for a clinical rating to correspond to each PA value.

Perceptual asymmetry tests

The dichotic word, nonsense, and tone tests have all been described elsewhere (Wexler & Halwes, 1981, 1983; Wexler & Heninger, 1979). The word test consists of 15 different single syllable word pairs, in which each member of every pair differs from the other only in the initial consonant, e.g. coat, goat. All words begin with one of six stop consonants, b, d, p, t, g, k, and are natural speech, spoken by a male voice. The initial consonant portion of one member of each pair was cross-spliced onto the vowel-final consonant portion of the other member on the DDP 224

Computer System at Haskins Laboratories. The members of each pair fuse into a single auditory image. Consequently, subjects are aware of hearing, and report only one word on each trial. Each pair is presented twice in 30 trial blocks, the second time with each word to the ear opposite that of the first presentation. Four such 30-item blocks make up the test, for a total of 120 trials. Subjects indicate what word they heard by making a line through it on an answer sheet that has four possible responses—both members of the dichotic pair and two other words differing from the dichotic stimuli only in the initial consonant. A score for each ear is determined by totalling the number of times the word presented to that ear is the one indicated: an error is scored when neither of the pair is chosen.

The nonsense test consists of dichotic pairs, made up of the six stop consonants, b, d, p, g, t, k, preceded and followed by the vowel 'a' (e.g. aba, aka). The stimuli are synthetic copies of natural speech. As in the word test, the members of each pair fuse into a single auditory image, and subjects expect, experience, and report only one response on each trial. Three of the 15 possible pairings of the six syllables were found to be subject to frequent phonemic blending errors (Halwes, 1969) and were therefore discarded. The remaining 12 pairs were presented twice in 24-pair sequences, the second time with each syllable to the ear opposite that of the first presentation. Four such blocks were combined to make a 96-trial test. Subjects indicate what syllable they hear by writing b, d, p, g, t, or k in a blank space between two a's on a prepared answer sheet. The test is scored as for the word test.

In the tone test, each dichotic pair consists of a 440 and a 450 Hz tone, differing in volume by 0–10 dB; subjects indicate which sounds louder. If subjects indicate that one ear signal was louder when in fact the opposite was true, the ear chosen is assigned a number of points equal to the number of decibels by which the stimuli differed in intensity. Right- and left-ear scores are combined to give a total error score. All test tapes were made at Haskins Laboratories in collaboration with Terry Halwes. Tapes were played on a Teac-2-track master recorder, and stimuli delivered through matched pairs of TDH-39 earphones. Calibration tones at the beginning of each tape were balanced to minimise channel effects, while remaining channel effects were controlled by reversing earphones after the first and third quarters of the tests. In all tests, subjects received practice trials before the start, to familiarise them with the procedure and to decrease anxiety and errors. On the words and nonsense tests, subjects were not told, and did not realise that they were getting different inputs in each ear on each trial. In the loudness test, they were told they would hear a different tone in each ear, and should indicate which sounds louder.

The visual dot localisation test was modelled after one developed by Levy & Reid (1978). Subjects are instructed that a single dot will appear in one of 40 possible positions within a circular frame, encompassing both the left and right fields; there are 20 different, randomly chosen positions, mirrored in the two visual fields. Stimulus slides are exposed for 120 msec. Subjects report the location of the dot from a hand-held response key. The circular frame extends to 3.5° lateral of fixation, with dot positions

ranging from 0.7° to 2.7°. Subjects sit 30 inches from the screen, directly in front of the central fixation point.

A digit, 0–19 is presented in the centre of the field on each trial and subjects report this digit before indicating dot location; this central digit subtends an angle of 0.2°. The pre-stimulus fixation/post stimulus noise slides consist of an x in the centre for fixation, and several different types of visual noise in the remainder of the slide. Dots are presented once in every position in each visual field for a total of 40 trials, with a two-minute rest after 20 trials. Trials on which the central fixation number is incorrectly reported are not scored, and are repeated at the end.

The numbers of items correctly identified in each field constitutes the score for each; the numbers of items identified incorrectly in both, together constitute a total error score. Results from subjects who made more than 75% errors were discarded. Prior to the test, subjects were familiarised with the procedure and stimuli by being shown similar slides, and were required to identify the central number and to indicate whether or not a dot was present.

Auditory acuity was tested in each ear by the method of ascending and descending limits, using tones of 250, 500, 1,000, 2,000, and 4,000 cps; no subject had a difference in auditory acuity between the ears of more than 5 db on more than two of the five frequencies tested. Handedness was assessed by observing which hand was used in each of eight tasks: writing, throwing a ball, dealing cards, opening a jar, threading a needle, brushing teeth, cutting with scissors, and striking a match. All subjects wrote and did at least five of the other seven tasks with the right hand.

Data analysis

Laterality scores were calculated by subtracting left-ear (or visual field) scores from right-ear (or visual field) scores, and dividing the difference by the sum of the two. Independent t-tests were used to evaluate mean differences between groups, paired t-tests to evaluate change within subjects over time, and Pearson correlation coefficients to evaluate relationships among the variables.

Results

Patients compared to controls

On the dichotic nonsense and tone tests and on the visual dot test patients, when first tested, tended to be less lateralised than controls, but none of these differences reached the $P < 0.05$ level of statistical significance. Mean laterality for the patients on the nonsense test was 0.03 ± 0.19 , for the controls 0.12 ± 0.18 , 2 tail $P < 0.10$, on the tone test 0.07 ± 0.59 and 0.33 ± 0.45 , $P < 0.17$, and on the dot test 0.03 ± 0.17 and -0.08 ± 0.21 , $P < 0.09$. Asymmetry on the word test among patients when they were acutely symptomatic was essentially identical to that among controls; 0.13 ± 0.14 and 0.13 ± 0.15 . Patients made more errors than controls did on the word test (pts. 5.4 ± 4.76 , con. 1.8 ± 2.1 , $P < 0.001$) and on the nonsense test (pts. 2.5 ± 3.1 , con. 0.38 ± 1.1 , $P < 0.05$), but patients and control error rates were not significantly different on either the

TABLE I

Changes in dichotic nonsense and dichotic words tests with recovery from acute psychotic episodes

<i>Nonsense (n=15)</i>	<i>Sick</i>	<i>Well</i>	<i>P value (2-tail)</i>
Laterality (PD index)	0.03 ± 0.19	0.07 ± 0.20	P=0.10
Errors	2.8 ± 3.2	5.1 ± 7.1	P=0.13
Word Test (n=26)	Sick	Well	P value (2-tail)
Laterality (PD index)	0.13 ± 0.14	0.06 ± 0.11	P=0.001
Errors	5.4 ± 4.6	4.1 ± 5.1	P=0.28

tone or the dot test. There were no significant correlations between error rate and asymmetry on any of the tests in either controls or patients. Results of each statistical comparison were essentially the same when sub-groups of the sample (depressives, schizophrenics, patients off and on medication) were examined separately.

Changes in asymmetry with clinical improvement (Table I)

Following treatment and clinical improvement there was a decrease in PA on the dichotic word test; initial mean laterality 0.13 ± 0.14 , retest 0.06 ± 0.11 , 2 tail $P < 0.001$. The sub-set of 15 patients who also took the nonsense test showed the same increase in laterality noted in an earlier study; laterality means 0.03 ± 0.19 and 0.07 ± 0.20 , one tail $P < 0.05$. Changes in the tone test (0.07 ± 0.59 and 0.02 ± 0.06) and in the dot test (0.03 ± 0.17 and 0.02 ± 0.14) did not approach significance. The degree of change in PA in individual patients on the nonsense test was not correlated with the degree of change on the word test (Pearson correlation 0.08). Changes in all tests were similar in sub-groups defined by diagnosis (schizophrenia or depression) or by medication status (group 1 off medication initial testing, on antidepressant medication second testing, group 2 on the same medication for both initial and repeat testing). Controls did not show significant changes with retesting on any of the four tests. Error rates on neither the word test nor the nonsense test changed significantly with clinical improvement.

When recovered, asymmetry on the word test in the patient group was significantly less than in the controls (pts 0.06 ± 0.11 , controls 0.14 ± 0.16 $P < 0.04$). Asymmetry on the nonsense test, which had been less than that of controls when patients were sick ($P < 0.10$), moved closer to control values (pts. 0.07 ± 0.20 , controls 0.13 ± 0.18 , $P < 0.26$). Asymmetry on both the tone and the dot tests moved farther from control values with recovery so that differences between recovered patients and controls were significant on both tests (tone test, pts 0.02 ± 0.47 , controls 0.33 ± 0.45 $P < 0.03$; dot test pts. 0.04 ± 0.4 , controls -0.08 ± 0.21 , $P < 0.05$).

Correlation between PA and clinical symptomatology

Two sets of correlations between PA and symptomatology were calculated; between initial PA scores and initial clinical ratings, and between recovery-related changes in PA and degree of symptom improvement. The correlations between PA on the tone and dot tests and clinical ratings were not significant in either of the sets with a frequency greater than would be expected by chance alone. The same was true for initial PA scores on the word and nonsense tests and initial nurses' ratings. Errors on the word test when patients were acutely symptomatic were positively correlated with ratings of odd or unusual thoughts (0.58 $P < 0.002$), anxiety (0.40 $P < 0.04$), disorganised thinking (0.53 $P < 0.006$), hallucinations (0.47 $P < 0.01$), paranoia (0.44 $P < 0.02$) and overall psychosis (0.57 $P < 0.002$). Correlations between errors on the nonsense, dot and tone tests, and clinical ratings were not statistically significant.

The correlations between recovery-related changes in PA and errors on the word and nonsense tests, and recovery-related changes in nurses' ratings are presented in Table II. All change variables were calculated by subtracting initial values from time two values (repeat or well values). Thus, an increase in laterality on the nonsense test would lead to a positive change score, while a decrease in laterality on the word test, a decrease in test errors, or a decrease in symptomatology would lead to a negative change score. Correlations between changes in PA on the nonsense test and changes in severity of symptoms were consistent with the recovery-related increase in laterality in group mean scores. Those patients with larger increases in laterality on the nonsense test had greater improvement in ratings of odd and unusual thoughts, disorganised thinking, hallucinations, paranoia, and overall psychosis. Neither changes in the overall depression rating nor in specific aspects of depression (ie. anxiety, withdrawal, agitation, motor retardation, decreased energy) were correlated with changes in asymmetry. In contrast, on the word test, only changes in the magnitude of social withdrawal, motor retardation and decreased energy—all symptoms associated with depression—were significantly correlated with change in PA. However, the direction of correlation was opposite what might be expected from the group mean decrease in asymmetry on the word test with recovery: those patients with smaller decreases in laterality exhibited greater improvements in symptomatology.

Decrease in errors on the words test was positively correlated with improvement in ratings of psychotic symptomatology. Although change in laterality on the nonsense test was also correlated with change in most of the same symptoms as was change in words test errors, the correlation between change in laterality on the nonsense test and change in overall errors on the words test was not significant (0.23). The extent of change in errors on the other PA measures were not correlated with change in ratings of symptoms.

Separate analyses were conducted for patients off medication when ill, patients on medication both ill and well, schizophrenic patients, and depressed patients. In patients off medication, PA on the word and nonsense tests when ill tended to be more highly correlated with ratings of

TABLE II
Correlations between recovery related changes in dichotic variables and recovery related changes in clinical ratings

	NPD	NERR	WPD	WERR	THO	ANX	WITH	AGIT	DIST	HOST	MRET	HALL	ENE	PAR	PSY	DEP	MAN
<i>Nonsense Test</i> Laterality (PD index)	R	1.00	0.08	-0.19	-0.59	-0.25	0.03	-0.09	-0.63	0.08	0.22	-0.68	0.14	-0.55	-0.61	0.14	-0.33
	P	0.00	0.50	0.78	0.03	0.37	0.91	0.75	0.01	0.78	0.44	0.005	0.63	0.03	0.02	0.62	0.23
<i>Errors</i>	R	-0.19	1.00	0.55	-0.14	0.07	-0.23	-0.134	0.27	-0.02	-0.23	0.14	0.04	-0.07	-0.11	-0.12	0.21
	P	0.50	0.00	0.73	0.03	0.82	0.40	0.62	0.34	0.93	0.42	0.63	0.88	0.79	0.69	0.68	0.44
<i>Words Test</i> Laterality (PD index)	R	0.08	1.00	-0.54	-0.11	-0.31	-0.36	0.02	-0.12	-0.30	-0.35	-0.06	-0.45	-0.21	-0.12	-0.26	-0.11
	P	0.78	0.73	0.00	0.004	0.12	0.07	0.92	0.57	0.14	0.08	0.77	0.02	0.310	0.55	0.19	0.59
<i>Errors</i>	R	-0.19	0.55	-0.54	1.00	0.40	0.43	0.38	0.51	0.21	0.03	0.45	0.07	0.33	0.40	0.06	0.29
	P	0.51	0.03	0.004	0.00	0.04	0.03	0.05	0.007	0.30	0.88	0.02	0.74	0.10	0.04	0.76	0.15

symptoms than they were in the entire group, but patterns of correlation in other groups were similar to those of the entire sample.

Discussion

The recovery-related increase in PA on a dichotic nonsense test, noted by Wexler and Heninger (1979), was replicated here; in that study, the degree of increase in asymmetry on this nonsense test was correlated with the degree of decrease in clinical ratings of paranoia, and this correlation was confirmed. In addition, the degree of increase in asymmetry was found to correlate with the degree of improvement in ratings of odd and unusual thoughts, disorganised thinking, hallucinations, and overall psychosis. When ill, patients showed less asymmetry than healthy controls; the increase in asymmetry with recovery moved patients' asymmetry values closer to those of controls'.

Perceptual asymmetry on a dichotic word test decreased as patients recovered. Despite the fact that this test was very similar in task, structure, and stimulus characteristics to the nonsense test, the recovery-related change in PA on this test was opposite in direction to that on the nonsense test. Recovery-related changes moved patients' asymmetry away from control values, so that although patients' group mean asymmetry was essentially identical to that of controls when the patients were ill, when they recovered, their mean asymmetry was significantly less. Those patients with the greatest decreases in PA with recovery tended to show the smallest decrease in ratings of social withdrawal, motor retardation, and decreased energy—which can be related to depression and/or side-effects of medication. The decrease in PA on the word test was not related to the change in ratings of psychosis or of psychotic thought processes.

Errors on the dichotic word test were positively correlated with ratings of psychotic thought and behaviour when patients were ill, while recovery-related decreases in errors were positively correlated with improvement in these ratings. The relationships between errors on the word test and the clinical variables were very different from those between errors on the other tests and these variables. When ill, patients made significantly more errors than controls on the word and nonsense tests, but not on the dot or tone tests; it was only on the word test that errors were significantly correlated with clinical variables. In controls, the word test consistently yielded fewer errors than any of the others. Together, these observations suggest that the associations between errors on the word test and clinical

variables reflect a specific aspect of brain dysfunction, and are not merely a reflection of the general decrement in performance associated with psychosis.

Data generated by the tone and dot tests were not robust: group mean values on neither test changed significantly as patients recovered, and neither asymmetry nor errors on either test correlated significantly with clinical ratings. However, when ill, patients tended to be less lateralised on both tests than were controls. With recovery, patients' values moved further from those of controls, and the differences became statistically significant.

Interpretation of these data is difficult: firstly, because recovery-related changes on different tests are in different directions, both with respect to initial illness values and controls, and secondly, because there are data from four different tests. It would be easier to consider those from only one or two, but such simplification would merely cause delay. Despite such efforts, however, an interpretation that incorporates all statistically significant findings has not so far been possible, but as the addition of new pieces to a complex puzzle provides places for those previously impossible to fit, future studies will perhaps allow interpretation of all current data.

Recovery-related changes in asymmetry

It is striking that asymmetry on two tests as similar as the dichotic word and nonsense tests would change in opposite directions with recovery. The fact that the change on the nonsense test has been replicated, together with the high statistical significance of change in the word test, suggests that the difference in direction is worthy of attention. There is also support from three other sources. Firstly, similar opposite changes in asymmetry on these same two tests have been noted in healthy subjects with administration of concurrent visual tasks (Wexler & Halwes, 1985). Secondly, the two tests fluctuate in opposite directions from day to day in depressed patients during the initial phase of ECT (Wexler *et al.*, unpublished). Lastly, schizophrenics have greater asymmetry than controls on a dichotic word test (Lishman *et al.*, 1978), and less on a dichotic nonsense test (Colburn & Lishman, 1979).

It seems unlikely that the differences between the word and nonsense tests result from merely procedural factors. In the previous recovery study, the nonsense test was given alone, and the same increase in PA with recovery noted; the nonsense test was given on successive days, as were the word and nonsense tests in the present study, and scores on each of the illness test days were lower than those on the well days. To control for test-order effects, in the present study, half the patients who received both

tests received the word test first, and half the nonsense test first: those who did not take the nonsense test showed the same decrease in PA as did the entire group. Finally, an unpublished study found inter-correlations ranging from 0.52 to 0.70 ($n=40$, $P<0.001$) among four word tests, each modelled after the one used in this study but differing in the emotional quality of stimulus words. Together with the high test-retest reliability of the word and nonsense tests, this makes it unlikely that the differences observed here between them are due to the administration of multiple tests.

Explanation of the results must be *post hoc*, since they were unanticipated, and requires validation. Three explanatory approaches were considered: that changes on the two tests represent different and independent regional brain dysfunctions; that dysfunction of a single brain region, anatomically defined, accounts for changes in both tests; and that a single change in brain physiology accounts for changes in both tests. The first, though supported by the absence of correlation between recovery-related changes in PA on the two tests, was rejected: each of the possible independent dysfunctions must differentiate between word and nonsense stimuli, altering one test but not the other. In addition, unless the number of such hypothetical functions and dysfunctions is to be increased, the same two functional alterations must be postulated to account for the changes in asymmetry seen in healthy subjects with concurrent visual tasks (Wexler & Halwes, 1985), and for the reciprocal day-to-day changes in asymmetry on these tests, noted in depressed patients during the initial phase of ECT (Wexler *et al.*, unpublished).

The second possibility—that dysfunction of a single anatomically defined area led to opposite changes in asymmetry—seems improbable. According to existing models of perceptual asymmetry, change in function of all areas that contribute to asymmetry on both tests would affect both tests in the same way. It is possible, however, that efficiency of information processing in a particular area varies along an inverted U-shaped curve as a function of activation (or other factors) of that area, and that because of differences in the functional demands posed by processing words and nonsense, these two processes are at times on opposite sides of the maximum point of this curve. Changes in the function of such an area with disease, concurrent visual tasks, or ECT could then have opposite effects on asymmetry on the two tests. Dysfunction of a single anatomical area then remains a viable explanation, and one that might be amenable to further experimental validation.

The third approach invoked the possibility that a single physiological alteration might account for the opposite changes on the words and nonsense tests. I have developed (Wexler, 1986) a physiological model of brain function, based on Luria's notion of cerebral functional systems: specific behaviours or cognitive functions are not localised in specific brain regions, but are the result of the concerted action of a functional system. The specific anatomical components of these systems may vary, while the behavioural or cognitive output remains constant, and from this perspective, integrative and interactive processes within and among functional systems are central in normal brain function. Major alterations in mood and behaviour would be associated with changes in these processes.

The simultaneous and unacknowledged presentation of different stimuli to each ear in dichotic tests create unnatural functional demands: perceptual asymmetry is not an index of ordinary function. It probably does not make sense, therefore, to speak of the functional systems for processing the dichotic word and nonsense stimuli. The present data suggest, however, that in recovery from acute psychotic states the functional systems for processing auditory information as meaningful or as nonsense are altered in such a way as to have opposite effects on PA in the dichotic words and nonsense tests. A unitary explanatory hypothesis suggests that there is a change in the relationship between the functional systems, leading to opposite changes in PA.

The degree of PA is thought to reflect, among other things, facilitation of information flow from the right ear by stimulus-specific activation of left hemisphere attention centres (Kinsbourne, 1975). Assuming that the degree of such activation is proportional to the activity of the stimulating functional system, the present data suggest that: (1) the functional system for processing auditory information as meaningful, and that for processing auditory information as nonsense, are normally in a mutually inhibitory relationship, and (2) in some cases of acute psychosis, the balance of activity of the two systems is altered so that the functional system for processing auditory information as meaningful is more active and that for processing information as nonsense is less active. Decreased activity of the functional system for processing auditory nonsense during acute illness would lead to decreased activation of left-hemisphere attention centres and, consequently, less facilitation of the right-ear input pathway and a decreased right-ear advantage with presentation of nonsense stimuli. A similar decrease in activity of the functional system for processing auditory words with recovery would

lead to the recovery-related decrease in asymmetry seen on that test.

The increase in errors on the word test, seen on initial testing, suggests that with a shift in the reciprocal balance between the word and nonsense systems toward the word system in acute illness, efficiency of the word system decreases.

Correlations between specific symptoms and laterality variables

Increase in PA on the nonsense test was significantly correlated with decrease in symptoms of psychotic thinking, but not with change in symptoms in energy level, or in motor or social activity. It is interesting that this was so in a group who were mainly psychotically depressed. The nature and extent of cognitive disturbance found in major depressive illness are variable, and these data suggest that PA on the nonsense test is associated with this important but variable component of the syndrome. Change in PA on the words test was not correlated with change in these symptoms, but instead tended to show an inverse correlation with change in social withdrawal, motor retardation, and lack of energy; those individuals with the greatest improvement in the symptoms showed less of the recovery-related decrease in PA that characterised the group as a whole. This is of interest because of the direction of the association, which suggests that a recovery and/or treatment-related change in brain function, as reflected in PA on the words test, may have a negative effect on certain symptoms. Identification of this process might aid in the improvement of treatment.

Errors on the word test when patients were ill were positively correlated with the severity of symptoms of psychotic thinking, and the degree of decrease in errors with recovery was correlated with the degree of improvement in these same symptoms. These associations are straightforward, and consistent with the recovery-related decrease in group mean errors. However, the association of errors on the word test with symptoms different from those associated with PA on that test eludes the explanation that errors on this test result from the same process that alters PA in it.

Asymmetry on the tones and dot tests

Asymmetry on these tests did not change with recovery for the group as a whole, and was not associated with particular symptom ratings. Some individuals did show changes on one or another of the two tests, and these may prove useful in defining clinically or physiologically meaningful sub-groups.

The absence of significant group-wide changes with recovery on either of these tests indicates some limits of the pathophysiological alterations revealed in the changes on the word and nonsense tests. Earlier writers suggested that global dysfunctions of one hemisphere or the other characterised the major psychiatric disorders. The interpretation here of opposite changes in PA on the word and nonsense tests is more specific, suggesting change in the functional relationship between two functional systems, each with important components in the left hemisphere, instead of a general left-hemisphere dysfunction. The absence of recovery-related changes in the dot and tone tests is consistent with such a limited hypothesis, but not with the earlier and more general one. The differences between controls and patients on these tests, when patients are recovered, do not fit the interpretation of recovery-related changes on the word and nonsense tests, however.

Conclusion

Four PA measures, with demonstrated reliability and validity, were used here to collect a large amount of data, which proved confusing, thus demonstrating the inadequacy of existing concepts and models in describing brain function and dysfunction. It has been postulated that normally, the functional systems

for processing auditory words and nonsense are in a mutually inhibitory balance. Considerable confusion remains, however, indicating the primitive stage of the experimental and theoretical work. Though difficult, multi-dimensional physiological studies are essential if the organisational principles of the brain as an organ are to be discovered.

The most significant suggestion from the data is that normally, the functional systems for processing auditory words and nonsense respectively are in a mutually inhibitory balance. It is interesting to consider such a possibility from the perspectives of both normal and pathological development, since at first, during childhood, all is nonsense. It is also interesting to consider it in relation to those symptoms of psychosis which might be considered to reflect excessive assignment of significance—ideas of reference, delusions, and hallucinations.

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Maintenance Treatment of Erectile Impotence by Cavernosal Unstriated Muscle Relaxant Injection

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Intracavernosal injection of phenoxybenzamine or papaverine has been tried on 127 men suffering from erectile impotence. In 113 of them it caused an erection which should have sufficed for coitus. Seventy-three men have used such injections, given at home by themselves or their wives, fortnightly or less often, to make coitus possible after each injection; 54 of them still do so.

Virag (1982) reported that injection of papaverine (80 mg) into a corpus cavernosum caused enlargement and stiffening of the penis, and a rise in pressure in the corpora cavernosa of up to 70 mmHg, lasting 10 to 20 minutes. He described using such injections, given every two or three months, as treatment. The purpose was not to enable patients to have sexual intercourse during the artificial erections caused by papaverine, but to achieve benefits lasting from one injection to the next. Such benefits were reported by some patients.

In August 1982, before Virag's work was published but after his discovery had been made, I found in self-experimentation that phentolamine or phenoxybenzamine caused erection when injected into a corpus cavernosum. These erections lasted a few minutes for phentolamine and several hours for phenoxybenzamine. I began almost at once to use phenoxybenzamine as maintenance treatment in erectile impotence, patients learning to inject themselves in order to have sexual intercourse the same day (Brindley, 1983). I became aware of Virag's earlier work on papaverine, and of his subsequent publications (Virag *et al*, 1983, 1984) in November 1983, and began to use papaverine as an alternative to phenoxybenzamine in December 1983.

Zorgniotti & Lefleur (1985) have reported using intracavernosal injection of a mixture of papaverine and phentolamine as maintenance treatment for impotence, and Virag (1985a) is now using papaverine in this way.

The general procedure of causing erection by intracavernosal injection of a drug needs a name. The drugs that are known to swell the penis (Brindley, 1986) are phenoxybenzamine and papaverine (both of which give erections lasting for hours, usually complete), phentolamine, thymoxamine, verapamil, naftidrofuryl, lignocaine and imipramine (which give erection, complete or incomplete, lasting a few

minutes) and guanethidine (which causes shrinkage of the penis for about the first hour, and then partial erection lasting many hours). All these drugs are relaxants of unstriated muscle. I therefore call the procedure CUMRI (cavernosal unstriated muscle relaxant injection), which has the merit of being easily spoken. The name cavernosal alpha-blockade (CAB), used earlier, was appropriate for phenoxybenzamine but is inappropriate for papaverine. Virag (1985b) has recently introduced the abbreviation PIPE (pharmacologically induced prolonged erection). Theoretically this term could be much broader than CUMRI, and could include drugs given by routes other than the cavernosal. As long as no other effective route is known, however, it carries the same meaning apart from excluding the short-acting agents.

The present paper reports the use of CUMRI to investigate 127 men with erectile impotence, and to treat 73 of them.

Table I classifies them by age and principal relevant disabilities. All impotent patients to whom at least one intracavernosal injection of a drug intended to cause erection was given before 31 December 1985 are included.

Technique

Since March 1983 the following procedure has been used. For phenoxybenzamine, 0.04-0.20 ml of the concentrated solution (containing 2-10 mg of the drug) is diluted with 10 ml of saline for doses up to 5 mg, or with 20 ml of saline for doses between 5 and 10 mg. For papaverine, the solution supplied by the manufacturer is undiluted. The dose is 16-120 mg (0.4-3.0 ml).

The injection is delivered through a 16 mm needle of 0.5 mm external diameter into one corpus cavernosum in the proximal third of the free penis. The corpus cavernosum into which the drug has been injected is squeezed firmly for

TABLE I
Present state of patients

Age	Spinal cord injury or disease ¹				Other patients ²				Total
	User	Former user	Non-user	Failure	User	Former user	Non-user	Failure	
20-29	10	1	11	1	1	3	3	1	31
30-39	9	—	3	—	3	1	3	—	19
40-49	6	—	3	1	5	4 ³	3	—	22
50-59	1	—	—	—	8	4	8	5	26
60-69	—	—	—	—	10	5	3	4 ⁴	22
70-79	—	—	—	—	1	1	3	2 ⁵	7
Total	26	1	17	2	28	18	23	12	127

1. Spinal cord injury 36, multiple sclerosis 7, spina bifida 1, poliomyelitis 1, paraplegia of obscure origin 1.

2. Mostly with no known relevant disease, but including 3 former alcoholics, 3 men with intermittent claudication, 2 with lesions of the hypogastric plexus, 2 schizophrenics formerly treated with phenothiazines, and 8 diabetics.

3. Includes one patient who died of coronary thrombosis after using CUMRI for 27 months.

4. Includes one patient who died of carcinoma of bladder.

5. Includes one patient who died of carcinoma of the colon.

Both the carcinomas had been diagnosed before the patients received their first injections.

about two seconds along its whole length with the thumbs and fingers of both hands simultaneously, to transfer the drug to the other side. The corpus cavernosum of the other side is then squeezed in the same way. Finally, the penis is pinched transversely with one hand at six or more different places so that the drug reaches both ends of the corpora cavernosa. This massaging procedure after injection is probably important in achieving consistency of effect. Stackl & Bucher (1984), who did not use it, reported failure.

I always give the first injection myself. Some patients lie supine, as a precaution against immediate fainting, but the majority sit for the injection. From the second or third injection the patient (or in a few cases his wife) begins to learn to inject. Patients inject standing or sitting. They are allowed to take a supply of the drug home when they have demonstrated their competence, and are asked to give the injection always in the same part of the penis, so that local fibrosis can be easily detected if it occurs.

I now always try papaverine first. If it gives an adequate erection it is the better drug to use, because side-effects are rarer and the dispensing procedure simpler. If the erection from papaverine is too brief, phenoxybenzamine usually gives more prolonged erection. Before March 1983, injections were made in the middle of the free penis, and the massaging procedure was less thorough. On 10 February 1986, for reasons explained below I ceased using phenoxybenzamine except in one patient.

At present, as a precaution against long-term harm, a limit on the frequency of injections is set at once per fortnight for phenoxybenzamine, and once per fortnight on average (i.e. 26 per year) for papaverine. The reason for making different rules for the two drugs is that there is no refractoriness for papaverine, but phenoxybenzamine works less well on the second of two trials within a few days of each other.

Results

In the majority of both impotent and normal men, CUMRI with phenoxybenzamine or papaverine causes the penis to become fully or almost fully stiff, so that it can be bent to right or to left through no more than 20° (i.e. to no less than 160°). The erection is restricted to the corpora cavernosa, the glans and corpus spongiosum do not swell, or swell very little. Some men develop only poor erections if they lie down to receive the injection and remain lying down, but the erection improves if they stand.

With papaverine, the greatest degree of erection is reached within 4-8 minutes. It commonly stays constant for 1-4 hours, and then subsides completely during 1 hour or less, leaving no obvious after-effect. With phenoxybenzamine, the greatest degree of erection may not be reached until 20 or even 40 minutes after the injection, though in some men it is attained within 10 minutes. It commonly stays constant for 2-6 hours, and then subsides slowly. The penis remains large for at least 24 hours, sometimes 72 hours, and during this period of enlargement erection is often easier to provoke by psychological or mechanical stimulation than before the drug was given.

Table I shows the state of all 127 patients, as known at 1 February 1986. In 113 patients, CUMRI with papaverine or phenoxybenzamine made the penis, for a period of at least 20 minutes, continuously stiff enough to resist bending at the middle of the shaft to 135° (i.e. through 45°) to right and left. This is roughly the threshold for successful coitus. In 14 patients, not even this degree of erection was achieved by CUMRI, though in some of them a larger dose might have succeeded. Failure is significantly more common in older patients. In 40 patients, CUMRI gave a degree of erection that could have allowed coitus, but has not been used at home for this purpose.

TABLE II
Classification of present users by date of first therapeutic use of CUMRI and drug used at 1 February 1986

Drug used at 1 February 1986	Therapeutic use of CUMRI began							Total
	Nov. 1982	Jan.-June 1983	July-Dec. 1983	Jan.-June 1984	July-Dec. 1984	Jan.-June 1985	July-Dec. 1985	
Papaverine	—	2	3	4	8	11	6	34
Phenoxybenzamine	1	2	3	5	0	5	1	17
Papaverine and phenoxybenzamine	—	2	—	—	—	—	—	2
Guanethidine	—	—	—	—	—	—	1	1
Total	1	6	6	9	8	16	8	54

Nineteen patients used CUMRI at home for a time, but no longer do so. One of these died suddenly after he had been using CUMRI (always phenoxybenzamine) for 27 months. Necropsy showed myocardial infarction from coronary thrombosis. He was well until a few hours before his death, and had sexual intercourse with the help of CUMRI 5 days before his death. I had palpated his penis 8 weeks before he died, and found it entirely free from lumps. He told me on that occasion that it was straight when erect.

Reasons given for ceasing to use CUMRI have included improvement, so that it was no longer necessary (3 cases); deterioration in the response to the drug (2 cases); loss of sexual partner (2 cases); and implantation of a penile prosthesis (1 case).

At 1 February 1986, 54 patients are still using CUMRI at home. Table II classifies them by drug used and length of use.

Benefits

All patients who have used CUMRI at home say that they have had coitus after every or nearly every injection, though they were always (48 patients) or usually (25 patients) incapable of intromission before treatment began. After papaverine injection, coitus is possible only in the first hour or few hours after injection. Of the 26 patients who have used phenoxybenzamine, 11 say it is often possible on the second, third, or fourth night as well.

No patient who was completely impotent when first seen has reported improvement other than the ability to have coitus in the first few hours (or for phenoxybenzamine the first four days) after an injection.

Among incompletely impotent patients, seven have reported general improvement. Three of these are now ex-users; two are non-users, having improved so much after one or three CUMRI in the clinic that they did not need to use it at home; and two are users, their improvement being insufficient to allow them to do without CUMRI.

Unwanted effects

Pain from the injection

Most patients feel a mild burning pain throughout the penis after injection of either drug. There is little difference in the severity of the pain between the two drugs, but it lasts 10-15 minutes for phenoxybenzamine and less than 2 minutes for papaverine. Accidental subcutaneous injection of phenoxybenzamine causes pain that is more severe than that from intracavernosal injection.

Haematoma

A haematoma, either subcutaneous or under Buck's fascia, appears after a small minority of injections. It is unsightly, but disappears in a few days and causes little pain or discomfort.

Injection into the urethra

A tetraparetic patient with defective penile sensation once accidentally injected papaverine into the urethra. Blood trickled from the meatus for a few minutes, but no other harm was noticed.

Priapism

The duration of action of both drugs is fairly consistent for a given dose in a given man, but it is dose-dependent, and for a given dose varies greatly between men. Priapism, defined as erection lasting continuously for at least 12 hours, has occurred in eleven patients (16 incidents). Some of these have been reported elsewhere (Brindley, 1984). Four incidents resolved spontaneously after 16-26 hours; all the other 12 were successfully treated with metaraminol. The cause was phenoxybenzamine in four incidents and papaverine in 12.

Decrease in the effectiveness of CUMRI

This has occurred in four patients: two using phenoxybenzamine, and two papaverine. In one phenoxybenzamine user the deterioration was slight, but since initially the erection produced by CUMRI was only just adequate for coitus even a slight deterioration made it inadequate, and he ceased to use the procedure. In the other phenoxybenzamine user the deterioration was severe. His erections from CUMRI with phenoxybenzamine were easily adequate for coitus on the first two fortnightly trials, not quite adequate on the third, and not at all adequate on the fourth and fifth. Six months later I did another CUMRI with phenoxybenzamine, and it again caused erection easily adequate for coitus. In the two papaverine users the deterioration was slight, and they continue to use papaverine successfully.

Non-local effects of papaverine

Two patients fainted within a minute of the injection, one of them twice. Both said they had fainted after other injections, and the faints were probably a reaction to the procedure rather than the drug. No other non-penile effects have been reported after papaverine, except for one report of trembling and sweating after 40 mg, though 80 mg had caused no trouble.

Non-local effects of phenoxybenzamine

Even as low as dose as 2 mg may make a patient aware of an increase in nasal resistance to air flow, although some patients deny it, on direct questioning, after 8 mg. Six patients have had evident postural hypotension; one fainted during a meal 3 hours after an injection of phenoxybenzamine (5 mg), one fainted at home after giving himself an accidental overdose, the exact amount being unknown; one fainted at a railway station 2.5 hours after injection (5 mg); and three patients reported minor episodes of feeling faint when standing, relieved by sitting down. The last five of these patients have continued to use the drug, at lower dosage. Three patients have reported dry orgasms, one on all of six trials and the others once each. Urinary incontinence was reported by one patient on two occasions, probably contributed to by his multiple sclerosis. Headaches, lasting several hours, were reported by one patient after each of seven doses of phenoxybenzamine (4 mg) which gave full erection for 2-4 hours. He then changed to papaverine (80 mg) which caused no headache and gave erection that was only slightly less complete and lasted long enough (1 hour). One patient had two episodes of dyspnoea, each lasting 3 minutes, during the first 12 minutes after injection of phenoxybenzamine (5.5 mg).

Local fibrosis

Repeated insertion of needles into any site might be expected to cause local fibrosis, by analogy with the fibrosis that occurs in muscles into which diabetics inject insulin. I recommend that patients always inject into the same part of the penis, to make it easy to detect fibrosis if it occurs. They are asked to feel from time to time for lumps at this

site, and I palpate the penis whenever I see a patient, i.e. at 6-monthly or (usually) shorter intervals. Patients are asked also to watch for changes in the curvature of the erect penis. No change in curvature has yet been detected in any patient, but one man, after only nine injections of papaverine, developed palpable firmness close to the injection site. He was placed on a waiting list for a penile prosthesis, and this was implanted in August 1985. The fibrosis found was very slight, and caused no difficulty in implanting the prosthesis.

Theoretical risks not yet observed*Infection*

There have been no signs or symptoms of infection after any injection.

Thrombosis

This was to be feared after prolonged priapism, but did not occur. The longest episode of priapism was 40 hours. After this, as after all other episodes, the penis returned rapidly to its normal flaccid size and flexibility. In 10 of the 11 affected patients, full erection occurred subsequently under CUMRI. One patient, who was incompletely impotent, has had no subsequent CUMRI, but he reports that his psychogenic and reflex erections, after being worsened for a few weeks by the episode, recovered and became better than before.

Oncogenesis

Phenoxybenzamine given in large doses by intraperitoneal injection to rats has been reported as causing sarcomata (Flind, 1984). It is approved by the Committee on Safety of Medicines for intravenous injection for very limited indications, but not for the treatment of impotence by intracavernosal injection. The Food and Drugs Administration in the USA does not allow the use of phenoxybenzamine by injection. Recently, further evidence has become available which shows that phenoxybenzamine given orally is carcinogenic in rats (personal communication, Flind, 1986). Consequently, although oral phenoxybenzamine has been in extensive use for many years in man and there has been no evidence of oncogenicity, I have ceased to use phenoxybenzamine for intracavernosal injection except in one patient for whom papaverine gives inadequate erection and who, when I first saw him, had a prostatic carcinoma with metastases.

Papaverine has been little used therapeutically except for CUMRI, and as far as I can discover it has not been subjected to tests of oncogenicity.

Discussion

Most of the users and some of the ex-users have expressed great pleasure at what the treatment has done for them. For a completely impotent man, coitus once per fortnight can be a great improve-

ment on no coitus at all, and for an incompletely impotent man, certainty of successful coitus once per fortnight can be a great improvement on never being certain of success. The fact that 54 men continue to use the treatment shows that they consider the benefits to outweigh the short-term side-effects. Only time will tell whether long-term harmful effects, especially fibrosis, are serious.

Choice and dose of drug

Papaverine is easier for the patient to dispense and has fewer side-effects than phenoxybenzamine. These are sufficient reasons for always trying papaverine first, and using it if it gives adequate erection. However, phenoxybenzamine remains useful in treating a minority of patients, because of its more prolonged action.

One patient gets good erections from 120 mg of papaverine but not from any smaller dose; on the other hand, one patient had priapism after only 18 mg. My present practice is to begin with 16 mg. If this is far from causing adequate erection, the rest of the ampoule (24 mg) is given on the other side 20 minutes later. The dose to be tried at the next consultation is estimated from the degree and duration of erection produced by the 16 or 40 mg given on the first occasion.

From January 1984 to February 1986 I used phenoxybenzamine only for patients in whom papaverine had given erections of insufficient duration. For such patients I began with 4 mg, and was willing to go up to 10 mg if smaller doses gave inadequate erection and no unacceptable side-effects.

Frequency of use

With four patients, I have yielded to their pressure and allowed the limit of 26 injections/year of papaverine to be slightly exceeded. One patient has, with my approval, greatly exceeded the ordinary ration for a single four-week period, but will thereafter not use CUMRI for many months because of his wife's absence. The majority of patients keep close to fortnightly use. A few use CUMRI considerably less often than I tentatively allow as safe. These either have no regular sexual partners or have absent or very defective penile sensation.

Relation between CUMRI and psychotherapy or counselling

Of the 127 patients, 28 have to my knowledge consulted a psychiatrist or psychologist practising sex therapy. They constitute over a third of those

patients without spinal cord injury or disease, and none of those with known spinal cord disorders. I have attempted no psychotherapy. I gave brief counselling to a few, but to the majority none at all beyond that which may have been implied by my questions and factual statements.

I hoped, from the first time I tried this treatment, that successful coitus with the help of CUMRI would improve the confidence of many patients in their sexual performance and that this would allow them to succeed without CUMRI. To my disappointment, only seven patients have improved other than after each CUMRI. In this respect my results are far less good than those reported by Virag *et al* (1983, 1984, 1985a).

Diagnostic significance of success and failure

If CUMRI causes full erection in an impotent man, this proves that the internal iliac arteries and their relevant branches outside the penis itself are adequate to support full erection when other conditions are favourable. It may not exclude atheroma in these vessels as a contributory factor in the impotence. The success of CUMRI presumably excludes cavernoso-venous or cavernoso-spongiosal shunts as the sole or principal cause of the impotence.

Success, even excellent success, does not exclude total destruction of the preganglionic peripheral nervous pathways of erection. One case establishes this. The patient had a complete flaccid paraplegia below T7 on the right and T6 on the left, following a road accident four years before CUMRI was tried. He had no trace of spontaneous or reflex erections; no lower limb, bulbocavernosus or anal skin reflexes; no tonic glandipudendal reflex (Gillan & Brindley, 1979); and no response of any skeletal muscle in the electroejaculation procedure (Brindley, 1981). Nevertheless, CUMRI with phenoxybenzamine (5 mg) caused his penis to stand perpendicular to his trunk, to be flexible only through 10° (i.e. to 170°), and to remain in this state for 70 minutes.

There is no direct evidence as to whether destruction of the postganglionic efferent pathways would prevent successful CUMRI. A theoretical argument suggests that it probably would not: the excitatory innervation of the relevant smooth muscle is mainly α_1 -noradrenergic, as the erectile action of α_1 -adrenoceptor blockers proves. If the innervation is lost, the α_1 -adrenoceptors should continue to be stimulated by circulating catecholamines, and the resulting smooth muscle activity should remain subject to annulment by phenoxybenzamine or papaverine.

Little can be concluded from failure of a single attempt at CUMRI with either drug, since the doses needed by different men differ greatly. If three attempts with increasing doses all fail, one can probably infer that the impotence is due to arterial occlusion or severe stenosis, or to a cavernoso-venous shunt.

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Orofacial Dyskinesia, Cognitive Function and Medication

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The presence of tardive dyskinesia in a sample of 43 patients with schizophrenia and 37 psychopaths who had been hospitalised for many years and exposed to large amounts of medication was assessed while testing their cognitive function. Subjects who showed no evidence of abnormal movements performed significantly better on the test of delayed recall, but there were no differences in performance on any of the other tests of cognitive function used. Multiple regression analysis revealed that age and the total lifetime dose of neuroleptic medication received (in chlorpromazine equivalents) were the only variables to predict the Abnormal Involuntary Movement Scale score, although a large amount of variance in this variable was unaccounted for. The duration of treatment with neuroleptics did not predict AIMS score.

In the thirty years since the introduction of the phenothiazines there have been numerous reports of orofacial dyskinesias attributed to these drugs. There is little doubt that these disorders are now recognised more frequently. In 1955 Mettler & Crandell (1959) found a large number of neurological disorders in psychiatric patients, but no reference was made to any syndrome resembling tardive dyskinesia (TD). This is important because the study was performed before the introduction of the phenothiazines. Crane (1968) observed that 90% of cases of TD had been reported since 1964, and that the majority of sufferers were chronic schizophrenics—especially those who had received physical treatments such as ECT, leucotomy and insulin coma. His impression was that large doses of drugs were responsible, with increasing age also playing a part. Kane & Smith (1982) found the average prevalence of TD to be 20% in neuroleptic-treated patients, compared with 5% in untreated patients. These figures originated from 70 prevalence studies, which overall suggested that increasing age and female sex were the two factors most consistently associated with an increased risk of the disorder.

Despite this, many psychiatrists believe that TD is related to prolonged exposure to neuroleptics, an opinion propounded most forcibly by Klawans (1973), and supported for example by the findings of Smith *et al* (1978) who found a small but significant correlation between TD and total fluphenazine intake in patients over the age of 50. They suggested that dose schedule may be more important than total dose. More recently, Mukherjee *et al* (1982) have demonstrated a significant association between TD and the use of high potency or depot neuroleptics, and also increasing age.

Neurological impairment has long been considered aetiologically important in TD, although as Kane & Smith (1982) suggest there is not much evidence in support of this. Famuyiwa *et al* (1979) found that schizophrenic patients with TD performed less well on a paired associate learning test than those who did not have the disorder, although there were no differences between the two groups on other tests of cognitive function, nor in indices of cortical atrophy on computerised tomography (CT) scan. Similarly, Jeste *et al* (1980) in their CT scan study were unable to find evidence of cortical atrophy in schizophrenic patients with TD.

There are many problems in comparing the results of different studies. Definitions of TD differ in the extent to which a pure bucco-lingual-masticatory triad is specified, or in whether the tremors of parkinsonism or axial and limb movements should be included. Different selection criteria are used, so that experimental groups differ in age, duration of illness and treatment, and environmental factors which may influence the assessment of abnormal movements through anxiety and arousal. Most studies have been based on limited drug history data, relying either on medication at the time of the study or on detailed information collected over limited periods of time. Given the difficulties in determining precisely the total amount of medication received during the course of an individual's illness, a more detailed investigation of chronic neuroleptic administration in relation to TD is particularly important at a time when the use of such medication is being questioned. This study attempts to do this, and also to investigate the relationship between TD and cognitive impairment, by examining the following hypotheses:

1. Patients who exhibit TD perform less well on tests of cognitive function than patients without the disorder.

2. Using multiple regression analysis the total cumulative dose of neuroleptics can be used to predict TD score.

Method

Patients were selected from two sources: four long-stay wards in the Royal Edinburgh Hospital, and four long-stay wards in the State Hospital, Carstairs. All contained stable populations of male long-stay patients. The case notes of all patients were examined to exclude those over the age of 65 and those with a history of alcohol abuse, head injury, leucotomy, serious physical illness, organic psychosis, or mental subnormality. The research diagnostic criteria of Spitzer *et al* (1972) were applied to the remaining subjects, and the duration of admission, number of ECTs, and years of education obtained from their case notes. Detailed drug information was obtained from prescription sheets, which were available (for all but four subjects, who were excluded) as far back as the original admission. These were checked against entries from the medical notes and nursing notes, ensuring that dates and amounts of medication prescribed were as accurate as possible. Occasional medication was not recorded. For each of the six most widely prescribed neuroleptics (chlorpromazine, trifluoperazine, thioridazine, fluphenazine decanoate, haloperidol and flupenthixol decanoate) the starting date, dose, date of change of dose and date of discontinuation were noted. This information was summarised by a computer program which calculated the total equivalent dose for all drugs, using chlorpromazine equivalents (Davis, 1974).

Mental state assessments were performed using Wiles' (1976) glossary for the Brief Psychiatric Rating Scale (BPRS) of Overall & Gorham (1962), and the Abnormal Involuntary Movement Scale (AIMS) (Guy, 1976) was used to measure movement disorders on two occasions a month apart. This is a five-point rating scale measuring abnormal involuntary movements in seven areas, including the muscles of facial expression, tongue, jaw, lips, upper and lower extremities, neck, shoulders and hips; subjects were thus rated for such movements in body areas other than the orofacial region. All measurements were made 60 seconds after the subject had started a motivation test, and continued over a 60 second period. The test consisted of a boring and repetitive task in which subjects were given a page on which the letter X was printed more than 1000 times; they were then asked to circle as many as they wished, the score being the number circled before stopping. The purpose of this test was to assess the extent to which volitional impairment—an important clinical component of chronic schizophrenia—affected performance on the tests of cognitive function. The AIMS ratings were therefore made under standard conditions, with subjects unaware that they were being observed. Following this, the Paired Associate Learning test described by Inglis (1959), Williams' test of delayed recall (Graham-White, 1969), and Raven's (1960) progress-

TABLE I
Prevalence of TD by diagnosis

	Schizophrenics	Psychopaths
Non-TD	7	22
TD	36	15

Corrected $\chi^2 = 14.23$, $P < 0.001$

ive matrices were given to all subjects. Data were analysed using the Statistical Package for the Social Sciences.

Results

Test/re-test reliability for the AIMS was satisfactory (Pearson's $r = 0.87$, $P < 0.001$). The presence or absence of movement disorder was established on the basis of the AIMS score over the two test periods; subjects with a total score of two or less were classified as having no abnormal movements. Eight subjects were excluded because they could only be tested on one occasion, leaving 25 subjects with no TD and 43 with TD. Although TD was more common in patients with schizophrenia a small number of psychopaths exhibited the disorder (Table I). A univariate model was used to identify relationships between TD and demographic data, treatment and test variables. With the exception of the total equivalent dose and amount of ECT received, which were both highly skewed, the distribution of all the dependent variables did not differ significantly from that of a normal distribution using the Kolmogorov-Smirnov 'goodness of fit' test. When the medication variable was transformed by taking its square root, it conformed more closely to a normal distribution.

Table II compares the demographic characteristics of the two groups. The TD group were significantly older, had been in hospital much longer, and had received more medication and ECT than the non-TD group, who had fewer psychiatric symptoms and performed significantly better on the motivation test than the TD group. On all three tests of cognitive function the TD group performed less well than the other group, although the differences were only significant for the delayed recall and its age-adjusted memory quotient. The greater age of the TD group appears to account for their performing less well on the progressive matrices; when the age-adjusted IQs are compared the differences disappear (Table III).

Discussion

These results are in accordance with other studies which suggest that TD is associated with increased age, longer hospitalisation, and exposure to large amounts of neuroleptics and ECT. As this is one of few studies to have attempted a direct measurement of the total intake of drugs, it is important to consider some of the weaknesses of this measure.

TABLE II
Subject characteristics

	<i>Non-TD (n=25)</i>		<i>TD (n=43)</i>		<i>F ratio</i>	<i>Significance</i>
	<i>Mean</i>	<i>s.d.</i>	<i>Mean</i>	<i>s.d.</i>		
AIMS score	0.39	0.60	8.55	4.94	96.79	<0.001
Age (years)	37.0	7.2	50.5	10.2	33.80	<0.001
Years of admission	10.2	4.6	21.2	10.6	23.74	<0.001
Years of education	9.5	2.9	10.6	2.1	3.07	NS
STE ¹	775	1477	3127	2397	19.41	<0.001
Total no. of electro-shocks received ²						
Mean	0.90		9.2			
Mean rank	32.4		44.0			

1. Square root of the total equivalent dose of neuroleptics received.

2. Mann-Whitney $U=500$; $P=0.0125$ (two tailed).TABLE III
Test results

<i>Test</i>	<i>Non-TD</i>		<i>TD</i>		<i>F ratio</i>	<i>Significance</i>
	<i>Mean</i>	<i>s.d.</i>	<i>Mean</i>	<i>s.d.</i>		
Brief Psychiatric Rating Scale	2.3	3.7	6.6	5.5	12.00	<0.001
Motivation test total score	434	377	234	300	1.04	NS
Paired Associate Learning test	19.8	15.8	26.1	16.9	2.33	NS
Delayed recall	4.6	4.4	11.0	9.2	10.50	<0.01
Memory quotient	113	19.3	97	18.7	11.12	<0.01
Progressive matrices	33.9	10.4	22.0	11.2	18.83	<0.01
IQ	96	13.1	93	11.7	1.05	NS

TABLE IV
Stepwise multiple regression with AIMS score as the dependent variable

<i>Variable</i>	<i>R² change</i>	<i>F ratio</i>	<i>Significance</i>
Age	0.22	17.55	<0.001
STE	0.10	9.05	<0.01
Total no. of electro-shocks received	0.02	2.19	NS
BPRS score	0.01	1.00	NS
Time on drugs	0.01	0.58	NS
Duration of hospital stay	0.00	0.31	NS
Motivation test total score	0.00	0.06	NS

TABLE V
Stepwise hierarchical multiple regression with AIMS score as the dependent variable

	Variable	R ² change	F ratio	Significance
Step 1	Age	0.21	20.25	<0.001
Step 2	STE	0.10	10.99	<0.001

Although the prescriptions were checked for accuracy against two other sources of information, there was no way of checking that subjects had actually taken the prescribed medication. The only exceptions to this were the depot neuroleptics; however, the cumulative totals of fluphenazine and flupenthixol together accounted for only 16% of the total equivalent dose, so it must be assumed that the measure of oral medication was open to distortion because of the possibility that subjects might have failed to take it from time to time. It has to be assumed that non-receipt of prescribed medication was randomly distributed throughout the sample.

The fact that the TD group were older could account for them having received more medication, the link between TD and medication being secondary to the more important relationship between age and TD. At this stage multivariate techniques were used, with the AIMS score of all 68 subjects as the dependent variable in two multiple regression analyses. Initially, the variables used to predict the AIMS score were age, motivation test total score, total equivalent dose (square root), total time (years) on medication, duration of admission, BPRS score, and amount of ECT received. For each regression, solution plots of the residual values showed no evidence of non-linearity of the relationship.

The results of the first regression are given in Table IV; the best predictors of the AIMS score were age and total equivalent dose. To investigate the relationship further it was hypothesised that age was the most important variable, the value of the total equivalent dose as a predictor of the AIMS score being dependent on its own relationship with age. These variables were entered together in a stepwise hierarchical regression with age on the first step, and the square root of the total equivalent dose (STE) on the second. The result (Table V) reveals that both age and STE predict the AIMS score, age alone accounting for 21% of the variance, and STE accounting for a further 10%. This suggests that age and total medication are associated with tardive dyskinesia, but the interpretation of this regression should be tempered with caution. About one-third of the total variance in the AIMS score is explained

by these two variables, indicating that other factors unaccounted for in this study may make a considerable contribution. No attempt was made to measure the amount of anti-parkinsonian medication received by the subjects. TD may result not only from prolonged dopaminergic blockade, but also from the additional prolonged use of anticholinergic drugs. A further difficulty relates to the fact that subjects were assessed for TD while on medication, raising the possibility that the AIMS score is not representative of the severity of TD.

The poor performance of the TD group on the tests of delayed recall is further evidence of an association between TD and cognitive impairment. The results suggest that with increasing age and medication there is a greater likelihood of developing TD. However, the fact that the age-adjusted memory quotients were significantly worse in the TD group implies that the different amounts of medication received by the two groups, rather than age, may explain the differences in delayed recall. Anticholinergic drugs may produce confusion and memory impairment, especially in elderly subjects. Most patients in this study received such drugs to control extrapyramidal side-effects, although their use was not measured, and most neuroleptics possess anticholinergic properties to a varying extent. It is possible to speculate that the combined anticholinergic properties of neuroleptics and anti-parkinsonian medication to which the subjects were exposed might explain the relationship between total equivalent dose and delayed recall performance, although it must be stressed that this is speculative, given the absence of measures of anticholinergic activity in this study.

Although the degree of short-term memory impairment in these subjects was not great, it is important to bear this in mind when considering the need for long-term medication in a population who already have a multiplicity of handicaps. Caution should be exercised when contemplating the use of large doses of neuroleptics, especially with anti-parkinsonian medication in elderly subjects. The routine prescribing of combinations of such drugs should be questioned.

Conclusions

This study has shown that tardive dyskinesia may be associated with poor short-term memory performance. No relationship emerged between TD and the duration of exposure to medication, although both age and total amount of medication were found to predict the presence of TD. However, this study assessed dyskinesia when the subjects were still on medication. As it is quite possible that neuroleptics mask the manifestation of TD, this, together with the absence of any data concerning anti-parkinsonian medication, may have been responsible for the large amount of variance in the AIMS

score not explained by age and STE. This problem can only be overcome by examining subjects before and after their medication has been discontinued, and obtaining more detailed information on the prescription of anti-parkinsonian medication.

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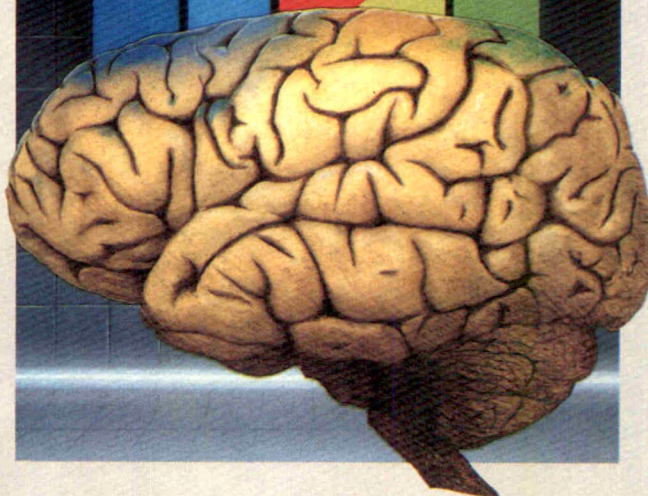
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A SCHIZOPHRENIC.

Adolf Wölfli spent 35 years in the Waldau Mental Hospital, Berne, suffering from schizophrenia. During that time he produced many drawings that have since gained widespread acclaim.

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The Role of Zinc in Senile Dementia

C KENN and E. GIBB

Zinc deficiency has been reported in association with dementia and linked with its pathogenesis. A group of 45 elderly patients admitted to a mental hospital were given diagnoses in accordance with ICD-9, and their fasting plasma zinc levels were recorded. No difference was found in zinc levels between patients with diagnoses of senile dementia and those with other diagnoses.

Previous studies have shown the relevance of plasma zinc levels to psychiatric symptomatology, including acute syndromes associated with the administration of histidine (Henkin *et al*, 1975), prolonged total parenteral nutrition (Kay *et al*, 1976), acrodermatitis enteropathica (Nelder & Hambridge, 1975), and the continuing debate on the relationship of zinc to anorexia nervosa (Casper *et al*, 1980; Bryce-Smith & Simpson, 1984).

Srinivasan (1984) has summarised the role of trace metals in psychiatry, and Tiggelen (1983) reviewed the work linking zinc in the form of metallo-enzymes as a co-factor in the pathogenesis of dementia. However in much of the literature the diagnosis of dementia appears to have been based solely on the presence of cognitive impairment. The present study attempted to compare plasma zinc levels in patients with a diagnosis of senile dementia, using the ICD-9 definition (World Health Organisation, 1975), with those of a control group of patients with ICD-9 diagnoses.

Method

The sample comprised 45 patients over the age of 65, recently admitted to a mental hospital. They received a formal psychiatric interview, full cognitive assessment, and physical examination, and were given an ICD-9 diagnosis. Those with a diagnosis of dementia were rated on the Hachinski scale for arteriosclerotic dementia (Hachinski *et al*, 1974); in the absence of abnormal biochemical profiles, scores of less than 4 were taken as indicative of senile dementia and those of greater than 7 as of arteriosclerotic dementia. The Hachinski scale was thus used as an adjunct to ICD-9 to refine the diagnosis of senile dementia.

Fasting blood samples with minimal stasis were taken for a routine chemical screening, and for measurements of plasma zinc and plasma vitamin E levels (the latter is involved in the same oxidative pathways as zinc: the results of this part of the study will be reported in a later paper). Plasma zinc levels were measured using atomic absorption spectrophotometry (Whitehouse *et al*, 1982). The authors were blind to the results of the plasma zinc levels during the study.

Results

During the study period, eight patients were excluded for the following reasons: readmission (4), refusal to allow venepuncture (2), immediate hospital transfer (2), of the remaining 45, 22 (18 female, 4 male; mean age 81.1 years) met the ICD-9 diagnosis of senile dementia (ICD-290.0). The comparison group (17 female, 6 male; mean age 75.3 years) had the following ICD diagnoses: manic-depressive states (ICD 296; $n=13$), arteriosclerotic dementia (ICD 290.4; $n=2$), paraphrenia (ICD 297.2; $n=2$) acute confusional state (ICD 293.0, $n=2$), organic psychosis unspecified (ICD 290.9 $n=2$) residual schizophrenia (ICD 295.6; $n=1$), and Korsakov's psychosis (ICD 291.1, $n=1$).

The numbers in each group receiving drugs affecting zinc bioavailability (i.e. steroids, diuretics, and lithium) were comparable. No patient suffered acute infection or trauma. One patient in the senile dementia group had malignant disease, compared with two in the control group. No patient had evidence of diabetes mellitus.

The plasma zinc results are shown in Table I. Analysis was undertaken using the Wilcoxon unpaired test; the difference between the senile dementia sample and the comparison sample was not statistically significant.

Discussion

The total body content of zinc is about 2g, 80% of which is bound intracellularly in metallo-enzymes and cell membranes. Those at risk of developing deficiency are diabetics, alcoholics, pregnant and lactating women, and the elderly (Tiggelen, 1983).

TABLE I
Plasma zinc measured in sample groups: $\mu\text{mol/litre}$

	Senile dementia ($n=22$)	Functional and other ($n=23$)
Median	10.45	11.20
25th-75th percentiles	9.3-12.5	10.1-12.5
Mean	10.92	11.21
Range	8.3-17.7	9-13

TABLE II
Comparison of results with literature results

Study	No. of patients	Mean plasma zinc level $\mu\text{mol/litre}$	s.d.
Present study			
Senile dementia	22	10.92	2.26
Other diagnoses	23	11.21	1.27
Hullin (1983) ¹			
Dementia			
Male	64	13.6 ²	2.6
Female	176	13.6 ²	2.6
Healthy control			
Male	22	15.9	3.1
Female	54	14.5	1.8
Tigellen (1983)			
Dementia (DSM-III)	23	15.81 ²	1.78
Healthy elderly control	28	18.05 ²	1.85
Bunker (1984)			
Healthy elderly	24	11	1.2
Healthy Adults	50	13	1.8

1. Long stay mental hospital (all ages).

2. Serum zinc levels.

Although widely used, doubt has been cast on the validity of plasma zinc as a sole indicator of total body zinc status (*British Medical Journal*, 1981); Abdulla (1983) concluded that liver zinc may be a more accurate parameter of deficiency. Prasad (1983) states that plasma zinc is a useful indicator, provided the specimen is not haemolysed or taken in conditions of acute stress or infection. As plasma zinc is mainly protein-bound, levels of albumin and binding protein (α_2 macroglobulin) will affect estimations. No patient in this study had an elevated white cell count (taken as indicative of infection) or hypo-albuminaemia. In a younger age-group (23–62 years), Halsted & Smith (1970) found no instance of an elevated plasma zinc level in a variety of disease states.

The normal level of plasma zinc in adults is 12–22 $\mu\text{mol/litre}$; recent work has suggested mean levels in healthy elderly adults of 11 $\mu\text{mol/litre}$ (s.d. 1.2) and in healthy adults of 13 $\mu\text{mol/litre}$ (s.d. 1.8 (Bunker *et al.*, 1984). The levels in the present study compare well with Bunker's elderly group (Table II). The work of Kosman *et al.* (1979) showed a small but not statistically significant difference between plasma and serum zinc. Direct comparisons between studies using either measure are thus valid.

Burnet (1981) hypothesised a genetically based,

age-related progressive inability of neurones to incorporate zinc ions into DNA-handling enzymes, leading to an Orgel 'error catastrophe', producing defective protein synthesis and cell death. Thus 'pathological ageing' occurs. This has led to the current research interest in the role of zinc as a co-factor in the aetiology of senile dementia.

Previous workers have found evidence of lowered zinc levels in patients with dementia. In a study of recently admitted elderly patients with dementia, compared with those with depression, Hullin (personal communication) found significant lowering of plasma zinc in patients with dementia, with the albumin-bound fraction accounting for this difference. The serum levels were of an order similar to the present study. When screening a chronic hospital population, Hullin (1983) found a significant lowering of serum zinc in patients with dementia compared with patients with schizophrenia and affective disorder and controls (Table II).

In neither of these studies, however, were the types of dementias classified. Hullin also noted (personal communication) that patients with dementia in the latter study had average body weights lower than the other patients and some 20 kg lower than normal elderly people living in the community. This raises the possibility that the drop in serum zinc

could be concurrent with a nutritional defect or be an abnormality of metabolism as a consequence rather than a cause of dementia.

Tiggelen (1983) found similar results (Table II) in acutely admitted dementia patients (according to DSM-III classification), compared with healthy controls, but 70% of these had abnormal dexamethasone suppression tests. He therefore questioned the diagnosis of DSM-III dementia in his study group. He also suggested a superimposed undiagnosed depressive illness, and postulates that an abnormality of the hypothalamic-pituitary adrenal axis caused by, or could cause, zinc deficiency.

There are no obvious reasons why the levels of plasma and serum zinc quoted in the various studies differ so widely. The most comparable study to this one is that by Hullin (personal communication) and, aside from the differences in classification mentioned, it is unclear why the two studies have produced contrasting results. Further research into this area is clearly warranted.

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Temporary Urinary Incontinence in the Acute Psychiatric Patient Without Delirium or Dementia

G. E. BERRIOS

In a prospective study of the 295 admissions in one year to an acute psychiatric ward, 14 subjects without delirium or dementia were found to have developed temporary urinary incontinence. When compared with matched controls ($n=56$) the incontinent patients were found to be suffering from psychosis ($P<0.0002$) and to have a history of childhood enuresis ($P<0.01$). Compared with psychotic controls ($n=22$), the incontinent patients were older ($P<0.05$), they had been exposed to a greater variety of treatments ($P<0.01$), and they had received more thioridazine ($P<0.04$).

It is not a rare clinical observation that psychiatric patients without delirium or dementia may exhibit episodes of urinary incontinence during the acute stage of their condition. The symptom has a good prognosis. Clinicians tend to explain this temporary disorder of micturition in terms of disturbed and disinhibited behaviour, stupor, psychotic disorientation, aggression or psychodynamic regression. Although of some relevance, it is unlikely that these factors are more important than variables such as central neurotransmitter dysfunction (Ambrosini, 1984), medication (Van Patten *et al.*, 1973; Nurnberg & Ambrosini, 1979) and medical history.

This paper reports a study designed to identify the clinical characteristics of acute psychiatric patients not affected by delirium or dementia who develop temporary urinary incontinence during the acute phase of their illness.

Method

This prospective study was carried out in a 30-bed general admission ward in Fulbourn Hospital, Cambridge, with a catchment population of about 125 000. There were 295 admissions during the period covered by the study (1 January 1984-31 December 1984).

Patients who met the following entry criteria were selected for the incontinent sample:

1. At least three episodes (diurnal or nocturnal) of urinary incontinence, defined as 'involuntary loss of urine constituting a social or hygienic problem' (Isaacs & Walkey, 1964)
2. The three episodes to have occurred within a period of no more than 48 hours
3. Absence of acute confusional state or dementia (ICD9)

A control sample of 56 patients was selected from the total cohort ($n=295$). As soon as a patient met the criteria

for urinary incontinence, four other patients were chosen as his/her controls from amongst fellow patients, according to the following criteria:

1. No clinical evidence of acute confusional state or dementia
2. Admission date no more than one week before or after that of the incontinent patient

If more than the needed number met the criteria, the four control patients were selected randomly.

A specially designed data sheet was completed for both the incontinent patient and his/her four controls. The form collected data on five areas: general, medical history, psychopathology, ICD9 diagnosis (as made by the consultant in charge), and treatment. Statistical analysis of the data was carried out by means of the Unistat Software Package for CPN.

Results

Fourteen patients (4.74%) met the inclusion criteria for urinary incontinence. There was no difference in age, sex, presence of urinary disease, parity, orthostatic hypotension or previous history of adult incontinence between the experimental group and the control group of 56 patients (Table I). However, the incontinent sample showed a higher proportion of patients with a history of childhood enuresis ($P<0.01$; Table I) and a higher proportion suffering from psychotic illness ($P<0.0002$; Table II).

There were 22 psychotic patients amongst the 56 controls, and this psychotic sub-group was compared with the incontinent set. No differences were found between the two groups in terms of family history of psychiatric illness, number of previous episodes (Point biserial correlation coefficient, $rpb=0.0012$; NS), duration of present episode ($rpb=0.0091$; NS), distribution of diagnoses (Table II), or symptomatology (Table III). However, the incontinent patients were significantly older ($rpb=0.28$; $t=1.75$; $P<0.05$) and had received a greater variety of treatment

TABLE I
Age, sex and medical history of 14 temporarily incontinent patients
compared with 56 control patients

	Incontinent (n = 14)	Controls (n = 56)	χ^2
Age (mean $\pm$ S.D.)	51 $\pm$ 9	42 $\pm$ 14.2	NS
Sex	8M/6F	37M/19F	NS
Urinary disease	3	2	NS
Diabetes mellitus	1	0	NS
Heart disease	2	3	NS
Abdominal surgery	2	3	NS
Cerebrovascular accident	2	2	NS
Parity (mean $\pm$ S.D.)	1 $\pm$ 1.2 ⁽¹⁾	1.5 $\pm$ 1.3 ⁽²⁾	NS (t)
Childhood enuresis	5	4	5.8*
Previous incontinence	2	1	NS

* $P < 0.01$.

⁽¹⁾ 6 incontinent women had had children.

⁽²⁾ 14 of the female controls had had children.

TABLE II
ICD9 diagnoses of 14 temporarily incontinent patients compared with those of control patients

ICD9 diagnosis and code	Incontinent (n = 14)	Controls	χ^2
<i>Psychotic disorders</i>	14	22	14.18**
Paranoid schizophrenia (295.3)	2	5	NS
Manic-depressive (manic phase) (296.2)	5	8	NS
Manic-depressive (depressive phase) (296.3)	6	9	NS
Alcoholic hallucinosis (291.3)	1	0	NS
<i>Non-psychotic disorders</i>	0	34	
Personality disorder (301.5/301.7)	0	11	
Hysteria (300.1)	0	1	
Neurotic depression (300.4)	0	12	
Phobic state (300.2)	0	4	
Other conditions ¹	0	6	

** $P < 0.002$.

¹Alcoholism (n = 3); chronic pain (n = 2); abnormal grief reaction (n = 1).

($P < 0.01$; Table IV). More of them had received thioridazine ($P < 0.04$; Table V) and the dosages prescribed tended to be larger ($t = 2.155$; just short of $P < 0.05$).

Discussion

The entry threshold for urinary incontinence used in this study was sufficiently high not to allow into the sample patients with occasional urinary loss. None of the 14 incontinent patients were suffering from delirium or dementia (each patient was examined by

at least two consultant psychiatrists). Three were found to be disorientated during the acute stage of their condition (two depressive and one manic); there was no evidence of organic aetiology, however, and the symptom cleared up after treatment. On discharge all subjects had recovered from both the psychiatric episode and the urinary incontinence.

The first conclusion of clinical value is that psychotic patients are more prone to developing temporary urinary incontinence than non-psychotic

TABLE III
Clinical features of 14 incontinent patients (all psychotic) compared with those of 22 psychotic control patients

	<i>Incontinent (n = 14)</i>	<i>Psychotic controls (n = 22)</i>	χ^2
Disorientation	3	0	NS
Hallucinations	4	2	NS
Delusions	9	9	NS
Depression	8	9	NS
Retardation	4	3	NS
Suicidal ideation	8	6	NS
Agitation	6	7	NS
Stupor	2	1	NS
Disinhibition	3	5	NS
Hyperactivity	6	9	NS
Acting out	4	3	NS
Drowsiness	6	8	NS
Duration of illness (days) ¹	27 ± 6.1	28 ± 11.3	NS (t)
No. of past episodes ¹	2.36 ± 2.2	2.20 ± 2.71	NS (t)

¹Mean ± S.D.

TABLE IV
Mean dosage of ECT (number of treatments) and drugs (dose in milligrams) for 14 incontinent patients (all psychotic) compared with 22 psychotic controls. TTS (Total Treatment Score) is the sum of the patient's individual treatment scores (1 point for each drug or ECT)

	<i>Incontinent (n = 14)</i>	<i>Psychotic controls (n = 22)</i>	t
Chlorpromazine	1650 (SD = 1222)	1500 (SD = 828)	NS
Thioridazine	690 (SD = 292)	337 (SD = 159)	NS
Haloperidol	114 (SD = 13)	80 (SD = 70)	NS
Tricyclic A-D	375 (SD = 129)	325 (SD = 172)	NS
Phenelzine	150 (SD = 25)	97 (SD = 53)	NS
ECT	4 (SD = 1.4)	3 (SD = 1)	NS
Lithium	3400 (SD = 309)	3000 (SD = 489)	NS
Benzodiazepines	64 (SD = 32)	48 (SD = 24)	NS
TTS	2.71 (SD = 0.91)	1.77 (SD = 0.86)	3.092*

* $P < 0.01$.

patients; the second is that the type of psychosis does not seem to be a relevant factor; the third is that accumulation of treatments (irrespective of their nature) seems to be related to the appearance of urinary incontinence. However, the conclusion that there is a cause-effect relationship between

an accumulation of treatments and incontinence, although tempting, cannot be drawn from our data. The alternative hypothesis is that such an accumulation simply reflects the severity of the psychotic illness and that the latter is ultimately responsible for the micturition disorder. It must be remarked,

TABLE V

Past and current treatment of 14 incontinent patients (all psychotic) compared with that of 22 psychotic controls: figures indicate number of patients receiving each treatment

Treatment	Incontinent (n=14)	Psychotic controls (n=22)	χ^2
Chlorpromazine	6	5	NS
Thioridazine	6	2	3.85*
Haloperidol	5	8	NS
Tricyclic A-D	3	7	NS
Phenelzine	3	2	NS
ECT	2	3	NS
Lithium	6	7	NS
Benzodiazepines	7	5	NS

* $P < 0.04$.

however, that no difference was found between the incontinent group and the control sample in terms of family history of psychiatric illness ($\chi^2 = 1.40$; NS), number of previous episodes ($rpb = 0.00129$; NS), duration of current admission ($rpb = 0.00919$; NS) or symptomatology.

Thioridazine had been prescribed more often to the incontinent sample ($P < 0.04$). An association

between this neuroleptic and an increased incidence of urinary incontinence in psychiatric patients has been reported anecdotally (Ananth *et al*, 1971; Renshaw, 1971; Crittenden, 1972) but no prospective study of this association seems to have been published.

It has recently been suggested that the central control of micturition depends upon a noradrenergic-dopaminergic balance, with dopamine agonists increasing control and antagonists increasing the likelihood of incontinence in predisposed individuals (Ambrosini, 1984). Why thioridazine should have a more marked effect than other neuroleptics is unclear.

Developmental delays in the acquisition of control of micturition have been mentioned as a major vulnerability factor; it is therefore of some interest that the present study found that a significantly higher number of incontinent patients had been enuretic during childhood. Orthostatic hypotension has also been mentioned as a concomitant symptom of urinary incontinence but our study showed no difference in this regard between the incontinent and the control groups.

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Depression in Schizophrenia: A Study of Prevalence and Treatment

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In an investigation of white, coloured, and black patients admitted to a psychiatric hospital, the prevalence and treatment of depression in schizophrenia was assessed and found to be 30% in group of acute, nuclear schizophrenics. While the prevalence was similar in the three groups, depression was clinically under-detected in black patients.

The presence of depression among schizophrenics has been recognised since the times of Kraepelin and Bleuler, and many clinicians feel it is an integral part of the illness (Knights & Hirsch, 1981; Planansky & Johnston, 1978). Though reports of the prevalence of depressive symptoms vary, recent evidence suggests that it is considerable (ibid and Roy *et al.*, 1983), and that there is a significant suicide risk among depressed schizophrenics. As such, it warrants more attention by both clinicians and research workers (Donlon *et al.*, 1976). An opportunity for a detailed examination of this phenomenon arose out of an investigation of sequentially selected acute schizophrenic patients admitted to a psychiatric hospital in Cape Town, South Africa (Teggin *et al.*, 1985).

As this group included white, coloured, and black (as defined by the Population Registration Act, 1950 (as amended)) patients, it was possible to focus on the issue of depression among blacks. There is diversity of opinion about the prevalence of depression in this group; early reports (Buchan, 1969; Prince, 1968) seemed to indicate that it occurred rarely, although recent evidence contradicts this (Leff, 1973; Singer, 1975).

The aims of this investigation were to determine the prevalence of depression among a specific group of acute schizophrenics; the extent to which depression was clinically treated; and whether the black groups differed from the other two in terms of both prevalence and treatment of depression.

Method

The Present State Examination (PSE) and the CATEGO computer programme (Wing *et al.*, 1974) were used both to select patients for inclusion in the present analysis and to assess their mental state. Only those schizophrenics with a CATEGO class 'S' diagnosis, i.e. characterised by either the presence of first-rank symptoms or other unequivocal symptoms of schizophrenia, were selected. Patients with coexisting organic conditions, e.g. epilepsy or drug or alcohol abuse, were excluded. Thus a circumscribed group with a clearly defined schizophrenic disorder was focused upon. The total sample comprised 56 patients (19 white, 15 coloured, and 22 black).

The PSE was conducted by a research team within three days of the patient's admission to hospital, while the patient's clinical diagnosis and management remained in the hands of the responsible clinician.

It is often difficult to distinguish between depressive symptomatology and certain symptoms of schizophrenia (Donlon *et al.*, 1976; Knights & Hirsch, 1981). For example, both depressed and schizophrenic patients may exhibit inefficient thinking or social withdrawal. It was therefore decided to select a sub-set of PSE symptoms—depressed mood (symptom 23) combined with observed depression (symptom 121)—to obtain a finer indicator of depression.

Information on the treatment of depression (using anti-depressants or ECT) by clinicians was obtained from an examination of the patients' hospital folders.

Results

Using the sub-set of depressed mood combined with observed depression, 30% of all the patients were found to be depressed, with no significant differences between the ethnic groups; 16% expressed suicidal ideas or acts (symptom 25).

While depression was treated by clinicians in 59% of the depressed group, further analysis revealed a significant difference between the black and the other two groups: 82% of whites and coloureds, compared with only 17% of blacks, were treated for their depression ($P < 0.05$; Fisher's Exact Test). The possibility that clinicians were using other criteria (such as the vegetative symptoms of depression) to detect depression in the black patients was considered. However, as only three black patients were treated they constituted too small a group for any clear patterns to emerge.

Discussion

Despite the small sample size, this investigation had the advantage of a methodology that enabled independence of the clinical and research judgements. The prevalence of depression found in this study confirms that a substantial number of acute schizophrenics are sufficiently depressed to warrant treatment. Similarly, the relatively high rates of suicidal ideation emphasises the need for an awareness of the risk of suicide in schizophrenia. The rate of treatment of depression by clinicians is similar to

that reported by previous investigations (Knights & Hirsch, 1981).

The finding that depression occurred as frequently in black patients as in other groups confirms recent related evidence, and is an important finding with obvious clinical implications. Despite this similarity, the low percentage of depressed blacks that were treated by the clinicians warrants some discussion. As the majority of clinicians do not speak Xhosa (the language spoken by the majority of black patients in the Cape), interpreters are vital to the understanding of the patient. The shortage of interpreters in the hospital frequently results in clinicians spending limited time with the interpreter, or having to interview black patients without one. Therefore, in dealing with schizophrenic patients, clinicians are likely to focus on psychotic symptomatology and

not investigate other symptoms in any depth. In addition, since many black patients may use metaphorical or somatic terms to describe feelings of depression (Gillis *et al.*, 1982), it is possible that the clinicians do not attempt to elicit depressive symptoms. This study, however, illustrates that not only are depressed mood and observed depression present in black schizophrenics, but that with the use of skilled interpreters these symptoms are ascertainable.

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Electro-Oculogram Changes at the Switch in a Manic-depressive Patient

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The correlations between eye movements on the EOG of a patient with a 48 hour cycle of manic-depressive type are described. They are used to confirm the fact that his switch from one state to another occurred at the same time of night whether he was awake or asleep.

Eye movements with a frequency of $\frac{1}{2}$ –1 Hz have been related to mood state in some patients with periodic psychoses, in whom it can be a good indicator (Gjessing *et al*, 1967; Stones *et al*, 1980a,b). This paper presents studies on one manic-depressive psychotic patient, who had a persistent and predictable 48 hour cycle. Information on this patient has been published previously (Goodwin *et al*, 1964; Harding *et al*, 1964, 1965, 1966; Jenner *et al*, 1967, 1968), following a decade of observations by clinical staff and over 300 electrocephalogram (EEG) recordings. However, some details of the EEG and electro-oculogram (EOG) findings have never been fully described, and may have more general interest, as well as providing information on factors relevant to less extreme and predictable illnesses. In particular, they illustrate that a timing mechanism is involved, but one which is independent of being awake or asleep.

Methods

The patient was studied between the ages of 48 and 58. He had a precise 48 hour cycle of manic-depressive type, which had persisted for 11 years, but was not on medication for most of the period of this study. The method of mood scoring was described by Jenner *et al* (1967). Nursing staff rated the patient's mood as follows: abusive, constantly shouting, quarrelsome and demanding (+3); abusive, expresses paranoid ideas and talks loudly (+2); overactive and overtalkative but good-natured (+1), normal or difficult to assess (0); quiet but makes spontaneous remarks (–1); lethargic, responds to remarks and reads the paper occasionally (–2); almost totally inactive but responds to direct approaches (–3).

Eye movement recordings were obtained using an EEG machine (time control 0.3 s high frequency 70 Hz, amplitude 100 μ V/cm). Electrodes were attached to the frontal and occipital areas to record the sleep stages, and to the orbit areas for the EOG. The montage used was the standard 10–20 system; channels 1, 2, 3 and 4 were respectively Fp1–F8, Fp2–F7, F2–C2, and C2–O2 (mid-occipital). Hence, at night, eye movements were recorded from the leads on the orbits and two EEG channels were taken from the frontal and occipital areas. A fifth channel was recorded for an electromyogram and used to confirm the stages of rapid eye movement (REM). Sleep latency and dream latency were assessed, and scored according to international criteria (Rechtschaffen & Kales, 1968).

Results

The scores of mood were found to be extremely predictable and reliable, with the patient in mania one day and depression the next; the mood change-over occurred at night. There was a persistent pattern of falling asleep later, when manic on retiring (sleep began later than 2300 hrs), and earlier when depressed (before 2300 hrs). The dream

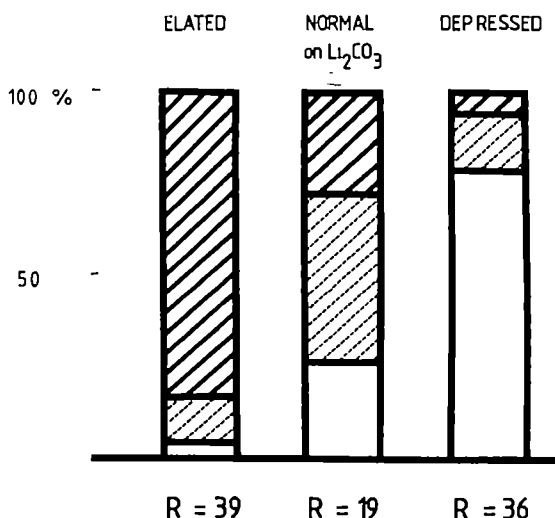


FIG 1 The results of 'blind' classification of EOG records into those dominated by (cross-hatched), showing some (cross-hatched with dotted lines) and showing no (blank) $\frac{1}{2}$ –1 Hz eye movement. R is the number of records assessed from each mood state.

delay in this patient was 15.1 min (sd 8.6), which is shorter than usual (Dement & Kleitman, 1957; 67 min; Oswald & Thacore, 1963: 1 hr; Feinberg *et al*, 1967: 69 min; Kahn & Fisher, 1969: 59 min). There was no difference at the 5% confidence level between the dream delays in the manic and depressive moods (mania 13.35 min, sd 7.1, depression 16.6 min, sd 9.8).

The EOG was dominated by $\frac{1}{2}$ –1 Hz eye movements when the patient was awake and in mania, but their presence in the EOG was negligible when he was depressed. Fig. 1 shows the relationship between presence of $\frac{1}{2}$ –1 Hz eye movements (assessed blindly) and mood. The proportion of $\frac{1}{2}$ –1 Hz eye movements was assessed in records of EOGs taken in three mood states: elated (+3), normal (0), and depressed (–3).

The records to be analysed for the validity of the correlation between the presence of EOG $\frac{1}{2}$ –1 Hz waves and the mood were taken from daytime recordings at 0800 hrs; the first 20 minutes of each was studied. They were selected because they had 20 minutes of a satisfactory continuous record. A similar number of manic, depressed, and treated days was selected. They were then shuffled and read blindly in terms of the EOG trace. Admittedly a clue was given by the EEG alpha rhythm, but the EOG was easier to assess and was the more reliable indicator, and the one on which we depended for Fig. 1. The correlation between those daytime recordings and mood, although striking, is not as high as that based on the first five minutes of the waking state, which consistently predicted the day's behaviour.

Figure 2 shows an example of the instantaneous changes which occurred at the switch. For these recordings the patient was kept awake at night and the EOG continuously

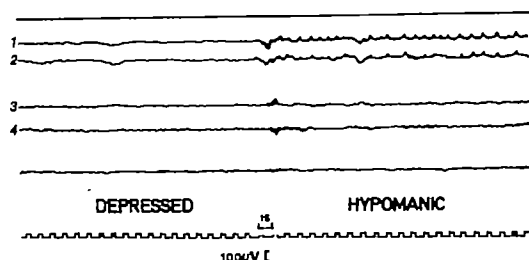


FIG. 2 The EEG and EOG at the time of a mood change switch, when the patient was kept awake for the appropriate part of the night.

recorded; the pattern altered abruptly at the mood change-over, which was between 0230 and 0300 hrs. The $\frac{1}{2}$ -1 Hz pattern would either disappear or appear suddenly, depending on the direction of the mood change. This phenomenon was recorded on eight nights, with identical results. On 209 occasions the EOG on waking was instantly that of the anticipated and observed mood, on nine occasions it was equivocal (i.e. showing some but not persistent $\frac{1}{2}$ -1 Hz recording for 5 mins), while on four occasions there was the paradoxical state (i.e. the mood and EOG were then inconsistent).

The sleeping EEG nearly always (99% of 204 satisfactory records) showed a period of REM sleep between 0230 and 0300 hrs, when the change was expected to take place. On the one occasion when that was clearly not so, there was a cycle defect, and the patient was manic both on retiring and on rising; there was no period of REM sleep at the critical time on that night. On one night, when the patient's mood swing was very obviously from a more aggressive mania than usual to a very profound, almost stuporose depression, he had a period of REM sleep which was longer than usual (51 min).

Cycle defects such as that mentioned above were rare (fewer than one/year). The patient would have two or more days of hyperactivity, and recommence the 48 hr cycle on the second or subsequent nights. Less rarely, the time of change-over from hyperactivity to inactivity was delayed from the usual time by as much as 10-11 hours (this occurred 10-12 times a year). The cycle was then

corrected on the following night, with the usual change-over of mood.

Discussion

The relationship between circadian and similar biological rhythm cycles of metabolism remains intriguing but obscure. The rare and rapidly cycling patients, especially those with 48 hour rhythms such as the one reported here, continually raise the possibility that their study will give an insight into some of the temporal aspects of cerebral organisation and affective psychosis.

The fact that this patient had eye movements on his EOG recordings when manic and not when depressed, and the fact that they began the moment he awoke manic or ceased the moment he fell asleep when manic, and then appeared as soon as half aroused, gives objective evidence that the time of switching was, for a biological phenomenon, peculiarly precise and independent of the sleeping state. The switch, when it occurred during sleep, was probably associated with paradoxical sleep.

Paschalis *et al.* (1980) described another patient who showed a sliding time of change, always in the daytime, in a cycle of almost 48 hours. The time of switching unusually jumped eight hours, and so 'never' occurred during sleep. This, as Paschalis *et al.* report, is different from the majority of patients with 48 hour cycles described in the literature. The collection of several such reports—which must be of isolated cases because the phenomenon is rarely so precise in clinical practice—may lead to an illuminating theory, or at least to heuristically valuable models. These models must ultimately, if complete, 'explain' the features of these cases which are similar but unrelated in detail.

This patient is now dead. His remarkable response to lithium therapy has been reported (Hanna *et al.*, 1972), but not the fact that in his terminal illness (with a carcinoma of the stomach), despite our inability to detect mood changes, he only requested opiates on alternate days.

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A Study of Violent Behaviour Among In-Patients in a Psychiatric Hospital

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A retrospective analysis of the violent incident forms returned over a one year period in a psychiatric hospital recorded 283 incidents involving 144 patients. Almost all were of a petty nature, none leading to lasting injuries. Compared with the average daily in-patient population, offending patients were more likely to be male, to be less than 45 years old, and to have a diagnosis of schizophrenia.

Violent incidents are almost daily occurrences in a district psychiatric hospital, but apart from Fottrell's 1980 survey of three hospitals there has been little research published on violent behaviour among psychiatric in-patients in the UK. This survey attempts to build on Fottrell's work, with the aim of identifying which patients, in which setting, become involved in violent behaviour.

Method

Violent incident forms returned for in-patients at the Central Hospital, Hatton, Warwickshire over the period 1 June 1983 to 31 May 1984 were analysed, and additional data collected during the next three months. The hospital provides all the in-patient psychiatric facilities for a semi-rural area with a population of about 200 000. Three acute admission wards, six long-stay wards, nine psychogeriatric

wards, an alcohol treatment unit, a rehabilitation unit and a ward for the adult mentally handicapped were included. A Regional Secure Unit was the only in-patient ward not included in the study, this treats forensic patients, and is not used as a place of transfer for violent or acutely disturbed patients from the rest of the hospital.

Nursing staff at the hospital are instructed to complete a violent incident form whenever a patient intentionally commits an act that leads to damage to property or injury to another person. As our study was retrospective, we were not able to include any measures to improve the reliability of reporting. Thresholds for reporting violent incidents differ, we consider them to be higher on acute admission wards, compared with the rest of the hospital. To ensure that no serious incident had gone unreported, we asked senior medical and nursing staff to list any such incidents they knew of. No serious omissions were noted.

The violent incident form provides information on the name, sex, status, and age of the patient initiating the incident, the time and date, any injuries to people or damage to property, and the nurses' assessment of any

precipitating events and action subsequently taken. All forms are countersigned by the ward doctor or duty doctor, who must examine and report on any injuries which have occurred. We applied Fottrell's three-stage classification of the seriousness of injuries to the data.

We estimated the characteristics of the average daily in-patient population over the 12 month period of study in the following way. The average daily number of in-patients ($n=435$) and their ward distributions were determined from the 12 end-of-month return figures at the records department. The age, sex, and diagnosis of all in-patients at the end of the study ($n=420$) was determined from case notes, and the figures scaled up proportionally. This method will produce some errors on the high turnover wards, where sampling of the characteristics of the in-patient population at more frequent intervals during the one year period is required to increase the accuracy of the 'average population' data.

During the period of study a total of 1220 in-patients passed through the hospital. Since the characteristics of the majority (800) of these were unknown, only those of the

TABLE I
A comparison of the characteristics of violent patients against the average daily in-patient population

<i>Characteristics</i>	<i>Average daily in-patient population (n=435)</i>		<i>Violent patients (n=144)</i>		<i>% (violent) -% (average)</i>
	<i>Number</i>	<i>%</i>	<i>Number</i>	<i>%</i>	
Sex					
Male	218	50	92	64	+14
Female	217	50	52	36	-14
Age					
<44	80	18	58	40	+22
45-64	114	26	35	24	-2
65+	241	56	51	36	-20
Diagnosis					
Schizophrenia	132	30	81	56.5	+26.5
Affective psychosis	86	20	20	14	-6
Dementia	145	33	29	20	-13
Neurosis	22	5	2	1.5	-3.5
Personality disorder	15	4	9	6	+2
Mental handicap	19	4	3	2	-2
Alcoholism	16	4	0	0	-4
Ward type			<i>Violent incidents (n=283)</i>		
Acute	57	13	69	24	+11
Alcohol	16	3.5	0	0	-3.5
Rehabilitation	25	5.5	5	2	-3.5
Medium stay	48	11	63	22	+11
Long stay	90	21	86	30	+9
Psychogeriatric	178	41	53	19	-22
Mental handicap	21	5	7	3	-2

'average population' are given as an approximate guide, and no statistical tests are used when comparing this group with the patients involved in violent incidents.

Results

A total of 283 violent incident forms were returned over the one year period, an average of 24 per month. The majority of incidents involved either no visible injury (69.6%) or minor injuries such as superficial cuts and bruises, which did not require further investigation (29.0%). The four serious injuries consisted of a deep cut, two cases of severe bruising, and a cigarette burn; none of them resulted in long-term injuries.

The commonest victims of violence were fellow patients (114), closely followed by nursing staff (92); there was a tendency for injuries to fellow patients to be more severe than injuries to nursing staff. The 283 incidents were perpetrated by only 144 patients; 103 were responsible for only one incident each, while 41 were multiple offenders. Amongst the latter one stands out as exceptional, with 33 reports of violence. This was a young schizophrenic woman from an acute ward.

Table I compares the patients who behaved violently with the average population: the former were more likely to be male young (under 45), have a diagnosis of schizophrenia, and to come from non-geriatric wards. A comparison of the characteristics of single and multiple offenders did not reveal any differences between the two groups.

An analysis of the times of day at which violent incidents occurred showed that they were distributed fairly evenly throughout the morning, afternoon, and evening, with very few at night. The hours in which violence peaked corresponded with the times when meals were being served on the wards.

Discussion

Violence leading to serious injury was rare throughout the one year period. This finding is in keeping with most other studies (Armond (1982), Fottrell (1980), and Folkard (1957)). However, Aiken (1984) found six incidents leading to third-degree injuries to staff on a 17 bed locked acute admission ward at the Maudsley Hospital over a six month period.

The most important finding here is the excess of violence amongst schizophrenic patients compared with the hospital population. Higher rates of violence amongst these compared with other diagnostic groups have been noted by Fottrell (1980) and by Hafner & Boker (1973). In Fottrell's survey 39% of the patients who behaved violently were schizophrenic, but it is not possible to say whether this is more than expected, since the proportion in the overall hospital population was not recorded. In our survey 56% of the patients who behaved violently were schizophrenic and, at the time of the end of the survey at least, only 30% of the in-patient population had this diagnosis. Since the rate of turnover

of schizophrenic patients is relatively low compared with that of other diagnostic groups, we believe that the average figure of 30% is fairly accurate. The excess of schizophrenics among the violent group is therefore significant.

The findings of excess violence amongst the younger age-groups and on the acute wards were expected, but the excess on the medium and long-stay wards was more surprising, and warranted further investigation. We found it was largely due to a small group of eight patients, who all had a diagnosis of schizophrenia. They had active delusions and hallucinations, which were resistant to medication and which seemed to precipitate their violent behaviour. This group contributed 56 incidents over the one year period, and continued to offend at about the same rate over the subsequent six months. We feel that they constitute an important group for further study and intervention.

Fottrell found that staff were the commonest victims at all three of the hospitals he studied, whereas in our survey patients more commonly assaulted fellow patients than members of staff. This probably reflects the differences in the types of ward populations studied. Of the violent incidents in our study, 71% occurred on the medium, long-stay, and psychogeriatric wards, which together account for 73% of the in-patient beds. The ratio of nursing staff to patients is much lower on these wards, so that when violence occurs in this setting it is more likely to be directed at fellow patients than at the nursing staff.

The timing of incidents at the Central Hospital shows a more even distribution than in Fottrell's study, where 30% of all incidents occurred between 7 a.m. and 9 a.m.; this period accounted for only 16.6% of the incidents at the Central Hospital. However, we agree with Ekblom (1970) that it is at times when patients are together on the ward, with little therapeutic activity, that violence is most likely. The violent incident form contains a section asking whether the nursing staff could identify any events which precipitated the incident. When such events were mentioned, they most often included arguments over food and personal space which occurred at meal times; this type of incident probably accounts for the peaks of violence at meal times. However, within the three acute wards the commonest precipitating events were patients being detained on the ward or given medication against their will—a finding in keeping with those of Aiken (1984).

Fottrell's finding that only a small percentage of the patient population behave violently was confirmed; in his study only 171 of the 1661 patients

treated in a one year period committed violent incidents (10.3%). At the Central Hospital 144 patients were involved, out of a total number of 1220 in-patients over the year (11.8%).

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The Influence of Psychological Factors on the Opiate Withdrawal Syndrome

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Psychological and drug-related variables and their effect on the severity of withdrawal symptoms were examined in a group of addicts being withdrawn from opiates on an in-patient drug dependence unit. Two psychological factors—neuroticism and the degree of distress expected by the patient—were related to subsequent severity of symptoms. Both are anxiety-related, and may serve to amplify withdrawal symptoms. Surprisingly, drug dose was unrelated to symptom severity.

A principal feature of opiate addiction is the withdrawal syndrome, which is often regarded as one of the defining characteristics of such an addiction. The withdrawal syndrome is recognised and described in general terms in most accounts of addiction, but has not been fully investigated. There are many aspects of it that require further research.

The fact that physiological symptoms such as increased perspiration, lacrimation, abdominal cramps, nausea, and diarrhoea are prominent features of withdrawal led the authors of early formulations to emphasise its 'physical' aspects; this is reflected in the use of the medical term 'syndrome'. However, research has shown that psychological

factors may also have a powerful effect on aspects of the withdrawal syndrome.

As early as 1927, Pavlov noted that classical conditioning of morphine-induced responses had been observed by Krylov; subsequently, Wikler (1948) proposed a theory that relapse after detoxification was due in part to conditioned stimuli that elicit aspects of the withdrawal syndrome. Many studies have since confirmed this, and have extended understanding of the role of conditioning in the addictive disorders (O'Brien, 1976; O'Brien *et al.*, 1977; Siegel, 1979).

Kleber (1981) stated that other psychological factors such as the patient's personality, his state of

mind at the time of withdrawal, the setting in which withdrawal takes place, and his expectations of the severity of the symptoms may all have a marked effect on the severity of the withdrawal. Indeed, Kleber suggested that their cumulative effect can be as great as that of any pharmacological factors. This observation is in accord with the views of many experienced clinicians, but has not received adequate empirical investigation. The present study looks at Kleber's suggestions as they apply to the withdrawal syndrome experienced by a group of opiate addicts being detoxified from drugs as in-patients on a graduated oral methadone reduction schedule.

Method

All heroin and methadone addicts admitted to the in-patient Drug Dependence Clinical Research and Treatment Unit of the Bethlem Royal Hospital in a five-month period in 1984 were studied. There were 34 such admissions, two of whom were excluded since they were drug-free at the time of admission; 24 were male and 8 female. Their average age was 25.9 years (range 18–38). The length of time for which they had used opiates varied between 1–19 years, with a mean of 5 years. In these respects the subjects are similar to the wider population of addicts attending London Drug Dependence Clinics (Gossop *et al*, 1984; Blumberg, 1981).

On admission, all subjects underwent a three-day assessment period during which their need for opiates was assessed. The initial daily dose level of methadone was established on the basis of clinical examination by a psychiatrist and of nurses' observations. The response to this trial dose was closely monitored during a three-day period, and if there were signs of intoxication or of opiate withdrawal the dose was adjusted as necessary. All subjects were then withdrawn from their drug of dependence over a 21-day period. During the withdrawal programme addicts received oral methadone three times a day on a linear reduction schedule. During the first 50 days they completed a self-report questionnaire designed to measure symptoms of opiate withdrawal. A version of this was used in the study reported by Gossop *et al*, (1984). The questionnaire contained 32 signs and symptoms; subjects were required to rate themselves on each item as it applied during the preceding 24 hours, using a four-point scale (nil, mild, moderate, severe). The 32 items were: feeling sick; vomiting; diarrhoea; poor appetite; dry mouth; stomach cramps; restlessness; eyes sensitive to light; headache; drowsiness; dizziness or giddiness; fainting attacks or lightheadedness; stiffness of arms and legs; spontaneous twitching (contraction) of muscles; trembling hands; feelings of coldness; feeling of unreality; 'gooseflesh'; hot and cold flushes; increased sweating; runny nose; trouble starting urination; passing more than usual quantity of urine; heart pounding; fatigue or tiredness; muscular tension; aches and pains; weakness; yawning, sneezing, runny eyes; and insomnia.

At the time of admission all subjects completed a further

brief questionnaire, in which they were asked to state on a four-point scale the severity of the withdrawal symptoms they expected to experience. They also completed the Eysenck Personality Questionnaire (EPQ) (Eysenck & Eysenck, 1975).

Results

A stepwise regression analysis was performed, in which the extraversion, neuroticism, and psychoticism scales of the EPQ, length of opiate use, withdrawal dose of methadone, age, and expected withdrawal distress were regressed against the maximum total withdrawal score obtained for each subject during the measurement period. Two variables were found to be significantly related to intensity of the opiate withdrawal syndrome—neuroticism, and the level of withdrawal distress expected, accounted for 36% (R^2) of the total variance in the withdrawal score. Neuroticism accounted for 24% of this (d.f. = 8.41, $P = 0.007$), and expected distress for 12% (d.f. = 7.0, $P = 0.04$). The main drug-related measures—methadone dose and length of opiate use prior to admission—were not significantly related to intensity of withdrawal. The mean value for methadone dose was 38.4 mg, with a range of 10–60 mg; more than two-thirds of the doses were within the 30–45 mg range. This finding is directly comparable to those of other studies at this clinic (Johns & Gossop, 1985, McCafferty & Gossop, 1981).

In a further analysis, simple correlation coefficients were calculated between the maximum withdrawal score and each of the other measures. The highest correlation was between neuroticism and withdrawal distress ($r = 0.46$), and this was the only significant result ($P = 0.01$). Duration of opiate use ($r = 0.36$) and expected distress ($r = 0.35$) were both slightly above the 5% level of significance ($P = 0.054$ and 0.066 respectively). Methadone dose was not correlated with maximum withdrawal distress ($r = -0.19$).

Discussion

These results confirm Kleber's observation (1981) that psychological factors play a major role in the opiate withdrawal syndrome: in particular, they show that the personality factor of neuroticism and the addict's expectations about withdrawal are strongly related to the maximum level of withdrawal distress subsequently experienced during detoxification.

Both these measures may be seen in broad terms as anxiety-related. Eysenck's neuroticism factor has been described as reflecting the reactivity and lability of the autonomic nervous system (Gossop, 1981); Eysenck & Rachman (1965) suggest that high neuroticism scorers are predisposed to respond more strongly, more lastingly, and more quickly with their autonomic nervous system when presented with stressful stimuli. In this respect, high levels of neuro-

ticism may act to amplify the response to opiate withdrawal. The same sort of process may also underlie the link between the addict's expectations about detoxification and his subsequent response to drug withdrawal. Eiser & Gossop (1979) found that most addicts were extremely frightened of withdrawal, and believed that this was also true of other addicts. The present study shows that the more frightened of withdrawal the addict is, the more discomfort he is likely to report during the actual process of being withdrawn from drugs.

It is interesting that in the regression analysis both the dose of methadone prescribed prior to the withdrawal programme and the length of opiate use were found to be unrelated to maximum levels of withdrawal distress, although there was a positive (but non-significant) correlation between duration of opiate use and withdrawal distress when simple correlation coefficients were calculated. Kleber (1981) suggests that duration of use may be related to withdrawal severity only up to a certain point, and that after two to three months it has little effect. It is more surprising that the dose of methadone was unrelated to severity. Kleber also states that the larger the daily dose of opiates, the more severe the withdrawal syndrome; this has been universally assumed, and seems almost self-evident; However, it is surprisingly difficult to find any empirical research to substantiate the claim. Several other definitive sources make the same link between dose and withdrawal severity, but like Kleber fail to cite any reference to original research (Goodman & Gilman, 1980;

Martindale, 1982; Wikler, 1980). The findings of the present study, coupled with the lack of empirical evidence, point to the need for further research to clarify this issue.

The main clinical implication of these results is that attention should be paid to the psychological state of the addict before and during the process of drug withdrawal. Research in other areas of medical psychology has shown that giving patients accurate information about what they may expect after surgical procedures reduces the pain and discomfort they subsequently experience (e.g. Melamed, 1977). Similarly, providing accurate information reduces the distress experienced by subjects undergoing other painful procedures (Johnson, 1973; Leventhal *et al.*, 1979). However, the latter found that the way in which the briefing was phrased was an important variable; words such as 'pain' could cancel the discomfort-reducing effect of the information. The response to drug withdrawal is similarly influenced by the extent of the patient's knowledge of and participation in the withdrawal procedure. Stitzer *et al.* (1982) found that knowledge of the dose reduction schedule serves to reduce symptom complaints, and they suggest that much of the withdrawal symptomatology may be a result of anxiety and uncertainty. These findings, together with those of the present study, suggest that when withdrawing addicts from opiates, providing the individual with information about the sort of intensity of symptoms that might occur could reduce withdrawal distress, but that this issue deserves further investigation.

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Seasonal Hypomania in a Patient with Cold Urticaria

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The clinical and biochemical disturbances of a patient with seasonal hypomania and cold urticaria are described and discussed with reference to histamine metabolism in mental disorder.

Allergic disorders have often been reported in association with mental disturbance, although interpreting the relationship is more complex than has often been assumed (Rix *et al*, 1984). Shared biochemical features have been suggested in recent work on the behavioural associations of histamine and antihistamines (Hough & Green, 1984). Histamine acts through two classes of receptors, both of which are affected by antidepressants and neuroleptics (Kanof & Greengard, 1978). Antagonism of the type designated H1 causes sedation, whereas unbalanced antagonism of the H2 type, produced by cimetidine, has been linked with psychiatric manifestations, which are often affective in nature (Crowder & Pate, 1980; Hubain *et al*, 1982; Titus, 1983).

This report describes a case of intermittent cold urticaria, occurring in close relation to unipolar affective disturbances over several years. There are no reports in the literature over the past two decades of any similar case, or of any recognised psychiatric morbidity associated with cold urticaria. It is estimated that about 0.4% of the population are affected by cold urticaria at some time in their lives (Champion *et al*, 1969).

Case History

Presentation of cold urticaria

The patient, a caucasian male born in 1961 first became ill in 1972, when he collapsed after swimming. During each subsequent winter he experienced episodes of diffuse

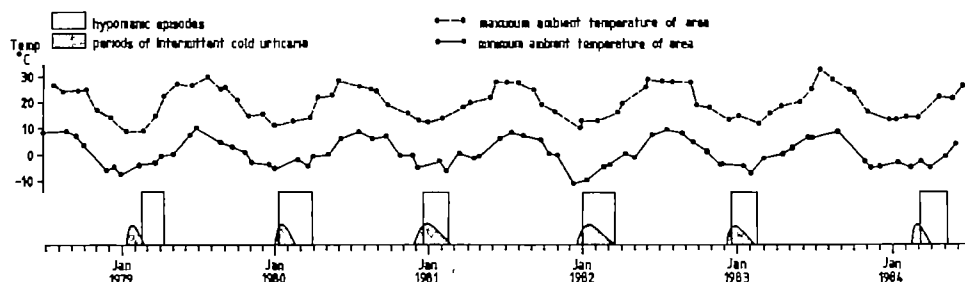


FIG 1 The chronological relationships of hypomaniac episodes, cold urticaria, and temperature extremes in the patient's area of residence.

swelling affecting his hands and face, often accompanied by headaches, from exposure to the cold.

In 1975 he was examined at St John's Hospital, London where dermatographia and a marked urticarial reaction to ice were noted. The following tests were normal or negative: immune globulin E, cryofibrinogen, cryoglobulins, C_3 and C_4 complement components, serum protein electrophoresis, lupus cells, anti-nuclear factor, rheumatoid factor, Wasserman reaction, and stool microscopy for ova and parasites. A diagnosis of idiopathic cold urticaria was made.

Psychiatric presentation

In the severe winter of 1978, when the episodes of swelling commenced, the patient (then aged 17) began to talk incoherently about electronic forces and of a plan he had to construct a magnetic train. Growing restlessness culminated in a sudden outburst of bizarre excitement, and at psychiatric examination a preoccupation with an electronic activity which fused mind and body was noted. On the day he was admitted to the psychiatric unit, a disturbance arose in which he jumped from one bed to another while stripping off his clothes.

Psychiatric assessment and observation

The patient complained that thoughts were crowding his mind, which tired him and prevented him from achieving his objectives, and that sounds seemed very loud. He heard the incoherent voices of a crowd talking to him and had a vague notion that someone was trying to harm him. His talk was coherent, though circumstantial, and he was markedly distractible. His mood was one of moderate euphoria, with transient feelings of dysphoria and marked irritability. Apart from impairment of concentration, his cognitive functions were intact. No abnormality of pre-morbid personality was elicited. There was a history of a single episode of severe depression in a maternal aunt. On physical examination no significant abnormality was found, but there were scratch marks on his hands and face in keeping with recent urticaria.

During his first week on the ward he was awake for most of the night, when he would bang his head on the wall or

roam about and shout. He climbed chairs and attempted to throw himself on to the floor, likening himself to an Olympic swimmer, and he smashed the glass of a door. During this time he was given a daily dose of 15 mg of trifluoperazine and up to 250 mg of chlorpromazine. He improved gradually during five weeks of in-patient treatment. A diagnosis of mania was made.

A month later, at follow-up, he was doing well and his medicines were reduced. Shortly afterwards he stopped his treatment, and did not return to the clinic because he remained well.

Clinical Course

Dermatological follow-up

The patient's next contact with St John's Hospital was in early 1981, when he was readmitted for further routine tests.

Following the application of an ice cube to his arm, blood samples from the antecubital fossa produced circulating histamine, reaching a maximum level of 9 ng/cm^3 after 14 minutes. He was placed on a cyproheptadine, which he used intermittently.

Psychiatric follow-up

Since the first psychiatric admission the patient has shown annual hypomaniac episodes, occurring only during the winter. His parents noticed that they built up over three or four weeks, after periods of unusually severe exposure to the cold. Although the psychiatric admission was delayed in 1984, it occurred during severe cold weather, when he spent several days working outdoors to repair his car. The onset was preceded by marked urticaria. Fig. 1 shows the chronological relationship between the two conditions.

Discussion

This patient, with proven idiopathic acquired cold urticaria, was manic on presentation and typically hypomaniac during each subsequent psychiatric dis-

turbance; the clinical features were observed and documented by one of us during three of the six episodes recorded here.

The onset of cold urticaria prior to the disturbed behaviour was observed by the patient's parents, who lived with him throughout the period in question. Selective recollection may have affected the accuracy of their observations, although for most occasions examples could be given of everyday activities leading to greater cold exposure just before the disturbances. The parents were not aware of any early affective changes which could have been responsible for increased cold exposure.

In cold urticaria, a massive release of histamine is detected in the circulation (Kaplan & Beaven, 1976) and in the urine (Granerus *et al.*, 1969), which is generally undetectable in other types of urticaria due to its rapid tissue uptake. There has never been substantiation of the view once held that parenteral histamine might benefit man by increasing cerebral blood flow and capillary permeability (Simpson & Kline, 1961). On the contrary, histamine release may correspond with a systemic reaction which includes light-headedness, a sensation of tightness in the chest, transient hypotension, and fainting. These effects may be expected to contribute to any emotional distress associated with cold urticaria, and were reported by our patient in relation to acute episodes, although during in-patient treatment his recorded pulse and blood pressure readings were normal.

Elucidating possible aetiological factors in individual examples of regular cyclical affective disorders is difficult (Jenner & Damas-Mora, 1983). Although our patient's clinical picture could have resulted from the remarkable coincidental concurrence of two independently seasonal conditions, it appears

more likely to be due to provocation of one by the other, or to a shared aetiological factor.

There is evidence to suggest that histamine crosses the blood-brain barrier in animals (Crossland & Mitchell, 1956; Snyder *et al.*, 1964), although this has not been shown in man. Endogenous release of hypothalamic histamine may be markedly increased during cold exposure (Taylor & Snyder, 1971), and its intraventricular injection in rats results in catalepsy and hypothermia (Pilk & Nowak, 1979). Besides histamine there are other products of mast cell degranulation, such as prostaglandin D₂ and the leukotrienes, and one of histamine's metabolites, imidazoleacetic acid, when given parenterally to rats, has been shown to cause increased exploratory behaviour, hyperactivity, ataxia, and catalepsy (Marcus *et al.*, 1971). Increased levels of conjugated imidazoleacetic acid have been found in the urine of schizophrenic patients (Kobayashi & Freeman, 1961).

Finally, the effects of cyproheptadine should be considered. It is the drug of choice for cold urticaria (Wanderer *et al.*, 1977), and has a greater therapeutic effect than other drugs with a more powerful anti-histaminic action, probably due to its influencing the kinin system at tissue level. Cyproheptadine is also a serotonin antagonist, which has encouraged the suggestion that its use might be associated with psychiatric disturbance (Pearce *et al.*, 1977). However, in our case cyproheptadine was only introduced after the first three psychiatric episodes, and it was taken irregularly for subsequent attacks of cold urticaria. The patient's family observed that in the short term the drug exerted a beneficial calming effect on the mood disorder, but it is not known if its overall effect could have been to contribute to the onset of some of the episodes.

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Priapism and Psychotropic Medication

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Priapism is a rare but serious adverse effect of psychotropic drugs resulting from their peripheral alpha-adrenoreceptor blocking action. Two patients developed this during treatment with oral phenothiazines: one was treated surgically with a venous shunt, and the second case resolved after intracavernosal injection of an alpha-adrenoreceptor stimulating drug.

Priapism is a pathologically prolonged and painful penile erection, usually unassociated with sexual desire or intercourse. It is caused by obstruction of the venous drainage from the corpora cavernosa, while that from the corpus spongiosum and glans penis remains unaffected. Priapism, therefore, can be clinically differentiated from a normal erection in that only the corpora cavernosa are turgid, the corpus spongiosum and glans penis being quite flaccid (Wasmer *et al.*, 1981). Prompt diagnosis and treatment are essential, or the resultant stasis will lead to thrombotic changes and ultimately to fibrosis of the corpora cavernosa, and thus to permanent erectile impotence. This can be restored only partially by implantation of a penile prosthesis—a difficult and sometimes unsuccessful operation (Pryor, 1982).

The usual treatment of priapism is surgical, initially involving aspiration and saline irrigation of the corpora cavernosa, the creation of a temporary

fistula between these and the corpus spongiosum and, if the venous drainage is still unsatisfactory, a corpus cavernosum saphenous shunt (Pryor, 1982). Recently, there have been encouraging preliminary results for injection of metaraminol, an alpha-adrenoreceptor stimulating drug, directly into the cavernosal space (Brindley, 1983, 1984; Stanners & Colin-Jones, 1984).

Priapism may occur at any age, being most frequent in the third and fourth decades, and may arise in a wide variety of circumstances; it often occurs on waking, but only occasionally following prolonged sexual stimulation. Some 30-40% of affected cases have the condition recurrently; about half are idiopathic. The remainder are associated with diverse factors, which can be grouped into three main categories: local pathology of the pelvis—a direct obstructive effect; blood dyscrasias, especially sickle cell disease, which facilitate thrombosis by increasing

blood viscosity; and factors, including drugs, which alter the neurological mechanism of normal erection and detumescence.

The main drug groups known to be associated with priapism are anti-hypertensives and anti-coagulants. However, there has been an increasing number of reports recently linking priapism with psychotropic medication. The two patients described here were treated in the same psychiatric unit within two years of each other.

Case reports

Case 1

A 38 year-old journalist with a known history of schizophrenia was admitted following a recurrence of the illness. He had had previous admissions to other hospitals, and treatment with phenothiazines and haloperidol, with no apparent adverse effects; there was no other relevant medical or drug history. On admission he was restless, thought-disordered, deluded, and hallucinating; physical examination and routine blood tests were normal. He was prescribed chlorpromazine (100 mg q.d.s.) and flurazepam (15 mg, nocte, increased to 45 mg, nocte), and gradually improved.

Three weeks after admission he complained of a persistent and painful erection, and was transferred to the urological unit. Aspiration of blood clots from the corpora cavernosa, followed by saline irrigation, were without effect, and a right corporo-saphenous and left bulbo-corporal shunt were created on the following day. His priapism gradually resolved, although he later developed a penile ulceration, which necessitated an in-dwelling catheter for ten days. The priapism was attributed to the chlorpromazine, which was discontinued. In the following four weeks he remained well, but as he subsequently absconded and did not return his long-term outcome remains unknown.

Case 2

A 42-year-old merchant seaman was referred following a suicide attempt. He said he had become increasingly depressed because he was unable to visit his seven year-old daughter, who was in the custody of his estranged wife. He also volunteered a history of chronic alcoholism and extensive abdominal surgery for malignancy, but these claims were later found to be greatly exaggerated, and other statements could not be verified. There was no history of blood dyscrasias or pelvic disease. At the time of admission he was receiving monthly injections of vitamin B12, combined with oral iron, calcium and pancreatic enzymes (Pancorex). Physical examination was normal, except for multiple operative scars on the abdomen. Blood tests showed a mild iron-deficiency anaemia and slightly elevated blood glucose: a glucose tolerance test was consistent with 'dumping syndrome'. Barium meal indicated a Bilroth I gastrectomy.

On admission he was dishevelled but co-operative, and appeared only mildly depressed. He was prescribed chlor-methiazole, (1 g b.d.) in a reducing regime, to which was

added, two days later, promazine (100 mg, nocte). On the fourth night he woke at 4 a.m. with an erection, which persisted and became increasingly painful. He reported this 12 hours later, when a diagnosis of priapism was made. Pethidine (50 mg, i.m.) and Prochlorperazine (12.5 mg i.m.) were given, and he was transferred to a urological hospital, where Doppler ultrasound examination of the penile arteries showed a high flow state, supporting the diagnosis of priapism. Procyclidine (10 mg, i.v.) had no effect, but infusion of metaraminol deep into the cavernous tissue was effective, resulting in the gradual resolution of the priapism. On return to the psychiatric ward, two days later, his penis was still enlarged and tender, but no longer turgid. He was not prescribed any further phenothiazines, and suffered no recurrence of the priapism in the subsequent six weeks. During this period his behaviour changed dramatically, in that he became unreasonable, over-demanding and abusive, thus reinforcing our initial diagnosis of a Munchausen syndrome. His discharge was precipitous, and he did not attend for follow-up.

Discussion

Contrary to popular belief, the excessively prolonged penile erection of priapism, far from being a sign of virility, is in reality a source of distress and embarrassment, and may lead to permanent impotence.

The pathophysiology is unclear. Normal erection results from sustained increase of arterial supply to the erectile tissue, which is probably achieved by relaxation of valves or 'polsters' (Wasmer *et al*, 1981), allowing increased blood flow into the corpora cavernosa, combined with simultaneous regulated partial constriction of 'shunt' arteries, which impedes venous drainage (Wagner *et al*, 1982). These vascular changes are under autonomic nervous control, both sympathetic and parasympathetic/vipergic, the latter depending on a recently identified class of splanchnic fibres, which utilize as a neurotransmitter vasoactive intestinal polypeptides (Polak *et al*, 1981) with atropine resistant vasodilatory responses (Bloom & Edwards, 1980). Preponderance of sympathetic over parasympathetic activation is associated with penile flaccidity, while the reverse results in erection.

Most psychotropic drugs are potent alpha-adrenoreceptor blockers, and could induce priapism by directly inhibiting the sympathetically controlled detumescence, which is probably the result of their peripheral action on those receptors lying within or close to the cavernosal space. Perhaps the strongest evidence for this mechanism comes from Brindley's elegant experiments on both normal men and men with erectile impotence, in whom he was able to induce penile erections by intracavernosal injection of the alpha-blocker phenoxybenzamine, which

were subsequently resolved by a similar injection of the alpha-stimulating drug metaraminol (Brindley, 1983, 1984). The effectiveness of this technique has been further demonstrated by the successful treatment of a patient with priapism secondary to chronic myeloid leukaemia (Stanners & Colin-Jones, 1984), and by the satisfactory treatment of our second patient.

The suggestion that other pharmacological properties of neuroleptic drugs, such as their anticholinergic or hypotensive action, could cause priapism (Meiraz & Fishelovitch, 1969) has not been substantiated. There have been no reports of priapism associated with the use of anticholinergic drugs alone, nor evidence of hypotension in cases of drug-induced priapism, including our two patients.

A wide range of psychotropic drugs have been implicated in priapism (Pryor, 1982; Barnes, 1984), but Mitchell & Popkin (1982) could find only 15 cases of priapism reported in which the drug involved was specifically identified; most frequently this was a phenothiazine, as with our two patients. Six of these patients were taking thioridazine, and seven others chlorpromazine, in one of whom priapism followed a single injection (25 mg, i.m.). One patient was taking thioridazine combined with trifluoperazine, and another was on mesoridazine. Since then, 11 cases have been reported in which priapism was attributed to trazodone—an antidepressant with known alpha-blocking properties (Scher *et al*, 1983).

However, if alpha-adrenergic blockade—a com-

mon property among psychotropic drugs—is the cause of drug-induced priapism, why is the condition apparently rare among psychiatric patients? It may be that in a proportion of cases it goes unrecognised or unreported because of its short duration and spontaneous remission, or because of the obvious difficulties of detecting such a feature in markedly psychotic patients. However, it is more likely that priapism is a complex and multi-factorial disorder, and that the interaction of several factors is necessary for it to become manifest. Possibly, psychotropic drugs induce priapism in only a small proportion of men who are sensitive to alpha-blockade at a critical site, perhaps at the putative valves or polsters (Lawrence *et al*, 1984), and only if additional factors such as cannabis or alcohol are present (Winter, 1981). This could explain the observed inconsistency of response of such men to psychotropic medication, as with our first patient (who had previously been treated with the same drug with no adverse effect) and with the patient reported by Dawson-Butterworth (1969). Our second patient had a long history of alcohol abuse up to the time of his admission.

Psychotropic medication is thus associated with a small but definite risk of priapism, which is increased with the use of phenothiazines and trazodone. Early recognition and prompt treatment are essential to prevent permanent impotence, and will depend on careful history-taking and alertness to known predisposing factors such as blood dyscrasias, especially sickle cell trait, and previous abnormal erections.

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Mania Following Bereavement

SIR: The assumption is not uncommonly made in papers that since there have been few reports of a particular event, relationship, etc., in the literature, this means that the phenomenon is rare or uncommon. A case in point is Rosenman and Taylor's case report of mania following bereavement (*Journal*, April, 1986, 148, 468-470). Surely such assumptions are quite erroneous. How many clinical psychiatrists take the trouble to report such cases in the literature? I wonder if there is the slightest connection between cases reported and cases actually occurring.

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SIR: The case report by Dr Rosenman and Ms Taylor (*Journal*, April 1986, 148, 468-70) is further evidence of the continuing difficulty there is in riding modern psychiatry of Freud's erotic fantasies. As Oscar Wilde might have said, the only absurdities tenaciously believed are the really unbelievable ones.

In their discussion, the authors refer to the "paradoxical nature of the response". The false paradox is in their use of the words "grief" and "mania" as opposites. Grief is a normal experience and the opposite might be happiness. The patient was not just happy but described as profligate, grandiose, talking constantly, overbearing and irritable. In a word, manic—an illness, the opposite of which is a state of endogenous depression which itself is not merely grief. Does anyone really expect mania to occur only after weddings?

While one can accept the seemingly jolly clown entertaining everyone while his heart is broken because of his lost love, it is going a bit too far to see the same 'manic defence' in the patient described whose clear bipolar affective vulnerability was shown by the puerperal depression preceeding the manic episode. A non-specific stress reveals a vulnerability (the nub of the matter is that this is metabolic and not primarily psychological) specific to the patient which might, in different individuals, be anything from skin disturbances to cardiac problems. Or was Dr

Rosenman tempted to treat the lady with antidepressants? Freud was very bad on the functional psychoses, unbelievably so.

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Gluten Free/Gluten-Load Trial

SIR: I read the paper by Dr Vliissides *et al* (*Journal*, April 1986, 148, 447-452) with interest, and have a few points to make.

The pathophysiological processes in gluten sensitive enteropathy or coeliac disease are totally independent of the pathophysiological processes in schizophrenia. I have raised this because constantly in the literature coeliac disease and schizophrenia are brought nearer to each other during discussion. In gluten sensitive enteropathy improvement in most patients is seen in one month, and one should not expect such dramatic changes in the form of improvement in schizophrenia in a short time, as the pathophysiological processes in causation of their symptoms are not the same.

Removal of glycoprotein gluten from the diet may help to improve symptoms of schizophrenia, but addition of gluten would not necessarily make symptoms worse. If one selects severely ill psychotics, whose symptoms are at a peak, further deterioration in clinical symptoms by addition of gluten to the diet cannot be adequately measured by existing clinical rating scales. One may expect only no change in scores.

Having used gluten free diet and essential fatty acids in the treatment of schizophrenia with some success, I recommend a minimum period of eight to ten months must elapse before the gluten free diet is considered not beneficial. Nutritional therapies generally take a long time to cause any changes in the body. The total period in the above papers was extremely short.

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A Unitary View on the Aetiology of Schizophrenia

SIR: The article by Wexler (*Journal*, April 1986, **148**, 357–362) is interesting and thought provoking. It reminds one of the theory of neural plasticity and shows that brain function is dynamic rather than static. I had earlier formulated a hypothesis on the aetiology of schizophrenia, attempting to explain the conflicting findings in schizophrenic research.

Some individuals are genetically determined to have weak and vulnerable dopaminergic systems. When subjected to stresses, part of the dopaminergic pathway ceases to function. To compensate for the loss, the surviving dopaminergic fibres give off additional nerve terminals and D2 receptors. This overreaction gives rise to a relative increase in dopaminergic activity and manifests as the positive symptoms of schizophrenia. In mild cases, the dysfunction is self limiting and dopaminergic transmission is able to restore to its previous level of functioning, and the process is completed. The psychotic episode is thus named a schizophreniform reaction.

In more severe cases, the dysfunction is more chronic and progressive. It results in actual degeneration of the dopaminergic pathways. These degenerative changes are supported by the CT scan studies, postmortem findings, as well as the regional difference in cerebral blood flow and PET studies. The significant reduction of CCK (hippocampus, amygdala), somatostatin (hippocampus), opiate receptors (caudate nucleus), 5 HT receptors (frontal cortex), angiotensin converting enzymes (globus pallidus and substantia nigra) and GABA (nucleus accumbens) also point to this degenerative shrinkage involving certain parts of the brain. Postmortem findings indicated subependymal gliosis in the diencephalon and hypothalamus in chronic schizophrenia. This is consistent with the CT scan evidence of a moderate enlargement of the third and lateral ventricles reported in approximately half of the chronic patients. Gliosis is usually a response to brain injury. The origin of this gliosis may lie at the midbrain ventral tegmental area where the dopaminergic neurones originate. The A10 group of the ventral tegmental area spreads along the mesolimbic and mesocortical pathways to end at the limbic structures and pre-frontal area. Because the DA content in the pre-frontal cortex is only approximately 1% of the DA concentration in the striatum, pathological evidence of cell loss or gliosis in this area may not be obvious. However, cerebral blood flow and PET studies do reveal the frontal region abnormalities.

Negative symptoms seem to result when the degeneration becomes more extensive and when the mesocortical dopamine system is involved. As the

effect of DA antagonist in this system is greatly diminished, the symptoms are irreversible with neuroleptic drugs. It may actually become more prominent when the positive symptoms are under control.

Cerebral lateralisation studies have established the asymmetric distribution of major neurotransmitters in the human brain. Dopaminergic systems are left hemisphere biased. It can be deduced that lesions of the dopaminergic system are reflected as left hemisphere dysfunction and such are found in the CT scan, regional blood flow, PET and EEG studies, as well as a specific increase of dopamine in the left hemisphere.

The reactive compensatory process may not be neurotransmitter specific. Hence an alternation in production of noradrenalin may occur in response to dopamine system degeneration, giving rise to affective symptoms which sometimes occur simultaneously or following schizophrenic systems.

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Endogenous Subtype of Depression

SIR: I read with interest the Young *et al* article (*Journal*, March 1986, **148**, 257–267).

Their conclusion rests on their use of the SADS. They interpret SADS item 326 as addressing anhedonia. Rather, this item addresses loss of interest or pleasure. Thus, someone who had lost interest in all activities but nonetheless could still enjoy some activities when actively involved in them would be rated 6 on this item. Thus, this is not strictly a loss of pleasure item and neither anhedonia nor DSM-III melancholia can therefore be accurately inferred.

From a theoretical point of view, Klein (1974) has hypothesized that pervasive anhedonia is the underlying hallmark of melancholia, in contradistinction to decreased interest in activities with retained ability to experience pleasure. From a practical standpoint, many patients report significant and often pervasive loss of interest in activities but still retain the ability to enjoy them once involved. SADS does not allow

for making this distinction, and I believe that collaborative study interviewers did not add additional questions to their interview to cover this distinction. If not, it is then impossible for the authors of this article to convert their SADS interview into a DSM-III diagnosis or to derive a sub-type characterized by pervasive anhedonia.

I submit that perhaps they have identified one group which has pervasive loss of interest without the traditional vegetative signs of depression while another group has pervasive loss of pleasure plus the neuro-vegetative signs, as has already been hypothesized by Klein.

It seems logically impossible for a patient to meet DSM-III criteria for melancholia without meeting at least probable criteria for RDC endogenous depression. Thus, it appears to be a mistake to report 14 patients with DSM-III melancholia who did not also have at least probable RDC endogenous depression.

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Reliability of the SRQ-20

SIR: We were interested to read the paper by Drs Mari & Williams (*Journal*, January, 1986, 148, 23-26). We used the SRQ-20 as a screening instrument among in-patients of a general medicine ward at the AIIMS over a six-month period. Using 11 as the cut-off point, we examined (using the ninth edition of the Present State Examination and a detailed psychiatric history) all those scoring 11 or above as well as a systematic sub-sample of those scoring below 10.

Out of a total of 326 administered the SRQ-20, 158 were examined and diagnosis was made on the basis of ICD-9. At the end of the study, specificity and sensitivity were determined at each possible cut-off point from 1 to 20, based on the 42 psychiatric cases and 116 non-cases. A correction factor was applied so as to generalise the results to the whole sample using the method suggested by Goldberg (1978).

We found great divergence between specificity and sensitivity. The original cut-off score of 11 had a sensitivity of 46.6%, and specificity of 74.1%. At 7/8, these were, respectively, 77.4% and 66.6% (sensitivity) and 51.1% and 54.4% (specificity). We found 9

the ideal cut-off, where sensitivity was 62.9% and specificity 62%. Females, and those educated at school level (matric) or less, scored significantly higher ($P < 0.05$ and $P < 0.01$ respectively, using the chi-squared test). The mean score for the total population was also high—7.78 (SD ± 4.95).

We feel, therefore, that the SRQ-20 may need considerable modification for use in known physically ill samples. Weighting certain questions, skewing questions toward the more "psychological", changing "false positive" questions, and defining the duration or severity of symptoms, may all have to be considered.

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Monoamine Oxidase Inhibitors

SIR: Professor Blackwell's warning (*Journal*, February, 1986, 143, 216-217) about the danger of minimising the risk of MAO inhibition is welcome, if only as the basis for informed discussion. However, as he will no doubt recall (Samuel & Blackwell, 1968), he himself was not always on the side of the angels.

At Bethlem Royal Hospital, the reaction to Rowe's initial observation, shared by Professor Blackwell, was one of ridicule, while my suggestion that Rowe's observation be taken seriously was treated with contempt. My persistence in pursuing the matter was encouraged only by Professor Linford Rees. It is to the credit of neither of us that this encouragement has never been recorded by Blackwell in any of his many publications, nor in the publication of which I was a joint author (Samuel & Blackwell, 1968). This letter will be the first time in the 23 years since this not altogether unimportant discovery was made, that Professor Rees' essential contribution will have been acknowledged in print. It was my pleasure, in 1983, finally to speak to Rowe. To my astonishment he was totally ignorant of the

sequel to his observation and did not know of his mention in any publication nor in Ayd and Blackwell's (1970) book, *Discoveries in Biological Psychiatry*.

I suspect that Professor Blackwell is once again suffering from 'cryptoamnesia', this time in his reference to "manufacturers' myopic files". In the 1970s, such files were part of his responsibility, when working for a "manufacturer", and if 'myopic' who should be blamed? However, such a situation is not true in the 1980s, here in the UK. In 1963, medical advisers in the pharmaceutical industry felt it was their job to push reports of adverse reactions aside ("it would be premature and unscientific to warn patients not to eat cheese": Weber, 1963). Today, all serious side-effects have to be recorded within 48 hours of notification to the company. We no longer shoot the messenger.

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On Serious Violence During Sleep-Walking

SIR: Ian Oswald and John Evans in their article (*Journal*, 1985, 147, 688-691) added important information to our knowledge about violence during sleep. Other sleep disorders, in addition to somnambulism (Podolsky, 1958) can also generate violent behaviour (Raschka, 1984). The term "sleep-related dangerousness" introduced by Moldofsky appears useful. Such behaviour can include car accidents related to narcolepsy and assaultive behaviour related to sleep drunkenness. The criminological and forensic implications of the latter were discussed by Bonkalo (1974). Sleep disorders generating sleep-related dangerousness are often influenced by drug intake including alcohol. The resulting behaviour is often preventable.

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INDEX TO VOLUME 148

We apologise for the delay in publication of the **Index to Volume 148 (January to June 1986)** which is now enclosed.

A HUNDRED YEARS AGO

"It might seem a painful thing to deprive of liberty persons who appear so little ailing, were it not that a repeated experience has shown it to be really a vain folly, if not a positive cruelty, to send them forth into the trials of life, when they are utterly unable to encounter them."

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Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey

Book Reviews

When Luck Runs Out: Help for Compulsive Gamblers and Their Families. By Robert Custer and Harry Milt. Oxford: Facts on File Publications. 1985. Pp. 239, £10.95.

This book, set very much in the American gambling scene, might be dismissed as inappropriate to the British reader. This is not the case because the problems it describes have their counterparts in this country. Legalised gambling and gaming has been available in a much more widespread form in Britain since the introduction of the Betting & Gaming Act 1960. In the way that where problems with alcohol are related to the availability of drinking so problems with gambling are related to the freedom with which gambling can occur amongst the population as a whole. Many members of society enjoy gambling as a pastime and recreation. They would object strongly to it being controlled or removed from their range of activities. The evidence is that rigorous control diverts gambling from legal to illegal participation and is therefore no solution to the problems of the few who become as dependent on gambling as others are dependent on alcohol. The Royal Commission on Gambling 1978 referred to this problem but felt that no recommendations could be made concerning any control since the size of the problem was not known but in any case was probably small. It offered the well worn cliché that more research is needed and did however recommend the setting up of a National Council on Gambling as one way of investigating the extent of the problem.

When Luck Runs Out describes vividly typical stories of compulsive gamblers. It gives a number of personal histories describing the effect on the individual, on the family and by implication on society. The authors bravely describe the personality and psychopathology of the compulsive gambler but recognise that while the traits and upbringing they describe may be common they are by no means universal. They do try to set out the typical picture of the individual but perhaps more important they describe those features by which the compulsive or pathological gambler may be recognised. So often, family doctors, social workers, psychiatrists, and probation officers recognise that there is a family problem, presenting perhaps as depression in the spouse, money shortages, disturbances in the children but not enquiring about the

possible cause; equally the family may be reluctant to divulge what is the hedonistic and unacceptable explanation of their problem.

A further chapter sets out the action which must be taken by the member of the family who identifies problems. Hopefully it will be the gambler who would recognise the difficulties and seek help. Much more commonly it will be the spouse and occasionally it will be the business associate who has been defrauded or otherwise put into financial difficulties who notices the problem and decides to take action. The role of Gamblers Anonymous and its related organisation Gam-Anon is seen as the cornerstone of treatment but brief mention is made of a psychotherapeutic approach linked with a referral to GA.

This book is a useful addition to the sparse literature on compulsive gambling and can be recommended to psychiatrists, social workers, probation officers, community psychiatric nurses and others whose lack of information about this syndrome may result in them overlooking the possibility of helping someone who is in need.

C. P. SEAGER, *Professor of Psychiatry, Northern General Hospital, Sheffield*

From Research to Clinical Practice: The Implications of Social and Developmental Research for Psychotherapy. Edited by George Stricker and Robert H. Keisner. New York: Plenum. 1985. Pp. 457. \$45.00.

Psychotherapy and research are uneasy, and at present controversial, acquaintances. The recent repetition of old, unproductive debates has highlighted the need for a new partnership. This book points to a more fertile mating by extending the notion of psychotherapy research to explore, as its subtitle suggests, the implications for psychotherapy of research in social and developmental psychology.

The book's editors attempt to show how knowledge of psychology research can refine the clinical practice of psychodynamic psychotherapy. In twenty invited chapters, relevant aspects of social psychology (eg leadership, non-verbal behaviour, aggression, and attitude change) and developmental psychology (eg infant and childhood development, gender role development, adolescence, and mid-life

change) are explored, and then a number of special topics examined (eg fantasy, religious experience, and psycho-linguistics).

As is inevitable in a multi-authored text, the contributions are variable in style and clarity. Each chapter concludes with a summary of general implications for practice, though these differ markedly in originality and applicability. More problematic is the discrepancy between the task and the essential belief system of some authors who attempt to write about psychodynamic application while resorting to narrow psychological principles. For example, Worchel and Worchel write about "aggression and psychodynamic therapy" from an essentially behavioural standpoint, without reference to Melanie Klein's work and with only passing reference to the important contributions of Kernberg to the elucidation of the central role of aggressive dynamics in much common character pathology.

I was more impressed by the application of research in developmental psychology, and in particular by Bloom-Feshbach's chapter on early childhood development. Here they relate research on child-rearing styles to attachment theory, illuminating the controversial debate about the importance of the therapist's warmth and empathy; and also elucidate some of the distinctive features and implications of the child's relationship with each parent.

Also of interest to me was the chapter by Nachmani on "psycho-linguistics and psycho-pathology" in which fundamental linguistic concepts are used to illuminate the contributions of Winnicott, Bion and Meltzer to the explanation of schizoid pathology, which is seen as a result of the disruption of a capacity to organise thought and express it through language.

There is something in this book then for every practising psychotherapist, as well as for those psychiatrists who are concerned with the role of psychological and social factors in generating and treating psychological disorder. The book should be available in all libraries, and is worth dipping into repeatedly.

MIKE HOBBS, *Consultant Psychotherapist,
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The Politics of Mental Health. By Ragnhild Banton, Paul Clifford, Stephen Frosh, Julian Lousada and Joanna Rosenthal, Basingstoke: Macmillan. 1985. Pp. 202. £20.00 (hb), £6.95 (pb).

Some twenty years ago, as a revitalised post-war psychiatry diffused outwards from its old asylum

base, it was met by the shrill voice of anti-psychiatry which in its various forms succeeded in condemning most aspects of psychiatric practice. Although anti-psychiatry is no longer so fashionable there remains a constant trickle of literature which attempts to build upon and extend the old critiques. This book is one of the latest and like its predecessors is a curious amalgam of insight, naivety and contradiction.

The analysis of contemporary mental illness develops along several interesting lines which seem to show the strong influence of Foucault. The authors, for instance, argue explicitly for a Foucaultian notion of power as coming from "below" (because it is a characteristic and product of all interactions) rather than power from above which simply reflects the dominance of one person or groups over another. They also adopt a social constructivist position with regard to the individual. Past critiques of psychiatry have stressed that mental illness is, in a sense, "constructed" by its social recognition and labelling; these authors employ parallel arguments to show that the individual, who is the focus of psychiatric intervention, has no essential or universal nature, but is similarly constructed in social interaction. And thirdly, they present a critique of the "community" which even in progressive psychiatry is usually little more than an idealised and romanticised "community remembered".

This critique might easily have led to a nihilist account of all psychiatry but these authors, all individual "mental health workers", outline an alternative radical therapeutic practice. In this scenario, however, much of the preceding critical analysis is somehow turned on its head. Thus the operation of power in its more traditional form of race, gender and class, it is argued, must be opposed. To be able to do this radical therapists apparently must themselves first be analysed: the authors regret that the new revolutionaries will have to fork out a lot of money for this in the capitalist market-place but such is the price of radicalism. These psycho-analytic skills—gained at such expense—would then be deployed in a community mental health programme, albeit a radical one which presumably ignores the fact that the "community" is itself a social construction of medicine over the last thirty years.

At a time when, apparently, "the forces of reaction threaten to wreck all vestiges of humane living" we are invited to join a "struggle that can contribute to the resistance against this upsurge in oppressiveness". Thus the therapist must enhance "subjectification" and replace silence with speech through intensive supervision of their clients: para-

doxically, in a world where humane living is so precarious there may be nothing more oppressive than this.

DAVID ARMSTRONG, *Senior Lecturer in Sociology, Guy's Hospital, London*

Depression in Multidisciplinary Perspective. Edited by Alfred Dean. New York: Brunner/Mazel. 1985. Pp. 245. \$27.50.

Despite increasing public subscription to the proposition that understanding depression requires a multifactorial approach, we all find difficulty in emerging from our particular corners to a mid-field viewing point and there is thus a continuing need for volumes such as this which make a panacea of the multidisciplinary perspective. Editing such collections however is not easy in the absence of more than a small number of really interdisciplinary studies. Volumes usually consist of a series of disparate contributions extolling the particular viewpoint from each corner—genetic studies, life events, personality characteristics and biological markers—barely welded together by the editor's introductory and concluding chapters. Alfred Dean avoids repeating this pattern, and has produced a book well worth adding to any collection of earlier such volumes. Issues involving treatment and diagnosis are given as much attention as aetiology and avoid the partisan flavour which occasionally, if unintentionally, accompanies the specialist contributions of experts from one special field. Even the chapter devoted specifically to cognitive behavioural treatments covers a whole range of such approaches, including the rather "less familiar" self-control methods of Rehm and colleagues; [and in outlining the theoretical rationale for these treatments it makes up for the rather hurried coverage of internal psychological resources by the editor's initial chapter on the epidemiology of depression]. One chapter gives a balanced discussion of the possible value of laboratory tests as a guide to prescribing—for example the DST is considered a better guide to length than to type of treatment. A similar caution about the DST emerges in other chapters, particularly in the one devoted to depression in the elderly where the difficulty of the diagnostic distinction between depression and dementia is discussed in detail (the DST has been found to relate to both, but computed tomography may help weed out the "pseudodementia's"). Diagnostic and treatment issues in childhood depression are given a similar thorough examination with similar conclusions: "ageism" at both extremes of the life course may

have exaggerated the degree to which affective disorders here differ from those found in the midrange of adulthood.

The section on psychosocial etiology has the merit of presenting studies with a relatively wide, if not completely multidisciplinary, range of variables, among them the editor's and his colleague's Albany-Troy-Schenectady prospective study. In all the standard simple combination of variables life-stress and support is supplemented by other factors such as mastery, personal competence, self-esteem, physical illness or life stage]. Furthermore the editor's own choice of the measure of "strong-tie social support" gives rise to a sensitive discussion of that area. However, some rather crucial issues seemed to be glossed over: one is the difficulty of establishing the time order of all these factors even in a longitudinal study. Little attention is paid to the need to exclude all with a depression whose onset predates the beginning of the study period, to cover life-stresses occurring right up to the time immediately before onset and to ensure that measures of psychological resources do not postdate (and thus possibly result from) such an onset. Moreover their use of the continuous CES-D score (instead of a cut-off at 16) is strangely at odds with some of the other contributions in the volume's diagnostic sections which stress the importance of distinguishing depressive symptoms from clinical caseness. A further issue—that life-changes as such predict depression less well than *undesirable* life events—only emerges sideways. Perhaps this is because the refinement of measures of different types of life events, and the findings concerning the predictive role of loss experiences, have tended to occur more on this side of the Atlantic—just another reminder of how many more multidisciplinary volumes we still need. But meanwhile this is a great help along the way, particularly in the fine lists of recent references which follow each chapter.

TIRREL HARRIS, *Department of Social Policy and Social Science, Royal Holloway and Bedford New College, University of London*

The Psychology of Schizophrenia. By John Cutting. Edinburgh: Churchill Livingstone. 1985. Pp. 475. £40.00.

In this book the author has bravely attempted to cover almost all the work ever done on the psychology of schizophrenia. His assumption seems to be that by presenting all this work (the fundamental facts as it were) in one place from an unbiased viewpoint a clear picture will emerge as to the true nature and cause of schizophrenia. I believe that this

"bottom-up" approach is misguided and has led to a disappointing book.

The first problem lies with the organisation of the material. Although the author speaks highly of the cognitive approach adopted in much recent research, he has chosen a very much older framework so that his chapters are divided into the faculties memory, attention, perception and so on. Much of the material he reviews sits uncomfortably on the boundaries of these categories. For example, Broadbent's 1958 filter model on which much schizophrenia research is based, attempts to explain *common* aspects of attention, memory and perception.

The second problem lies with the selection of the material. With so much to be covered in so little space the author has to be selective in what he reports. Given his neutral standpoint, however, he has no criteria for deciding which is the most important aspect of each study. Every reader is bound to disagree with some of the author's selections although for different reasons. To take just one example, Goldberg *et al* (1981) are quoted in relation to the "alien hand sign", but their conclusion that this is due to unilateral frontal rather than corpus callosum lesions is not mentioned.

The third problem lies with the lack of an overall structure for the conclusions from the various sections. There are many details in this book which are fascinating and well worth reading, but they do not add up to a coherent story. For example the author suggests that first rank symptoms have been over-emphasised in the definition of acute schizophrenia, and also that chronic schizophrenia should be treated as a separate entity. Even if they are incorrect, these ideas could have been used with advantage in discussing other aspects of research, but the author has not systematically adopted such a framework.

The author's final conclusion is that acute schizophrenia is due to some unspecified, but relatively discrete brain damage (labelled "hemisphere imbalance") whereas chronic schizophrenia is caused by some unspecified, but more generalised brain damage. It is not at clear to me how these disorders of the brain relate to the symptoms of schizophrenia or to the results of the hundreds of psychological experiments reviewed.

I believe the author could have written a much better book if he had used a "top-down" approach, reviewing the literature from some definite and biased viewpoint. Furthermore since his book is entitled the psychology of schizophrenia I would have expected the final conclusions to be in terms of psychology rather than the brain. It is perfectly possible to understand schizophrenia or any other mental illness in terms of some psychological deficit

without specifying the underlying damage to the brain. In the case of schizophrenia such specification is premature and cannot be derived solely from psychological studies.

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The Social Science Encyclopaedia. Edited by Adam Kuper and Jessica Kuper. London: Routledge & Kegan Paul. 1985. Pp. 916. £29.95.

I've never actually read an encyclopaedia before, preferring to keep it for my desert island. Nor, of course, can anyone actually read through an encyclopaedia from Abnormal Psychology to Wilhelm Wundt, not at least with understanding or authority. However, I found myself carried along by this stimulating and interesting book; it is lovely to get away from the tunnel vision of one's professional reading.

I took my samples from areas I know reasonably well, from areas I know a bit and would like to know more, and from areas I have always found difficult (phenomenology, sociobiology and Piaget, for example). The many biographies complement the articles and make compelling reading—even about people one didn't particularly wish to know about! Rather like the compulsive qualities of a Radio Three talk, listened to by accident during the concert interval.

Five hundred contributors from 25 countries provide 700 entries: the editors must be superpersons! The major social sciences—anthropology, economics, political science, political theory, psychology and sociology—have master entries of immense learning; good examples of the perennial lure for academics to compose outstanding essays to put in relatively obscure places. None more so than Ralf Dahrendorf's "Social Science", a 1500 word essay of breathtaking scope, scholasticism and coruscating intellectuality.

If you are middle-aged and intellectually burned out or young and looking for something different I can thoroughly recommend this book. Not, unfortunately, for the psychiatry, whose entries are banal and poorly informed (schizophrenia, suicide, neuroses, ECT, for example). One can only wonder how they could be composed let alone accepted. However, of the areas closest, psychology and sociology are excellently done. One persistent impression is the abiding influence on the core social sciences of Karl Marx. The entry: "Marx's theory of history and society" is fascinating and one wonders how it is that his impact on psychiatry—even social and community psychiatry—has been minimal.

So, while one cannot have resource to the book reviewer's perennial recommendation "for the departmental library", this is certainly a book for the family library: interesting, clear, well printed and bound, and for these days not over-priced.

SIDNEY CROWN, *Consultant Psychiatrist, The London Hospital*

Mental Handicap: A Multi-Disciplinary Approach.

Edited by Michael Craft, Joan Bicknell and Sheila Hollins. London. Baillière Tindall. Pp. 421. £30.00.

This outstanding book consists of a series of essays by a team of experts involved in practical provision for the mentally handicapped. In her introduction, Professor Joan Bicknell refers to the book as a successor to Tredgold's classic text. This may be so in that the publishers are the same and that this bids fair to become the standard text on the subject. But this new work, as Professor Bicknell says, reflects an entirely fresh approach. Public and professional opinion has undergone a U-turn in regard to intellectual handicap since Tredgold's time. One aspect of this change of course, as Professor Bicknell points out, is reflected in the choice of a multi-disciplinary team to write this book instead of relying on the "medical model" as previously.

Tredgold was an enthusiastic original member of the Eugenics Society, believing in Morel's doctrine of national degeneration, as modified by Mott and others. In 1909 he warned that, in relation to mental defect, the chief evil was that of "propagation". It was eugenic concern of this kind which in part led to the very restrictive Mental Deficiency Act of 1913 and to the steep increase in institutional population of the mentally handicapped which followed the First World War.

In contrast to these attitudes is the commitment of all the writers in the present volume to community care and the sympathetic and helpful chapter on 'Sexuality and Personal Relationships' by Ann and Michael Craft. Malcolm Johnson, in his chapter on 'Social Policy and Service Development' refers briefly to the epidemiology of mental handicap, mentioning the paucity of reliable statistics and difficulty of definition; though there is more agreement about the more severely mentally handicapped. It seems that the effect on prevalence of better life expectation may be balanced by prevention. Michael Craft in his chapter on "Classification, Criteria, Epidemiology and Causation" deals more fully with incidence and prevalence, pointing out that, even in the well defined entity of Down's syndrome, the situation is not clear. Offsetting

increased life expectation is a reduced incidence due mainly to lowering of maternal age, associated with more frequent termination of pregnancy in older women on grounds of age. Craft points to the limitations of assessment in the mildly retarded, particularly when used as a prognosis for future intellectual development.

Each chapter in this book is clearly written and the editors are to be congratulated on the success of their efforts. The presentation is appropriate for students in all the disciplines involved and the text is suitable for reference.

Malcolm Johnson, writing from his chair at the Open University, points to an obvious shortcoming in the present service when he regrets the lack of any informal care network to link different agencies and units. He says that money is not enough, but he might equally have said "There is not enough money", for he names a succession of ministers who have failed to deliver the goods.

BRIAN KIRMAN, *Formerly Consultant Psychiatrist, Queen Mary's Hospital for Children, Surrey*

Overcoming Resistance: Rational Emotive Therapy with Difficult Clients. By ALBERT ELLIS. New York: Springer Publishing. 1985 Pp 228. \$21.60.

Ellis is the originator of Rational-Emotive Therapy (RET), one of the earliest approaches to cognitive behaviour therapy and this book is devoted to the question of resistance in therapy. Although the book is addressed mainly to RET practitioners, it contains sufficient background information to be comprehensible to therapists of other orientations.

The many forms in which resistance may be encountered are examined in detail. A distinction is made between "normal" resistance, which can be expected to occur frequently as an intrinsic part of the process of changing, and other types of resistance which require special attention if therapy is to be successful. Ellis concentrates on resistance by patients with more serious disturbances such as borderline personalities and alcoholics. He offers an array of techniques and practical suggestions, with numerous examples from his extensive clinical experience. These range from the forceful methods which are characteristic of RET, to the more gently persuasive methods advocated by cognitive therapists such as Meichenbaum. Of particular interest is the discussion of ways in which the therapist himself may cause or contribute to the patient's resistance, and how this may be tackled.

One negative aspect is in the book's presentation. There is a considerable amount of repetition and self-congratulation in Ellis's style of writing which readers may find off-putting. In spite of this, however, the book deserves attention for its useful contribution to this important but neglected topic.

ANNE GODDARD, *Lecturer in Clinical Psychology, The London Hospital Medical College*

Secure Provision: A Review of Special Services for the Mentally Ill and Mentally Handicapped in England and Wales. Edited by LARRY GOSTIN. London: Tavistock. 1985. Pp. 318. £14.00 (hb), £7.95 (pb).

This book is divided into four parts: a history of the development of secure provisions, services, the law, and a discussion of concepts of dangerousness and social control.

The issues covered are relevant not only to forensic psychiatrists but also to general psychiatrists. Faulk argues that much of the anxiety felt by general psychiatrists about the care of the 'dangerous' patient is due to lack of contact with them. Special Hospitals are covered by Hamilton in the second part of the book; particularly worrying is the increasing difficulty found in transferring patients from Special Hospitals, the average period of time to wait now being several years.

Professor Gunn outlines five fundamental problems within the prison medical service: recruitment and training, compulsory treatment of mentally abnormal offenders, the production of court reports, widespread prejudice towards doctors who practise in this setting and the ethical conflicts this creates in the doctor, and finally the ever increasing number of individuals the prison medical service is required to deal with.

Regional secure units developed largely in response to the 'yawning gap' between secure Special Hospitals at one extreme and the NHS hospitals at the other, which have been identified by the Butler and later Glancy Reports. The historical development and current state of regional secure units are discussed by Professor Bluglass, and current practice in these units by Treaseden, who shows how they have narrowed but not yet completely closed the aforementioned gap. The legalistic approach towards psychiatric practice is adopted by Hoggett in part three of the book. I shall largely gloss over this section, which I felt was overly technical and legalistic with a distinctly 'thou shalt not' tone. It was with relief, then, to turn to Bowden's entertaining exposition on issues in which dogmatism and certainty have little place, i.e. what is dangerousness?, the relationship between mental disorder and dangerousness, the relationship between victim and offender, and questioning whether forensic psychiatrists are aspiring to an expertise that in reality they do not possess. In a somewhat disturbing but constantly engrossing and thought-provoking final essay, Bean argues that secure provision for mental patients are the mirror image of the prison in the legal system. The view of psychiatrists as agents of social control is a role which most psychiatrists are reluctant to acknowledge. However, it is perhaps a responsibility that should not be shed away from. Control can be less damaging ultimately to the patient than a policy of radical non-intervention, allowing the patient to "die with their rights on", which the legal approach threatens to create. (From penal/legal systems). . . "they will always be on the welfare of their patients, rather than, say, a strict demand for legal justice".

GILLIAN MEZEY, Registrar, The Maudsley Hospital, London

Sleep and its Disorders. By J. D. PARKES. Eastbourne: Holt-Saunders. 1985. Pp. 499. £29.50.

Sleep because it is a universal experience rarely fails to fascinate the reader, and its disorders often present difficult diagnostic and treatment problems. Hence the immediate appeal of a new book on the subject. It is part of a series on *Major Problems in Neurology* edited by Sir John Walton. This volume is by a single author who has a wide experience in the field, both from a practical and research point of view, and can thus be expected to offer an up-to-date perceptive view, which is amply fulfilled. The chapters are grouped into three main sections, "Normal Sleep", "Sleep Disorders" and "Pharmacology and Sleep", each in a wealth of detail with ample bibliography. The first, occupying one third of the book, provides an account of normal sleep and its variants, its anatomico-physiological basis, and the relationship between circadian rhythms and sleep. Each is divided by many side headings providing helpful pointers, particularly for the reader wishing to gain access to the text from the valuable index.

The comprehensiveness is emphasised for example by sub-sections on shiftwork, jet lag and hormones in relation to sleep, again sub-divided. The chapter on parasomnias covers a wide range of topics such as sleep myoclonus, sleep paralysis, and sleep walking where historical asides, an account of the phenomena and possible treatment are given. Some may feel that the text is skimpy in parts, including those on psychiatric matters, but appropriate references are given when amplification is required. The chapters on daytime sleepiness and sleep apnoea furnish the expected range of views enlivened by historical allusions and useful tabulations, characteristic of much of the book, as well as illustrations, some in colour. The treatment section on the use of hypnotics and stimulants is somewhat disappointing, though there are comprehensive listings, an overall perspective does not emerge. Somewhat disappointing also is the abrupt end of the book, after a section on the treatment of cataplexy. A final chapter on where research is leading, what new insights on sleep apnoea are likely to emerge, for example in relation to cot deaths, would greatly enhance the book, which is otherwise recommended both for the general and specialist reader.

D. F. SCOTT, Section of Neurological Sciences, The London Hospital

Recovery from Schizophrenia: Psychiatry and Political Economy. By RICHARD WARNER. London: Routledge & Kegan Paul. 1985. Pp. 380. £25.00.

"To render schizophrenia benign we may, in essence, have to restructure western society." The last sentence of this book suggests a political polemic but it is much more than this. As well as a useful and comprehensive review of such familiar areas as problems of diagnosis, prevalence, treatment and prognosis, the reader is urged to consider schizophrenia in a broader context than that of the traditional clinical and family approach. The author's central theme is of the hitherto undervalued contribution of

"political economy" in the production and perpetuation of schizophrenia.

The author identifies a link between unemployment and adverse outcome of schizophrenia. He also speculates that the competitive pressures of working in an industrial society may be inimical for a recovering schizophrenic, while a role in a non-industrial community based on subsistence needs is less demanding and more appropriate. This may contribute to the better outcome of schizophrenia in developing countries where it is also facilitated by the lack of stigma and alienation endured by the schizophrenic and his relatives in the West. The author also believes that upper class individuals in the Third World are more at risk of the illness but, as industrialisation approaches, this social class gradient switches round to conform to the pattern familiar in the West.

An important debate which is aired concerns the place of antipsychotic medication. The argument is put forward that, while such drugs have value in management, the outcome of schizophrenia has not improved since their introduction. Deinstitutionalization in Britain began before they appeared and was largely due to social therapies and the stimulus given to rehabilitation by the labour shortage after the war. Medication is considered to be unnecessary in a significant proportion of patients and to increase the risk of relapse in drug-withdrawn patients. The concern is expressed that antipsychotic drugs have, in some community care programmes, been used as alternatives rather than adjuncts to psychosocial care.

The author writes with passion and concern in a highly readable and stimulating way and the reader is challenged to clarify his own views about schizophrenia in the course of a highly recommended read.

ALAN KERR, *Consultant Psychiatrist, St Nicholas Hospital, Newcastle upon Tyne*

Bisexualities: Theory and Research. Edited by FRITZ KLEIN and TIMOTHY J. WOLF. New York: The Haworth Press 1985. Pp. 255. \$24.95 (hb), \$16.95 (pb).

A new liberation movement has just started. Another sexual minority group are desperately looking for an identity: the bisexuals. Rejected by heterosexuals for being a threat to monogamy and persecuted by homosexuals for sitting on the fence and not admitting to their homosexuality, they are closing their ranks and encouraging each other to "come out of the closet" and announce their bisexuality. Little wonder that this book has far more politics than science. It challenges many sexual dogmas and questions some of the widely accepted views on what is considered 'natural'. It does so, very convincingly at times, very passionately always, but with very little serious research to back up its claims. The book does, however, give some interesting statistics: 10-15% of the population are bisexuals, there are thirty to forty million bisexuals in the USA, 10% of married males also participate in some homosexual activity and 18% of males demonstrate as much homosexual as heterosexual behaviour during their lives.

For many people, including some professionals, bisexuality is nothing more than a gender confusion or psychological hermaphroditism. If you accept the statistics quoted above and the results of some of the research papers published in the book (one of them had a sample of 18 cases), you will have no difficulty in accepting some of their conclusions. You will accept that 'bisexuals are not rare, only rarely identified', 'bisexuality is the norm, exhaustive homo or heterosexuals are moved away from the norm by cultural conditioning', that 'bisexuality can be life enhancing... can facilitate marital adjustment' and will even believe that bisexuals are lucky because they 'can enjoy the best of two worlds'.

Whatever you think of these conclusions, one thing is certain, they will make you doubt some of the widely held beliefs about what is considered by the majority as 'normal'.

The book ends by an account of the growing bisexual organisations in New York, San Francisco and Chicago and an exhaustive bibliography of 400 references on bisexuality.

This book can be an eye-opener for those who feel uncomfortable with non-traditional sexual behaviour, has little practical value for the general psychiatrist, but can be an important source for those who are actively involved in gay counselling or working with other sexual minority groups.

WAGUIH R. GUIRGUIS, *Consultant Psychiatrist, Psychosexual Clinic, St Clements Hospital, Ipswich*

Spheres of Group Analysis. Edited by T. E. LEAR. London: Group-Analytic Society. 1985. Pp. 141. £5.50.

In the preface to this short but readable account of group analytic psychotherapy in practice, the editor acknowledged our debt to the teaching of S.H. Foulkes. This book is like a group analytic group. The individual contributors include an educationalist, a social scientist, a chaplain, a nurse, and several psychiatrists whose fields of practice are as diverse as alcoholism and sexual deviancy. Each chapter is interesting and worthwhile in its own right, but together they are more than the sum of the individual's experience. They are united by a common theoretical approach, stressing the potential of the group to value and strengthen the unique contribution of each individual.

The initial chapters provide a theoretical perspective which is complemented by others on large groups and uses in a family therapy setting. Multi-author books are often weakened by their diversity of style and content. In this case the diversity only demonstrates the broad ranging applicability of the group analytic approach.

This book will be useful for students on the Institute training courses but also can be expected to appeal to a much wider general audience.

I. F. MACILWAIN, *Consultant Psychiatrist, Netherne/St George's Hospital, London*

Alcohol and Aggression. Edited by PAUL F. BRAIN. London: Croom Helm. 1985. Pp. 228. £22.50.

"The Drunkard, in his drunkenness, killeth his friend, revileth his lover, discloseth secrets and regardeth no man." The Elizabethan author of the *Anatomie of Abuses* might not have believed that by 1985 to attribute aggressive behaviour to alcohol might be seen as distorted thinking generated by 19th century capitalists' desires (couched as the Temperance Movement) for greater worker discipline. He might also have been surprised on reading this impressive compilation that even the explosion of 20th century science still leaves the question of whether alcohol increases aggression as a matter of if and but.

Even if he could not quite follow studies of alcohol-related changes in latency of cats' quiet biting attacks, or the Convict Cichlid's response to male conspecifics, or the influence of alcohol on GABA receptors, testosterone levels and nerve cell membranes, he would be impressed at the breadth of physiological, behavioural and pharmacological research reviewed here. Both he and a 20th century psychiatrist might regret that more human individual and clinical data could not be included. How do drunken assaults unfold? The epidemiological and sociocultural view of the association between alcohol and interpersonal violence, though well covered, leads to a slight weighting of the evidence in favour of drunkenness as an excuse for wife beating (for example) rather than wife beating being a result of drunkenness.

Dr Brain is at pains to point out that aggression is a complex notion and covers a wide range of behaviours. In the same animal, alcohol may increase one type of aggressive behaviour and decrease another, and the effects may be reversed as dosage changes. Ethological studies hold promise. For example, it has been possible to show that alcohol increases aggressive behaviours in submissive monkeys and dogs in the group, but not in the dominant monkey. Interestingly, if the dominant male monkey becomes intoxicated there is a relaxing of social restraints—a phenomenon for study at the medical staff dinner? But we are warned of the risks of extrapolation. Contributors Berry and Smoothy quote P. G. Wodehouse. "No more alcohol for me," said Buffy. "Look what it does to the common earth worm." "But you are not a common earth worm," I said, putting my finger on the flaw in his argument right away.

JONATHAN CHICK, *Consultant Psychiatrist, Royal Edinburgh Hospital*

The Culture-Bound Syndromes: Folk Illnesses of Psychiatric and Anthropological Interest. Edited by RONALD C. SMONS and CHARLES C. HUGHES. Dordrecht, The Netherlands: D Reidel. 1985. Pp. 516. £54.25, US\$69 00, Dfl. 195.00.

The contributors to this volume on folk illness are psychiatrists, sociologists and anthropologists. Much of the bizarre behaviour described, albeit shaped by the cultural background in which it occurs, may be found in any psychiatric text-book but difficulties arise when attempts are made to categorise culture-bound disorders in accordance

with western nosology. Although it is true that none of the three versions of the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association can conceptually accommodate the syndromes described, I suspect that if there were a third world equivalent of the DSM-III similar difficulties in classification would arise with disorders familiar to western psychiatry. Ethnocentric bias in medicine probably correlates well with levels of misunderstanding elsewhere. It says a great deal for the combined wisdom of the editors (one a psychiatrist and the other an anthropologist) that this problem is addressed and in many instances resolved. Unfortunately there are occasions when emphasis on semantics blurs rather than clarifies meaning.

When modern diagnostic criteria and methods are available, folk beliefs and supernatural explanations for them fade. They can then be understood more readily in neurophysiological and biological terms. One example is *old hag*, a syndrome described in a small hitherto isolated community in Newfoundland and characterised by a paralysis of arms and legs with hallucinations. It became easier for the diagnosis of sleep paralysis to be made after a road was built yielding access to a medical centre.

Hysterical phenomena and psychotic behaviour are frequently found in connection with the culture-bound syndromes. It seems highly likely that media influences shape and spread disturbed behaviour. Indeed *amok*, a violent psychosis peculiar to the Malays, may be difficult to distinguish from mass murder carried out by those whose personality deficits are exaggerated by chemical intoxication. Indiscriminate terrorism reminiscent of *amok* and based on group expectations has already been exported to international airports. *Koro* or the genital retraction syndrome appears as a 'neurotic' epidemic in South East Asia, but it would be interesting to speculate what would bring the universal fear of the shrinking penis to the surface of Western man.

The strength of this volume lies in its understated plea for the transcultural understanding needed in our multi-ethnic society. I would strongly recommend it to all psychiatrists working with minority groups.

DENNIS FRIEDMAN, *Medical Director, Nightingale BUPA Hospital, London*

Emotions, Cognition and Behaviour. Edited by CARROLL E. IZARD, JEROME KAGAN, and ROBERT B. ZAJONC

Noting the recent and dramatic growth in the school of cognitive psychology, the editors felt that this trend should be paralleled by a similar emphasis on emotional and social development. Hence the need for this book, which arose from a series of American discussion groups on child psychology. Eighteen chapters describe recent empirical and theoretical work aimed at furthering our understanding of the relationship between emotion and cognition in the developing child.

Particularly interesting among the theoretical issues are chapters on the interaction of emotion and thought in the development of empathy, the distinction between attributing failure to lack of effort (leading to guilt) or to others

(leading to anger), and the relevance of good versus poor imagery in the development of phobias. Other interesting chapters present evidence that the frontal region of the left hemisphere is concerned with pleasant emotions, that an abundance of positive emotions facilitates the development of altruism, and that hypnotically induced emotion can influence learning and memory.

There is a wealth of important material in this book, but it is difficult to find, being hidden in some very long and detailed accounts of research. The editors provide an introductory chapter which is little more than a non-evaluative overview. It would have helped to give more of a commentary on each chapter, drawing attention to the highlights or, at least, to have drawn together the important advances in a concluding chapter. Because this has not been done, my impression is that few people will take the trouble to sift carefully through this book which can reveal some fascinating insights into the ways that children develop

DAVID K. B. NIAS, *Senior Psychologist, Darenth Park Hospital, Kent*

Theory and Treatment of Anorexia Nervosa and Bulimia: Biomedical, Sociocultural, and Psychological Perspectives. Edited by STEVEN WILEY EMMETT. New York: Brunner/Mazel. 1985. Pp. 332. \$30.00.

This is an American compilation issue. It consists of 17 chapters by authors of a wide variety of professional backgrounds put together by a minister of the church who is also a psychotherapist specialising in the treatment of eating disorders. The volume is divided into three sections which cover biomedical, sociocultural and psychological perspectives of anorexia nervosa and bulimia followed by a concluding section.

The quality is very uneven and mixed. Some chapters, notably those by Spack on Medical Complications of Anorexia Nervosa and Bulimia and the one by Copeland on Neuroendocrine Aspects of Eating Disorders, provide detailed and comprehensive reviews of the literature in these areas. Others are much more personal and reflect the viewpoint of the author, for example, Orbach's interesting chapter giving a feminist perspective of anorexia nervosa and White Jr.'s chapter on intervention strategies and outcome considerations give synopses of their work.

The book contains a great deal of information but unfortunately it is not presented in an orderly way and therefore does not hang well together. A fundamental fault is the failure to provide definitions for the terms 'anorexia nervosa' and 'bulimia' and it is by no means certain that each author utilises the same diagnostic criteria. Indeed, several chapters do not attempt to distinguish these conditions.

Although the label bulimia nervosa was originally coined to describe a variant of anorexia nervosa, the diagnosis has established itself as a distinct disorder. Some patients have a past history of anorexia nervosa and others do not. There still remains considerable confusion and uncertainty concerning the most appropriate diagnostic label to attach to those patients who have suffered from anorexia nervosa and who binge and vomit, especially if

their weight approaches the average range. It is therefore of the greatest importance to clarify the diagnostic criteria in respect of bulimia nervosa and anorexia nervosa if the results of research in these areas are to produce fruitful distinctions. Unfortunately this book has fudged this clarification of such a fundamental issue although it contains a range of interesting perspectives.

EDWARD STONEHILL, *Consultant Psychiatrist, Central Middlesex Hospital, London*

Literature and Medicine. Vol. 4. Psychiatry and Literature.

By PETER W. GRAHAM. Baltimore, Maryland: Johns Hopkins University Press. 1985. Pp. 173. Subscription: \$12.95 (individual), \$25.00 (institutions).

"Our criticisms, from age to age, reflect the things that the age demands". The application of T. S. Eliot's dictum to this collection suggests a demand for rather stodgy fare.

This volume consists of a series of disparate essays, on the broad theme of the connection between psychiatry and literature; and in particular between "madness" and authorship. It gets off to a bad start with a contribution by Thomas Szasz, the originator of 'Conventional Western Psychiatry'—the good commercial guys wearing pale hats and the bad institutional guys wearing dark ones. The bullets duly whistle past and through the dummies stuffed with false antitheses, but no real blood is drawn and our hero, I fear, will live to make many more 'B' movies.

There follow several studies of individual authors: Holderlin, Ezra Pound, Hayden Carruth, Hilda Doolittle and Mary Kempe. In so far as they bring out some obscure authors for occasional inspection, these essays are valuable, but they are written in the solemn Eng. Lit. style, as if Academe was breathing in the vast thick pants of expiring PhD theses. There is the occasional stimulus of disagreement. The essay on Pound quotes one of his beautiful and allusive translations of oriental lyrics—*The Jewel Stairs' Grievance*. It is condensed and impressionistic. The critic concludes, from the fact that 'it refuses to advertise its meaning', that 'it is a poem of and for a paranoid mind'. Well really! This clumsy Malleus Poetorum would strike down a whole bookshelf of poets, including our own genial Will Shakespeare himself.

J. L. T. BIRLEY, *Consultant Psychiatrist, The Maudsley Hospital, London*

Mind and Body: The Psychology of Physical Illness. By STEPHEN A. GREEN. Washington: American Psychiatric Press. 1985. Pp. 205. \$22.50.

The author's aim is commendable: "Proper medical treatment requires an understanding of what illness means to a particular individual at a particular time in his life. This . . . requires listening to patients and understanding what they communicate in words and by their silence. This book is designed as a framework for achieving such an understanding." Central to the approach adopted by Dr Green is his belief that abnormal psychological responses occur when the patient is unable to grieve effectively the losses incurred by ill-health. That theme is discussed at length. In

the remainder of the book, common responses to major physical illness are described and the concept of "illness dynamics" is outlined.

Of interest are those chapters in which the author presents extracts from his psychotherapeutic interviews with patients whose psychological responses to a variety of physical illnesses include denial, anxiety, anger, depression and helplessness. On reading these *verbatim* extracts, one gets a strong impression of the author's genuine warmth, empathy and considerable clinical skills.

Less satisfactory are Dr Green's excursions into theory and the advice he proffers regarding therapy. In the theoretical discussions, readers will look in vain for clearly stated, refutable hypotheses derived from factual observations. Instead, psychoanalytically based insights are presented with an air of certainty which belies the flimsy anecdotal evidence upon which they depend. Moreover, these insights are not merely harmless flights of fancy; Dr Green is convinced that, for at least some medically ill patients, what is required is "a relentless probing of unconscious material". The mind boggles. In your reviewer's opinion, patients suffering from serious physical illness require relentless probing of their unconscious like a hole in the head.

STEVEN GREER, *Reader in Psychological Medicine, King's College Hospital, London*

Principles of Behavioural Neurology. By M-MARSEL MASULAM. Philadelphia: F. A. Davis. 1985. Pp. 405. \$68.75

This volume is one of the much valued black volumes in the Contemporary Neurology series, the Editor-in-Chief of which is Fred Plum. Other useful volumes of interest to psychiatrists in the series have included "Management of Head Injuries", edited by Jennett and Teasdale, and "Mental Retardation and Related Disorders", edited by Charles Barlow.

The present volume is certainly a worthy member of the series, and gathers together chapters by distinguished authors on a variety of topics in what might be termed "higher mental function", which no longer find an easy home in texts of either neurology or psychiatry. However, the enormous territory to be covered leads to rather skimpy treatment of some important areas. For example, a neurologist or a psychiatrist who wishes to know more about the current thinking of those with a special interest in memory might well do better to go to some paperback written for the general reader with an interest in this subject.

A useful chapter is from the Editor, Dr M-Marsel Masulam, on the phenomenon of attention. It is intriguing, to say the least, that the performance of patients with unilateral neglect of space to the left can be improved by financial reward. This is a good example of the central theme of the book—attempts to understand the integration of the behaviour, unseen cognitive processes and feelings of the individual with his disordered brain.

In his preface, the Editor reassures psychiatrists that there is no inexorable movement towards reclassifying all

psychiatric diseases within the realm of neurology. He writes that, for the vast majority of patients who seek psychiatric help, a neurological approach is no more useful, or desirable, than a chemical analysis of the ink would be for deciphering the meaning of a message. However, new discoveries on the cerebral organisation of emotion and personality are prompting the inclusion of neurological causes into the differential diagnosis of many conditions that have traditionally been attributed to idiopathic psychiatric disorders.

ANTHONY HOPKINS, *Physician in Charge, Department of Neurology, St Bartholomew's Hospital, London*

Mental Health Care in the European Community. Edited by STEEN P. MANGEN. London: Croom Helm. 1985. Pp. 278. £16.95.

Steen Mangen has compiled an account of the mental health policies of the (then) nine countries of the Common Market. The authors, who come from different mental health disciplines, are often natives of the countries whose policies they describe. The editor contributes a most interesting introductory survey of the European scene and chapters on the United Kingdom and Germany. Some of the other chapters are less well written, although Professor Stromgren's chapter on Denmark is particularly interesting and readable. The book is produced in camera-ready form and there are too many typographical errors.

In spite of its multilingual origins, the English is generally good.

Although we have shed some of our traditional insularity we still know little of the social policies which influence the work and the remuneration of our European colleagues. Those who take the NHS, the Mental Health Acts and the declining populations of mental hospitals for granted, will be interested, perhaps surprised, to read of the complicated insurance systems, archaic mental health legislation and the hospital provision of other countries.

This book, like travel itself, broadens our outlook and in some ways it is a book for the specialist the general reader will find much to interest him. Everyone should read Steen Mangen's excellent introductory chapter.

DOUGLAS BENNETT, *Emeritus Physician, Bethlem Royal and Maudsley Hospitals*

Textbook of Child Neurology. 3rd edition. By JOHN H. MENKES. Philadelphia: Lea & Febiger. 1985. Pp. 827. \$64.25.

This is the third edition of a well-established textbook of *Child Neurology* first published in 1974. Chapters are organised predominantly according to aetiology, for example hereditary degenerative diseases, chromosome anomalies, toxic and nutritional disorders and tumours of the nervous system, with a final chapter by Kinsbourne on disorders of mental development. Presentation is systematic for specific disorders covering pathology, clinical manifestations, diagnosis and treatment. Typography layout and production are good.

Since the last edition there has been significant updating in relation to new diagnostic procedures and syndromes, and there is a newly added chapter on neurological manifestations of systemic disease.

Throughout, this text is probably stronger on pathology than clinical descriptions or treatment. For clinical descriptions of hereditary disorders psychiatrists may find other more liberally and appropriately illustrated, and more clinically focused texts more useful. With regard to treatment there is almost no mention of genetic counselling (it is not even indexed) and nothing about the impact of a handicapped or chronically ill child on his family. This is regrettable. Furthermore there is remarkably little mention of associated emotional and behavioural disorders and their treatment even in Kinsbourne's chapter. This final chapter on disorders of mental development is in many respects balanced but noticeably more illuminating in the author's areas of special interest: learning disorders and hyperactivity. There are fewer up-to-date references on for example infantile autism and malnutrition. Curiously absent is an emphasis on the relevance of a broad-based child psychiatric assessment in learning disability and hyperactivity.

A. D. COX, *Senior Lecturer in Child Psychiatry, Institute of Psychiatry, London*

Treatment of the Borderline Personality. By PATRICIA M. CHATHAM. New York: Jason Aronson. 1985. Pp. 585. \$40.00.

It won't be long before we need a guide book not only to borderline states but to books on borderline states. It does actually seem that the more books we have and the fatter they are, the more diffuse and vague the conclusions will be.

Many of these volumes are serious and helpful but it is seldom that they add anything new. This particular book, like many of the others, is by a clinical psychologist in America and has many of the virtues of the systematic thinking in which American universities excel. The author describes the borderline patient from the point of view of object relations theory as understood by Kernberg and people influenced by him. There is quite a lot of emphasis on developmental theory, especially that of Margaret Mahler, and some account of Kohut's theories and their relevance.

It is interesting to examine the index to get some idea of the author's sources of influence. Sigmund Freud is mentioned nine times, Anna Freud four times, and Melanie Klein ten times. Of more modern writers Winnicott is mentioned eighteen times, Bion four times, Rosenfeld three times and Balint three times. On the other hand, Kohut is mentioned fifty-five times, Masterson ninety-three times, Rinsley one hundred and forty-two times, and Kernberg two hundred and nineteen times. This seems a strange emphasis to an English psychoanalyst but I think is probably entirely typical of current American thinking.

It would be easy to be unfair to this book, and if it were the only one of its kind I think it would be quite useful.

Unfortunately there are so many similar ones that it would be difficult to justify spending forty dollars. And even if you did, I am not sure you would know when to apply supportive psychotherapy leading to intensive psychotherapy or supportive confrontative psychotherapy. The Americans think we are very unsystematic, but I find it a relief to simply think of psychoanalytic psychotherapy which we try to apply even with difficult patients. If we take care over the setting and rely on co-operation and help from psychiatrists, social workers and other colleagues, I think many patients can be helped without the need to subdivide the types of therapy we employ.

JOHN STEINER, *Consultant Psychotherapist, The Tavistock Clinic, London*

Eating Disorders: Prevalence and Treatment. Edited by S. W. TOUYZ and P. J. V. BEUMONT. Sidney: Williams & Wilkins 1985. Pp. 160. £24.

This book is based on a symposium on eating disorders held at the University of Sydney. The contributors, with one exception, work in Australia and come from a variety of disciplines, although the overall emphasis of the book is psychiatric/psychological.

The first section is on anorexia nervosa and includes a useful chapter by Touyz and Beumont which describes in detail a "comprehensive, multidisciplinary" approach to management. A relatively novel feature of this approach is a more "lenient" behavioural programme in which patients have greater control over the refeeding process. The programme was as effective as more conventional programmes in terms of weight gain and also was associated with higher patient motivation and was more economical in terms of nursing care. Other chapters consider general aspects of the disorder, nutritional counselling and criteria for recovery.

The second section on bulimia includes chapters on prevalence and classification. In terms of treatment, Suzanne Abraham and colleagues favour a flexible "eclectic" approach and Gail Huon presents some preliminary information on "therapy at a distance", i.e. by mail, with particular emphasis on help-seeking, compliance and drop-out.

The final section on obesity covers epidemiology, the metabolic basis, the effectiveness of psychological treatments and the psychological factors in current surgical procedures. There is an interesting chapter by John Collins on a careful appraisal of the use of hypnosis, which produced encouraging improvement both in terms of weight loss and in terms of psychological factors such as body image and body concern. The book is well written with a nice combination of research evidence and practical advice on management.

Its principal drawback, however, is its price. Even at today's prices, £24 for a 160 page paperback seems excessive.

ERIC BUTTON, *Lecturer in Clinical Psychology, Royal South Hants Hospital, Southampton*

Psychotherapy and the Terrorised Patient. Edited by E. MARK STERN. New York: The Haworth Press. 1985. Pp. 116. \$16.95.

The thirteen brief contributions to this book (mainly by clinical psychologists in private practice) are also published as Vol 4, No. 1. (Summer, 1985) of "The Psychotherapy Patient". The book reads like one of those meals which comprise a series of small dishes. Each contributor says what he/she wants to say, then stops. Though the editor, in the first two contributions, sets out precisely to define the term 'terrorised', most of the succeeding contributors adopt a fairly liberal interpretation: which is all to the good, since it enables them to develop their own idiosyncratic themes.

Mahrer *et al* echo a long felt conviction of my own that to force the patient, in the initial session, into the role of information provider is to suffocate him. It is implied that therapists impose restraints upon patients in order to stay in control. Gagnon confesses "I think I was frightened of losing control of my own emotions and it was frightening for me, therefore, to see others lose control of theirs". Tirnauer develops the idea of the defensive use of power by therapists, although he also stresses that "there are times when a psychotherapist needs to be very aggressive and insistent". At the same time Virginia Fraser Stern cautions that the terrorised patient will just pull away if the therapist is too active.

I was especially impressed by Ellis's account of the six fears "that lie at the very heart of us" and intrigued by Wapnick's proposal that the purpose of therapy is "to offer our patients the healing love of God through our forgiveness of them". In similar vein Dublin, referring to a young musician who lost all who were dear to him in an earthquake avows "I will love the hell out of him". He also brought a patient flowers.

I found this a heartening and inspiring book.

JOHN BIRCHNELL, *Scientific Officer and Honorary Senior Lecturer, MRC Social Psychiatry Unit, Institute of Psychiatry, London*

Ministering to Minds Diseased. A History of Psychiatric Treatment. WILFRED LLEWELYN JONES, London: Heinemann Medical. 1983. Pp. 157. £7.50.

Psychiatry, insofar as it can be differentiated at all from medicine in general, has a relatively brief history. Nevertheless, English scholars, particularly in relatively recent years, have written treatises on various aspects of psychiatric history, the most fecund period being the late 18th and the 19th century.

Dr Wilfred Llewelyn Jones is a good example. He offers us in this slim volume a history of psychiatric treatment which, as he explains in the preface, is designed not only for psychiatrists in training, but for all who work in, or who are interested in the field, not excluding patients and their families.

Dr Jones succeeds in what he sets out to do, and he does so by virtue of his command of the subject and the clarity of his writing. An additional bonus are the several well

chosen illustrations, all of them beautifully reproduced.

If there is fault to be found, it is that Dr Jones attempts to include too much material in too little space. Even so, for those really interested, the book may serve to wet the appetite and encourage further reading.

HENRY R. ROLLIN, *Emeritus Consultant Psychiatrist, Horton Hospital, Surrey*

Disadvantaged Post-Adolescents: Approaches to Education and Rehabilitation. By REUVEN KOHEN-RAZ. New York: Gordon & Breach. 1983. Pp. 224. \$37.50.

This book is the first of a series on 'Special Aspects of Education'. The term 'post-adolescent' refers to the age group extending from the mid teens. Part I contains a discussion of the psychodynamics of adolescence, which is not as thorough as readers of this *Journal* might wish, but nevertheless leads quite naturally to Part II, in which models of normal and abnormal mental development at adolescence are considered alongside problems of the school curriculum. Part III consists of accounts of schemes which have been developed in Israel with the object of achieving at least some measure of educational rehabilitation of disadvantaged post-adolescents. The relevance of these programmes for the UK is not considered.

The central theme of the book is that substantial progress can be made in the education of disadvantaged youth, but that this depends on the development of special educational programmes based on careful analysis of exactly how it is that disadvantage creates the social, emotional and cognitive deficits which an orthodox curriculum merely reinforces. The arguments for this point of view are not perhaps entirely novel, but they are worth studying in the convincing form in which they are presented here. There is not much discussion of the particular educational difficulties of the psychiatrically disturbed adolescent, although these are by no means discounted by the author. The book is not essential reading for psychiatrists; those with a particular concern for this age group, however, will find it thought-provoking.

HUGH G. MORTON, *Consultant Child & Adolescent Psychiatrist, Ninewells Teaching Hospital and Medical School, Dundee*

Towards a Whole Society: Collected Papers on Aspects of Mental Health. Edited by RUTH TERRINGTON. London: Richmond Fellowship Press. 1985. Pp. 176. £5.00.

Thomas Szasz, Maxwell Jones, Ronald Laing, Anthony Clare, Metropolitan Anthony of Sourozh, Kay Carmichael and Robert Gosling, chaired by Elly Jansen: what a start for the Richmond Fellowship's Jubilee celebration book!

The subsequent discussions, which Clare is rumoured to have won, seem minor oscillations about Szasz' position. The section will interest anyone wishing to grasp how discussion on what psychiatrists should do is secondary to the conclusion that they should be. This is illustrated by Dr Malekpour's question to Szasz: what should he do with the

suicidal melancholic? The tentative answer was further questions: who really made who come to whom and why, who made whom the expert arbiter, off which hooks does who let whom off by being a psychiatrist defending concepts of illness, which issues in society are avoided, etc. etc. Szasz, though, pessimistically conceded that people won't give up the concept of mental illness, so psychiatrists will treat them. Without a clear definition Clare seemed to assert that doctors (psychiatrists) should listen to Szasz but must limit themselves as doctors to treating (mental) diseases. All agreed many problems are nevertheless political and religious and over-medicalised.

Sections two and three on Community Care Elly Jansen introduces by confessing that psychiatrists are useful when radical because they alone have special power in the Realpolitik to change their own dominating role. Mosher's favourable summary of the Italian experiment follows, then there is a very human piece by Jean Vanier on L'Arche, and a shrill appeal from the backstreets of Scotland by Jimmy Boyle facing up, to humbug spoken about and done for heroin addicts.

This little book continues by packing in many more easily read, overtly sociopolitical and Antipodean comments on community care. It all represents a well-deserved and well-organised tribute to Elly Jansen and the Richmond Fellowship for a quarter of a century's contribution to the theory and practice of something ill-demarcated but closely related conceptually to psychiatry. For this area the Fellowship clearly remains an important forum for debates, as well as a refuge for our clients.

F. A. JENNER, *Professor of Psychiatry, University of Sheffield*

The Misuse of Alcohol, Crucial Issues in Dependence, Treatment and Prevention. Edited by NICK HEATHER, IAN ROBERTSON AND PHIL DAVIES on Behalf of the New Directions in the Study of Alcohol Group. London: Croom Helm. 1985. Pp. 284. £19.95.

Anyone who travels the circuit of alcohol conferences will know the authors of this book and be familiar with their views. They may be Members of the New Directions Group and hopefully they will have bought a copy of this book. If they have not they should get a copy immediately.

For those of us who don't travel the circuit or who are new to it, like me, then this book is a must. The New Directions Group consists of individuals involved in treatment, education or research in the area of alcohol use and misuse. The Group has no particular axe to grind but is at the forefront of exploring new ideas about alcohol. These new ideas are reflected in the three themes in the book: changes in how we understand harmful drinking behaviour ('dependence'); changes in our confidence that drinking behaviour can be effectively modified in the traditional context of treatment; and changes in our understanding of the effective prevention of harmful drinking.

The first section, on dependence, does not provide any answers. However, it gives us plenty of information and diverse views so that we can make up our own minds about the usefulness of the 'alcohol dependence syndrome'.

Is it theoretically sound, or is it the 'disease theory of alcoholism' in a new guise?

The second section, on treatment, is a bit repetitive, with authors from different backgrounds saying much the same thing. The message, however, is clear. Before we can answer the question as to whether or not treatment works we need to understand the client's needs and we need an analysis of the therapist-client relationship.

The third section on prevention is the most important and the clearest part of the book. The public health approach to the prevention of alcohol problems is succinctly argued by Bill Saunders. The mess of recent Health Education Council Campaigns concerning alcohol and the lessons to be learnt are honestly described by Anthony Thorley.

Alan Maynard completes his chapter on economic measures in preventing drinking problems by quoting from Mao Tse-Tung: 'Complacency is the enemy of study. We cannot really learn anything until we rid ourselves of complacency. Our attitude towards ourselves should be "to be insatiable in learning" and towards others "to be tireless in teaching"'. This book goes a long way in following Mao Tse-Tung's advice.

PETER ANDERSON, *General Practitioner and Senior Registrar in Community Medicine, Oxfordshire Health Authority*

Child and Adolescent Psychopharmacology. By MAGDA CAMPBELL, WAYNE H. GREEN and STEPHEN I. DEUTSCH. London: Sage Publications. 1985. Pp. 167. £19.75 (hb), £10.00 (pb).

The prescription of medication in the treatment of psychiatric disorders in children and adolescents is a considered decision and seldom made in isolation from other therapeutic interventions. It is a pity, therefore, that this book, the second volume of six in a series on Developmental Clinical Psychology and Psychiatry, concentrates solely on psychopharmacology rather than the broader issue of the place of medication in comprehensive treatment programmes. It appears that this was not in the author's brief and they accept this limitation in their work.

The main body of the book consists of five chapters, each devoted to a particular drug or class of drugs. The authors build up the case for the use of medication, in particular diagnoses, by reviewing a large number of studies. I was pleased to see that the case in favour is balanced, in each chapter, by discussion of the contraindications and untoward effects. The first chapter of the book provides a useful introduction to the field with a description of aspects of research methodology giving emphasis to those issues particularly relevant to the field of child and adolescent psychiatry.

The authors achieve the goals they were set. The fact that the subject matter concerns only one therapeutic option reduces the clinical usefulness of the volume. However, the interested reader could make use of the book as a reference source on child and adolescent psychopharmacology.

PAUL A. CAWTHRON, *Registrar, Drug Dependence Unit, Maudsley Hospital, London*

Developmental Theory and Clinical Process. By FRED PINE.
London: Yale University Press. 1985. Pp. 269. £25.00.

In 1975 Margaret Mahler published her major work *The Psychological Birth of the Infant*, it delineated the progress from the stage of a symbiosis with the mother at the age of nine months to that of psychological individuation by the middle of the third year. Now one of her co-authors, Fred Pine, has continued the story in his own book which combines psychological observation, theory and analytic practice. His description of himself as a quiet radical and a flexible conservative is apt.

Four foci may be seen in the development of analytic thought since Freud began his pioneering work. Initially Freud's dynamic theory was based upon the satisfaction of drive instincts which were later subsumed in his theory of ego functioning. The next generations of analysts explored in turn the nature of object-relations and the development of the self. Pine does not accord pride of place to any one of these foci but argues that for each, the individual develops his own, necessary psychology. In an interesting corrective to the over-emphasis on trauma in much analytic literature, he stresses and gives examples of the importance of quiet pleasure and moments of intense experience in child development. In the second half of the book, Pine relates developmental process to clinical intervention. Winnicottian holding and Kohutian mirroring both contribute to the development of a stable self-experience, low keyed pleasure serves as a foundation for a joyous mother-child relationship. In one therapy conducted by the author, being unconditionally available at a set hour for a patient whose approach always turned into flight was of equal importance to the exploration of her conflicts. In this, Pine follows Redl and suggests that the developmental perspective allows the therapist to make specific interventions when faced with deficient functioning.

While the text is wordy and technical, the content is of considerable interest. The volume provides a broad, contemporary view of psychological development and therapy for psychoanalytic practitioners.

MARK AVELINE, *Consultant Psychotherapist, St Ann's Hospital, Nottingham*

Treating the Oedipal Patient in Brief Psychotherapy.
Edited by ALTHEA J. HORNER. New York: Jason Aronson. 1985. Pp. 246. \$25.00.

This book presents the work of an American brief psychotherapy clinical and research project. It is in the form of a series of chapters by different members of the project setting out to cover aspects of theory, patient selection, therapeutic technique, follow up and evaluation. I found the account mostly superficial and lacking in rigour of approach.

The "oedipal patients" treated are described as non-seriously ill, intelligent, psychologically minded, well motivated and with strong egos. In addition they present with a circumscribed area of complaint which can be interpreted in terms of the oedipus complex defined as the wishes, fears and frustrations associated with the opposite

and same sex parents. Any "non-oedipal" issues which might be present must be such that they can be ignored. Their method of patient selection in fact seemed very non-specific and rested on excluding disturbed patients and ascertaining whether patients were prepared to work on an "oedipal focus". There was no examination of the theoretical or clinical validity of creating such an "oedipal focus".

The psychotherapeutic technique used follows the approaches of Sifneos and of Davanloo. The therapist is active and confrontative in "relentless pursuit of even minor resistances in the patient" and in maintaining the so-called oedipal focus and the emotional tension of the therapy. The description of technique is illustrated by vignettes from sessions, but there was very limited explanation of how the examples given did justify the points made. Perhaps they were unconvincing because of the very activity of the therapist. Again theoretical questions did not seem adequately discussed. For example this "activity" of the therapist may preclude certain kinds of activity on the part of the patient. An apparently inactive therapist may be very busy listening to and thinking about what the patient brings and what is happening between him and the patient which holds within it a respect for the patient and a profound challenge to him to take himself seriously. The here and now of the experience and the emphasis on the patient creating his own answers would be considered by some the matrix for change in psychotherapy. From other perspectives there has been interesting work on locus of control in relation to therapeutic change and "pupil" participation in relation to lasting learning. However these and many other issues are not touched upon, and no theoretical justification of the approach is offered. In addition there is no data or significant description of follow-up and evaluation. Instead the reader is offered a rather irritating polemic in which the therapy presented is described with words like courage, enthusiasm and excitement in contrast to a version of classical technique which seemed based on misconception more than anything else.

The therapy presented lacks many features which could be considered essential or particular to a psychoanalytical approach. It is described as having a cognitive flavour but seems without the systematisation of approach offered in cognitive therapy itself. It is impossible to evaluate from the information given in the book and there is little theoretical justification offered for its basic postulates.

KATE DUFTON, *Senior Registrar, Claybury Hospital, Essex*

Counselling in Rehabilitation. By WILLIAM STEWART
Beckenham, Kent: Croom Helm 1985. Pp. 315. £10.95.

This book is aimed at wide readership of therapists and others engaged in the rehabilitation of disabled people. It describes the 'Wessex Model' of individual counselling which promotes self-awareness and insight, combined with a problem-solving approach, and it also highlights the differences, as well as the similarities, in the application of this model to rehabilitation practice. Some of the moral and ethical problems inherent in the process of rehabilitation are briefly considered, and some of the psychological aspects of pain, stress in surgery and sensory loss

are discussed. The book also pays welcome attention to the impact of disability on the family and to the relatives' role within the rehabilitation programme. Brief case reports are used to illustrate some of the issues.

The structure of the book is somewhat confusing: stigma, which concerns all disabilities, is discussed in the chapter on sensory loss; a fairly lengthy account of the clinical picture of rheumatoid arthritis is given in the chapter on pain. Moreover, the whole is marred by occasional repetitiveness and a certain lack of selectivity: lists of aetiological factors of depression and of symptoms of multiple sclerosis are hardly relevant. On the other hand, a mention of other models of counselling would have been appropriate. The book also virtually ignores the field of psychiatric disabilities—a serious omission. Although it would be useful for personnel involved in the rehabilitation of the physically disabled, it is not recommended for psychiatric libraries.

MOUNIR EKDAWI, *Consultant Psychiatrist, Netherne Rehabilitation Service, Surrey*

Recent Advances in Alcohol and Drug Problems Vol. 8

Edited by REGINALD G. SMART, HOWARD D. CAPPELL, FREDERICK B. GLASER, YEDY ISRAEL, HAROLD KALANT, ROBERT E. POPHAM, WOLFGANG SCHMIDT and EDWARD M. SELLERS. New York: Plenum. 1984. Pp. 333. \$49.50.

The eighth volume in this series presents a collection of varied papers of interest to research workers and clinicians in the field of drug and alcohol dependence. Some earlier volumes concentrated on particular aspects, but as the editors make clear volume 8 is a more varied selection of recently published research. All of the articles are interesting and provoke speculation which is relevant to clinical practice, albeit of more specific concern in some instances to the research worker. Thus the first paper by Lars Terenius on the Role of Receptors and Endorphins in Opiate Tolerance and Dependence confirms for the clinician the extreme complexity of the endorphin system; emphasizes the existence of large numbers of biologically active neuropeptides; alludes to inter-species differences; in passing mentions the lack of cross-tolerance between drugs with slightly different receptor profiles; and generally provokes considerable speculation regarding the specific role of drug effects within that complex phenomena known as drug addiction, which the medically qualified member of the multidisciplinary team is responsible for representing to colleagues. This article alone is important. Articles on alcohol self-administration by animals and the genetics of alcoholism in relation to human and animal studies again emphasizes the complexities of the possible interactions involved between environment and genetics. Supported by over 200 references this article states "it is crucial that the scientific community educate the lay public that inheritance does not account for all risk factors, and that the providers of alcohol treatment not over-interpret the current evidence to the point of claiming that all forms of alcoholism are inherited, leaving no room for the importance of environmental conditions. Many individuals with no known genetic predisposition become alcoholic, whereas

even more individuals with strong genetic predisposition do not become alcoholic." In Britain this sound observation must be borne in mind during political and economic discussions where there are considerable forces with a vested interest in the concept that only a minority of individuals are at risk of developing alcohol problems, thereby avoiding the need for general public health measures and education. These are only the first two of 11 excellent contributions reviewing physical consequences of alcohol consumption; analysis of the methodology of studying alcohol-related road accidents; the importance of prospective studies in understanding aetiology; the problems of measuring alcohol consumption in research studies; and a paper by Barry Brown of the National Institute on Drug Abuse regarding treatment issues in non-opiate dependency which is highly relevant to clinicians responsible for establishing drug dependence services in the light of the Government's offer of certain funding for this purpose and the need to achieve a proper balance between opiate and non-opiate dependence.

As with previous volumes in this series, this book is an essential addition to the professional library of any Health District which purports to be providing any service in alcohol or drug problems.

MARTIN MITCHESON, *Consultant Psychiatrist, County Hospital, Hereford*

Video Violence and Children. Edited by GEOFFREY BARLOW and ALISON HILL. Sevenoaks, Kent: Hodder & Stoughton. 1985. Pp. 182. £8.95, £4.95 (pb).

This book represents the final report of an informal working party looking into the influence of watching violent video films on children's lives. The book is multi-author and consists of a series of surveys and clinical reports compiled by members of the working party from different professional disciplines.

Two general population surveys are reported which indicate the extensive number of households which now contain a video. In the National Viewers Survey of 4500 school children, some 50% of households contained a video and in the NSPCC survey of 722 children, some 30% had a video in their homes. Both of these surveys also indicated that somewhere between a half and two thirds of the children in both of these samples had seen one or more videos considered to be violent. Some of these videos have undergone prosecution under the Obscene Publications Act of 1959. These chapters are followed by three Professional Surveys indicating the influence psychiatrists and paediatricians consider violent videos have had on the problems children present in clinical work. The overall opinion is that video nasties may exert an adverse influence on child development and on the development of symptoms. Anecdotal case studies from child psychiatrists further indicate that at the very least the potential detrimental influence of videos on child mental health cannot be ignored.

This demonstrates the large numbers of videos in people's homes exposing children to a wide range of new visual phenomena, including the video nasty. The data

presented make important and illuminating reading. The design and methodology of the surveys makes it difficult to draw firm substantive conclusions concerning the mechanisms by which video nasties may be influencing children; however, the potential adverse influence described emphasises the need for further research in this area.

Overall the book is easy to read and stays within its brief of reporting the findings of its working group. The conclusions are reasonable, as is its price. It is important for professionals involved in child health care to be aware of the potential issues raised in this publication.

I. M. GOODYER, *Senior Lecturer in Child & Adolescent Psychiatry, Royal Manchester Children's Hospital*

Definition of Suicide. By EDWIN SHNEIDMAN. New York: Wiley-Interscience. Pp. 256. \$26.50.

Despite its title, this book is more about understanding than defining. It offers a distillation of old and current views and brings together several approaches, extending from cosmology, systems theory and the American novel to psychoanalysis, logic and psychology. With its 26 chapters aptly arranged from A to Z, this personal account of a well-known suicidologist and Thanatologist transmits an unmistakable air of closure. Obviously, there is a definition too. Taken from its historic origins, the word "suicide" reaches our "current Western world" as a sort of "multidimensional malaise" to become a "self-induced annihilation" for those individuals who, having first "defined an issue" view this act as "the best solution" to their predicament. For nearly every word in his four line formal definition there is an "explication and exegesis".

Of particular interest to workers in mental health is Shneidman's list of "ten common characteristics". Selected on idiographic rather than nomothetic criteria, as the author himself frankly admits, these characteristics have an appealing ring of truth and convey a direct message for suicide prevention. The common stimulus for suicide, he says, is "unbearable pain": reduce it; its common stressor "frustrated needs": fill them; its common purpose "to solve a problem": suggest better and more viable solutions instead, its common goal to "cease consciousness": indicate other aims as clear alternatives; its common emotion depression and helplessness: inject hope, chemically or otherwise; its common internal attitude "ambivalence": exploit it to gain time; its common cognitive set "constricted" vision: open it up for other options to come in view; its common message to others "communication of intent": make sure you hear the message and open the channels to pass it on; its common action "escape": if you have to, keep the exits blocked; its common consistency is with a "life-long coping pattern": activate a positive attitude and draw upon the person's earlier and more constructive coping style.

In this context, suicide prevention calls for a joint effort from all possible angles. Shneidman's choice is a rather grand "Therapeutic Council" in which he will have "a biologically oriented psychiatrist, a psychoanalytically

oriented therapist, a sociologist, a logician-philosopher, a marriage and family counsellor and an existential social worker". In our current state of financial stringency, can we trust our much more pedestrian "multi-disciplinary team" to fit this bill?

This is a scholarly and important book about suicide. It may not break new ground but as a fresh and elegant synthesis of ideas on a much trodden upon subject, it is a pleasure to read. And although some will find parts of it somewhat esoteric (as many as 40 pages, for example, attempt to unravel the "Psycho-Logics of Moby Dick") it would make a valuable addition to every psychiatric library. Some librarians may ponder whether to place it in the Arts or the Science section but, given our present knowledge on suicide, it should fit equally well in both.

D. J. PALLIS, *Consultant Psychiatrist, Graylingwell Hospital, Chichester.*

Assessment and Taxonomy of Child and Adolescent Psychopathology. By THOMAS M. ACHENBACH. London: Sage Publications. 1985. Pp. 200. £19.75 (hb), £10.00 (pb).

The Sage series on *Developmental Clinical Psychology and Psychiatry* is a welcome source of information on children and adolescents.

In this third volume, Achenbach authoritatively discusses the central themes of assessment—"identification of the distinguishing features of each case"—and taxonomy—"the grouping of cases according to their distinguishing features"—in the psychopathology of the young.

Some books dealing with psychopathology have an ideological undercurrent reflecting the author's commitment to one set of beliefs. This unbiased monograph is a "meta-analysis" comparing the currently fashionable clinical and research assessment and taxonomic paradigms dealing with psychopathology.

Achenbach discusses the problems encountered in clinical information processing, and clarifies the ubiquitous nature of judgemental biases relating them to current assessment practices. Dealing in turn with the medical, psychodynamic, psychometric and behavioural paradigms and then focusing on the Kraepelinian, psychodynamic and multivariate taxonomic paradigms, he leaves the reader uncomfortably convinced that information processing biases exist to such a degree in each framework that the notion of "unbiased objectivity" is an illusion.

He systematically classifies the sources of distortions inherent in each system, highlighting the complexities confronting the clinician and researcher in dealing with the psychopathology of the child and adolescent.

Achenbach's exposé should not be dismissed (as he states) as a philosopher's hobby horse. Rather his critical analysis and synthesis should stimulate creative discourse, heighten the necessary tensions between firmly held, polarised views on the nature of psychopathology and eventually, one hopes, lead to a unified theory of development that includes a sound rationale for assessment, taxonomy and intervention in dealing with troubled youngsters.

GEORGE HALASZ, *Consultant Child/Adolescent Psychiatrist, Austin Hospital, Heidelberg, Australia*

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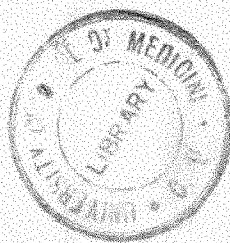


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The standard of statistical work in papers submitted for publication has proved to be very variable. Authors using any statistical methods at all are strongly advised to study the paper 'Statistical guidelines for contributors to medical journals' (D. G. Altman *et al*, *British Medical Journal*, 7 May 1983, **286**, 1489-1493) at the earliest possible stage in preparing their work for publication.

7. **Manuscripts** Two high-quality copies should be submitted and the author should retain a copy. Articles must be typed on one side of the paper only, **double spaced with wide margins**, and all the pages must be numbered sequentially, starting with the title page.

a. The **title** should be brief and to the point. If necessary, a sub-title may be used to amplify the main title. The names of the authors should appear on the title-page, and **their names, degrees, affiliations and full addresses at the end of the paper**.

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d. **Symbols and abbreviations** Follow *Units, Symbols and Abbreviations: A Guide for Biological and Medical Editors and Authors* (3rd ed. 1977), £2.00, available from the Publications Office of the Royal Society of Medicine. Terms or abbreviations which might not be understood by the average reader should be explained at first occurrence.

e. **Tables and Figures** Each table should be on a separate sheet, with an adequate heading; table and heading should be self-explanatory without reference to the text. The desired position of the table relative to the text should be indicated in the left margin.

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THE BRITISH JOURNAL OF PSYCHIATRY

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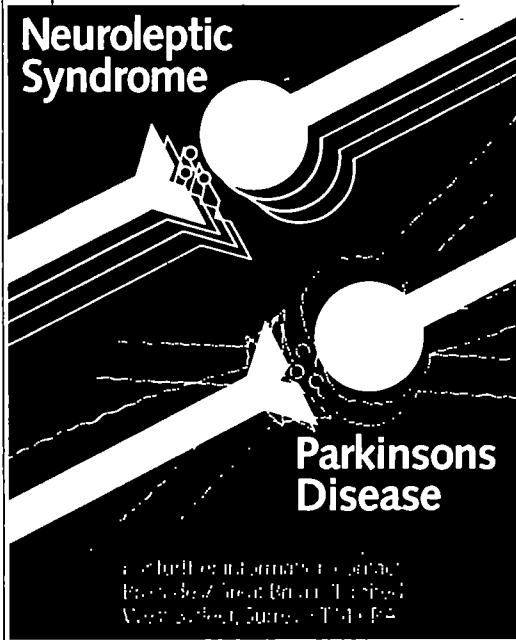
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For further information contact: Tricia Carroll, Department of Psychiatry, Charing Cross & Westminster Medical School, 24 St Dunstons Road, London W6, Tel. 01 748 2040 ext. 3010.

The course is approved for study leave expenses under HM(67)27.

Final date for registration is Friday 17th October.

Vital New and Revolutionary Principles of
Human Psychology, Physiology And Psychogenic Pathology

by

M. S. Rao, 2nd revised edition, 832 pages, demy 8vo, hard bound, art jacket, £12.

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This book shows that while the psychology and physiology of the other animals are simply instinctive and self-preservative, the peculiar human psychology and physiology (because of the unique human brain) are complex; and, besides being instinctive, they are also idea-determined, intellectual and super-instinctive, so that they are naturally very alterable and *pervertible* or disorder-prone, so that they often become counter-instinctive, suicidal and self-destructive, as shown in cases of schizophrenia, hypochondria, hysteria, melancholia, paranoia, etc., called 'neuroses and functional psychoses', or 'the major psychiatric disorders', in today's psychiatry and abnormal psychology.

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This book also shows how the very special (alterable and *pervertible*) human psychology is also corrigible, and improvable or *supervertible*, so that we are now able to improve one's mental health (rectitude), with hopes and ambitions, with new will-power, courage and self-confidence.

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'A review of your outstanding book was most rewarding.

I express my sincere appreciation of your excellent book. The (new) basic premises on which your book is based are quite sound. I compliment you on this concise representation, and on the scholarly and outstanding text. It is a most interesting, informative and scholarly work. . . a most distinguished book. . . It will be warmly received and widely read in India as well as other countries' Similarly—

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Even the Orthodox Psychiatrists (generally committed to Kraepelinian and genetic theories) appreciate and recommend these 'new ideas' or new theories of this book. Thus, Prof N. S. Vahia, a senior psychiatrist of India, says in a long review of this book in—

The Indian Journal of Medical Sciences, "The author's view-points deserve serious consideration by all those physicians, psychiatrists and clinical psychologists interested in new ideas."

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So, obviously, this is a book for all psychologists, psychiatrists, general medical men, and other intellectuals, educationists etc., and for all libraries. It is already in the more-important libraries of London. Published by Universal Psychology Publishers, D-36, Subhas Marg, Jaipur-1, India. London distributors:

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Lord Chancellor's Medical Visitors

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The Visitor to be appointed will be expected to carry out about 6 visits a year, in Wales, Hereford and Worcester and Shropshire. Reserve Visitors for other areas may also be needed.

Applications should be sent to Mr I. R. Tapster, Lord Chancellor's Department, Neville House, Page Street, London SW1P 4LS by not later than the 30th September 1986.

Application forms will be provided on request to that address, or by telephone to 01 211 8084.

UNIVERSITY OF LONDON NORTH THAMES PSYCHIATRY MEMBERSHIP COURSE

The Autumn term of the course will be held in the Post-graduate Medical Centre, at Channing Cross Hospital. The Royal College of Psychiatrists have recently agreed to major alterations to both parts of the MRC Psych Exam and in accordance with this, the old course will be abandoned and replaced with a fresh approach to teaching of psychiatry and the basic sciences for the new exam. When fully operational six modules will be offered. During this transitional term, Module 1 is offered which is appropriate to Part I. For Part II the course continues as usual this term for the last term before modifying its syllabus in accordance with the new exam.

Module 1. To be held Tuesday afternoons from 3.00–6.15 p.m. from 7th October to 9th December to include Basic Clinical Skills; Descriptive Psychopathology; Neurosis; Affective Disorder; Schizophrenia; Acute and Chronic Organic Brain Syndromes; Dynamic Psychopathology—personality and personality disorder; Basic Psychopharmacology.

Part II, Term II. To be held Thursday afternoons from 3.00–6.15 p.m. from 9th October to 11th December to include Neurotic illness; Personality disorders; Affective Illness and Behaviour Therapy

Approval has been given for study leave and expenses under HM(67)27. Enquiries to Tricia Carroll, Course Administrator, Department of Psychiatry, 24 St Dunstons Road, London W6. Tel (01) 748 2040 x 3010.

West Midlands Institute of Psychotherapy

INTRODUCTORY COURSE IN DYNAMIC PSYCHOTHERAPY

An introductory course in DYNAMIC PSYCHOTHERAPY will be held at Uffculme Clinic, Birmingham, beginning on 20th January 1987. The course will be held on Tuesday afternoons and will extend over a period of two years.

Throughout the two year course members will attend a small group. The first year will also include seminars on both relevant theory and practice, together with case discussions.

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The programme is open to members of the medical and nursing professions, social workers, probation officers and other related disciplines.

The closing date for applications is *30th September 1986*. Shortlisted applicants will be required to attend for interview on Wednesday, 22nd October 1986, at 2 p.m.

Fees for the 1987/8 course will be £260 per annum.

For further information and application forms, please contact: Mrs. Valerie Peake, Secretary W.M.I.P., Uffculme Clinic, Queensbridge Road, Birmingham B13 8QD.

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If you are interested in obtaining detailed information *Contact:* Dr Jim Carson, Chief Policy Advisor Office of Psychiatric Services, interviewing in London from 15 September. Telephone: 836 2656 (Victoria House, Strand WC2)

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For further details of speakers and their specific topics and for registration forms, please contact: **Diane Carter, Charter Clinic, 1/5 Radnor Walk, London, SW3 4PB. Tel no 01-351 1272.**

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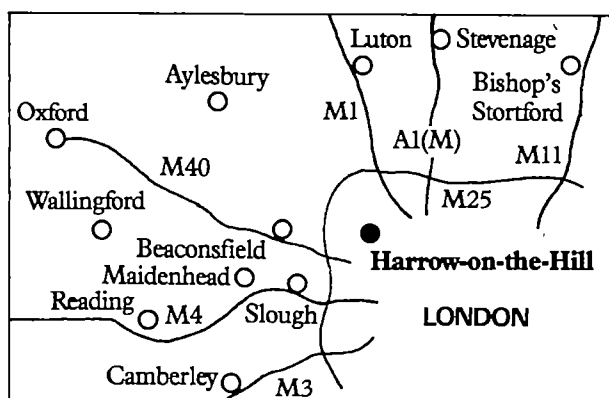


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Mental Illness Among Immigrant Minorities in the United Kingdom

M. LONDON

Cross-cultural studies on immigrants from Pakistan and the New Commonwealth are reviewed, with emphasis on epidemiology and differences in clinical presentation. Their referral to the psychiatric service is also examined and deficiencies are noted. Awareness of transcultural issues among health professionals need to be increased in order to achieve diagnosis and improvements in health care.

Just under 3% of the population in Britain today were born in the countries of the New Commonwealth and Pakistan (Registrar-General, 1983). The majority came from the West Indies and the Indo-Pakistan subcontinent during the 1950s and 1960s to satisfy the need for unskilled labour. Others, mostly from Africa, arrived for the purpose of further education. With the passage of time a British-born generation has emerged, but they have received relatively less attention from researchers than their parents who migrated to this country. This is largely due to the practice of using the place of birth to identify subjects for research.

Earlier migrations from Ireland and Eastern Europe have been associated with increased rates of psychiatric disorder (Clare, 1974; Murphy, 1955; Mezey, 1960). Murphy (1977) has put forward three explanations for this relationship. Firstly, the observed differences may have little to do with the migration itself, and may only reflect a different pattern of disorder in the migrant's culture of origin. The second explanation, known as the selection-theory, applies mainly to schizophrenia and suggests that during the early incipient phase of the illness the likelihood of migration is increased because of failing interpersonal relationships (Ødegard, 1932). The third explanation lies in the stress associated with migration and resettlement. Many endure hardships, such as split families, prejudice, poor housing and employment (Cox, 1977; Hashmi, 1968; Littlewood & Lipsedge, 1982), and single men may experience sexual difficulties (Risso & Boker, 1968). Social isolation and insecurity provide fertile ground for paranoia (Eitinger, 1960). To cope successfully one has to undergo a series of internal changes very similar to the process of bereavement, in which the old culture is abandoned and the threat to one's identity resolved (Garza-Guerrero, 1974).

Recent reviews of the transcultural research undertaken in this country have noted the problems

in gauging the true state of psychiatric morbidity among immigrants (Littlewood & Lipsedge, 1979; Rack, 1982a; Cochrane, 1983). Earlier studies (Gordon, 1965; Hems, 1967) failed to recognise demographic differences when comparing results with the native British population. Immigrants have a younger age structure and show an excess of males in the initial stages of immigration, before being joined by their wives and children. British-born control groups are often drawn from the same inner city areas where most immigrants reside. These areas are known to have unusually high levels of morbidity (Faris & Dunham, 1939; Hare, 1956). It is also uncertain whether variables such as social class and occupation carry the same meaning for each group, as menial employment in this country may nevertheless represent an improvement over a peasant economy (Stopes Roe & Cochrane, 1980). Accurate assessment is often hampered by lack of epidemiological data for the country of origin. Much research also assumes a homogeneity among immigrant groups, which is only partly justified. Although many West Indians come from Jamaica, others originate from islands that differ markedly in their culture, size and economic activity.

Immigrants from the Indo-Pakistan subcontinent have been grouped into those from India and Pakistan, neglecting religious, cultural and language differences. Some studies have inappropriately placed Asians from East Africa together with those from the subcontinent, thereby failing to take into account the many differences – in particular, their arrival in this country as refugees. Finally, there are difficulties of small sample size, especially in the evaluation of individual diagnoses.

West Indians

Studies based on hospital admissions (Cochrane, 1977; Dean *et al*, 1981; Carpenter & Brockington,

1980) and on the Camberwell Case Register (Bagley, 1971a; Rwegellera, 1977) show an increased overall rate of disorder among West Indians. The sex difference is as expected, with an excess of females (Cochrane, 1977; Dean *et al*, 1981, Carpenter & Brockington, 1980). A recent study (Ineichen *et al*, 1984) asserted that there was no significant difference in the rate of voluntary hospital admissions between West Indian and English patients living in the centre of Bristol. One in six of the population in these inner city districts belonged to ethnic minorities, and this was reflected in the proportion of voluntary admissions during 1981: 30 out of 175 admissions. However, although 16 admissions were of West Indians the authors made no direct comparison with census statistics for West Indians alone. Furthermore, as already noted, comparisons with inner city areas may in itself indicate an excessively high rate. By contrast, West Indians were noticeably over-represented among compulsory admissions (Harrison *et al*, 1984), numbering 27 out of the 89 formal admissions between 1978 and 1981.

Since they sample hospital in-patients, these studies are biased in favour of the major psychiatric disorders and may therefore relate more narrowly to those illnesses that require admission rather than to the total psychiatric morbidity. A more representative sample may be provided by the Case Register, which documents all contacts with the psychiatric service in a defined area. However, not all psychopathology reaches the attention of the psychiatrist. Two small general practice studies (Pinsent, 1963; Kiev, 1965), undertaken on what were at the time recently arrived immigrants, showed an increased morbidity; no similar studies appear to have been carried out since then.

With a few exceptions (Rwegellera, 1977; Hems, 1967) schizophrenia appears to be the commonest diagnosis (Cochrane, 1977; Dean *et al*, 1981; Gordon, 1965; Bagley, 1971a; Bebbington *et al*, 1981). This is in keeping with international research findings (Sanua, 1969) and lends support to the selection theory. However, those British studies that have defined their categories more strictly have shown an increase of atypical psychosis. Tewfik & Okasha (1965) found that only 155 of psychotic West Indian in-patients suffered from an illness typical of those seen in Britain: half suffered from mania and half from chronic hebephrenic schizophrenia. The remainder appeared to have a schizophrenia-like illness characterised by a continuum of symptoms with confusion and paranoid symptomatology at either end. The overall prognosis was good, with 43% making a complete recovery and 77% returning to work. The authors felt that this illness was a dissociative

one and a response to stress. Similarly Gordon (1965) found schizoaffective disorder among a quarter of his admissions, and also diagnosed acute schizophreniform reaction with a greater than average frequency. Less than 5% of his series had a positive family history for psychosis, and only 7% had been hospitalised prior to migration.

These early studies may be criticised on methodological grounds, and comparisons with British-born patients are unreliable. Nevertheless, these clinicians did find that a large proportion of the West Indian patients that they treated presented atypically. Despite the passage of years these atypical psychoses have been reflected in more recent epidemiological research. On reanalysis of hospital diagnoses Carpenter & Brockington (1980) showed that nuclear schizophrenia fell from a previously high rate to within the expected British range, while paranoid psychosis was increased. A criticism of this study, however, is that the ethnic minorities were insufficiently differentiated. Africans and West Indians were combined for the purpose of hospital diagnosis, and the reanalysis was carried out on immigrants as a whole. Littlewood & Lipsedge (1981b) studied a small group of sixteen patients with religious delusions but lacking first rank symptoms. They were typically women belonging to charismatic churches whose psychoses followed a precipitating event and whose illness was of short duration. With the passage of time, and presumably the influence of a dominant culture, the presenting picture changed to one of depression. Although the acute psychotic reaction is widely recognised in developing countries (German, 1972; Littlewood & Lipsedge, 1981b), it is not a diagnostic category generally used by British psychiatrists. The blanket use of schizophrenia as a diagnosis may mask such distinctions.

Symptoms prominent in schizophrenia and allied disorders are religious beliefs (Kiev, 1963; Gordon, 1965; Ndeti & Vadher, 1985b) and paranoid delusions (Gordon, 1965; Rwegellera, 1977; Ndeti & Vadher, 1984c). The former arise out of the fundamentalist Christian beliefs of the West Indian community (Kiev, 1963), while the latter has a recognised association with migration. Schneider's first rank symptoms are present in 43–53% of patients with schizophrenia (Chandrasena, 1980; Ndeti & Vadher, 1984d) as opposed to 72% of British-born patients (Mellor, 1970). Disturbed behaviour also appears to be more common prior to admission (Harrison *et al*, 1984; Rwegellera, 1970).

The sex ratio for patients with affective disorders may be more exaggerated than that for British patients (Cochrane, 1977; Bebbington *et al*, 1981; Hems, 1967) but overall frequencies are similar

(Dean *et al.*, 1981; Carpenter & Brockington, 1980; Bagley, 1971a). A community survey noted that the sex ratio narrowed with age, although the severity of the depression was not clearly defined (Burke, 1984). Mania is significantly more common among West Indians (Leff *et al.*, 1976; Bebbington *et al.*, 1981). Rack (1982b) has attributed this to reactive mania, a type of stress reaction (Tyrer, 1982). Alternatively, cyclothymic individuals may be more inclined to migrate.

The results for neuroses and personality disorders are inconsistent, with some studies showing reduced rates (Cochrane, 1977; Bebbington *et al.*, 1981; Bagley, 1971a; Gordon, 1965), increased rates (Rwegellera, 1977) or no apparent difference (Dean *et al.*, 1981; Carpenter & Brockington, 1980) when compared with British control groups. This ambiguity is significant because these 'lesser' disorders are much commoner than the major psychoses among British-born patients. This discrepancy may reflect a difference in the utilization of the psychiatric services.

Burke (1976a, 1979) found the attempted suicide rate among those who had migrated to be greater than the indigenous rate for Jamaica, but it had not reached the same level as the native British population. He attributed this to the problems of acculturation and adaptation to an industrialised urban environment. However, this study was a retrospective one, and the inclusion of only those cases where the place of birth had been recorded may have biased the sample. The suicide rate is less than that of the native British rate, and is also one of the lowest among all the ethnic minorities (Cochrane, 1977; Adelstein & Mardon, 1975). As shown by research in Australia, the indigenous pattern of suicide among immigrants may be expected to persist unchanged for many years (Burvill *et al.*, 1982).

West & Farrington (1973) found an increase in delinquency among West Indian boys despite a low rate of criminality in their parents (Rutter *et al.*, 1975). Although Burke (1982) ascribed this to repeated separation experiences, his study of eighteen female offenders was too small to substantiate his assertion, particularly with regard to male offenders. Studies of West Indian children showed an abnormally high rate of conduct disorders (Rutter *et al.*, 1974; Nicol, 1971) of a kind that was unusual in many respects. It occurred predominantly in girls, was specific to the school situation and was not associated with impaired peer relationships (Rutter *et al.*, 1974). It also correlated with poor performance on tests of intellect and reading abilities (Yule *et al.*, 1975). Suggested explanations included overcrowded home conditions, deprivations in early life,

particularly for those born in the Caribbean islands, and the poor quality of the schools themselves (Rutter *et al.*, 1974; Yule *et al.*, 1975). Cochrane (1979) replicated Rutter's study six years later, and although he found some support for the previous trend it failed to reach statistical significance. He concluded that the educational system may have successfully assimilated immigrant children during those intervening years.

In order to evaluate epidemiological findings accurately one must consider the interaction and referral patterns of West Indians with the Health Service. Burke (1984) concluded that general practitioners failed to reach a psychiatric diagnosis for 21.3% of West Indians identified as being depressed in a survey. This was significantly greater than the 12.8% identified in an English control group. These subjects, drawn from a general practice register, were initially screened with the General Health Questionnaire and probable cases were then interviewed. Although depression was operationally defined the criteria were not clearly described; nor was it possible for the assessment to be blind.

Subsequent referral of West Indians to the psychiatric service differs from the indigenous British population both in the high rate of compulsory admissions and in the degree of involvement by an agency other than a general practitioner. Compulsory admissions have correlated with the excessively high rate of psychosis among West Indian admissions (Harrison *et al.*, 1984; Rwegellera, 1980; Littlewood & Lipsedge, 1981a; Gordon, 1965). Rwegellera (1980) analysed the Camberwell Case Register for psychiatric referrals during the 1960s, including out-patients and domiciliary contacts. Significantly, only half of the West Indian patients were referred by general practitioners or hospital doctors, in contrast to 61% for a well-matched English control group. Many were brought by relatives. Another study of hospital admissions during the 1960s found support for this trend, although West Indians were not differentiated from other New Commonwealth immigrants (Hitch & Clegg, 1980). Recently, Harrison *et al.* (1984) showed greater police involvement in the referral of West Indians from an inner city area, compared with indigenous British patients living in the same locality. Littlewood & Cross (1980) also found self-referrals to be more common among a mixed group of New Commonwealth immigrants. There are correlations between compulsory admissions, psychosis and disturbed behaviour (Harrison *et al.*, 1984; Rwegellera, 1980). Relatives may prefer to resort to legal sanctions and call the police to avoid the stigma of mental illness (Hitch & Clegg, 1980). As a minority group West Indians may also feel alienated from

society's institutions, and this may affect their attitude to general practitioners. Their reluctance may also be related to ignorance of how a GP can help, or the belief that he is not the correct person to consult regarding mental illness (Rwegellera, 1980). The authorities may be less tolerant of deviancy among immigrant groups, and some have regarded compulsory admission as a form of social control (Mercer, 1984). Most importantly, if the illness remains untreated for a long period the patient may become more seriously ill, losing insight and necessitating a formal admission. Harrison *et al* (1984) found that many West Indians formally admitted were young women living with their families. Despite this close contact, many were detained by police in public places and brought to see a psychiatrist. In view of the research pertaining to atypical psychosis one may suspect that earlier intervention and treatment may prevent the psychosis developing. Although Littlewood & Lipsedge (1982) note that the sudden onset of the acute psychotic reaction may prevent early detection, the psychological well-being of these patients prior to onset has not been proven. Depression and minor psychiatric illness may remain untreated for the reasons discussed above. Deterioration may lead to a psychosis through subcultural or dissociative mechanisms. This is an area which requires further research.

At its height, immigration from the West Indies was unrestricted and even encouraged by British employers through recruiting drives. Although such conditions favour the selection model, this may only partly explain the apparent excess of psychoses. The duration of stay in the United Kingdom prior to the onset of symptoms is a relatively lengthy one of 5–10 years (Littlewood & Lipsedge, 1981a; Bagley, 1971a). This suggests that stress plays a significant role, whether as a precipitating factor in those biologically predisposed (Bagley, 1971a) or as an aetiological agent in a reactive psychosis (Littlewood & Lipsedge, 1981b). Such stress has many facets. Bagley (1971b) advocated a model of 'status inconsistency' where the subject strives to achieve goals but fails to recognise the restriction of opportunity implicit in the environment. The discrepancy between the perceived ideal and the true self results in loss of self-esteem. Drawing on the learned helplessness model, Fernando (1984) argued that racism engendered depression by denying its victim any control over external events. Poor self-esteem, rejection and loss exacerbate the phenomenon. These complex theories are difficult to test in practice, as it would require a large, well-constructed study to eliminate the many associated factors. In a community study measuring individual stresses Burke (1980) was

unable to demonstrate overall differences, although he noted an emphasis on interpersonal conflicts among West Indians which may have been enhanced by greater isolation from neighbours and relatives.

Indians and Pakistanis

Epidemiological studies on overall morbidity have produced conflicting results. Carpenter & Brockington (1980) showed that Asians have an increased rate of first-time hospital admissions, although they failed to differentiate between those born in India and Pakistan. Dean *et al* (1981), with closer attention to the place of origin, showed that Pakistanis have fewer first-time admissions than the British, while Indians have a higher rate. Cochrane (1977) analysed national hospital admissions for 1971 and showed that both Indians and Pakistanis have fewer hospital admissions than the British (including readmissions). The comparability between the studies by Dean *et al* (1981) and Cochrane (1977) is hindered by differences in techniques used to analyse the data. Furthermore, Dean *et al* (1981) were able to identify the place of birth in a higher proportion of their sample; this information was absent in 30% of national admissions. A further source of error is the hidden number of patients who, though born in India, come from British colonial families.

Schizophrenia is the commonest hospital diagnosis in both populations, and occurs with a higher than expected frequency (Cochrane, 1977; Dean *et al*, 1981; Carpenter & Brockington, 1980; Shaikh, 1985). When readmissions are excluded, Pakistani men fall to within the British range (Dean *et al*, 1981). Rack (1982b) has drawn attention to the pitfalls of diagnosing schizophrenia when English is not the first language and where cultural beliefs differ. Ndeti & Vadhver (1984d) found that Schneider's first rank symptoms were present in only 58% of Indian immigrants where schizophrenia was diagnosed according to the CATEGO programme. Unlike research on West Indians, atypical psychoses among Asians have received relatively little attention. When noted, they do not appear to be increased, although these studies were based on routine hospital diagnoses (Dean *et al*, 1981; Carpenter & Brockington, 1980).

Studies both of first admissions (Dean *et al*, 1981; Carpenter & Brockington, 1980) and the Camberwell Case Register (Bagley, 1971a; Pinto, 1970) show no significant difference in the rate for affective disorder in comparison with the native British population, although Cochrane (1977) found it to be comparatively less when all admissions for 1971 in England and Wales were analysed. In Asiatic and other

developing countries depression is characterised by prominent somatic symptoms, whereas those psychological features more usually recognised in the West may be concealed or even absent (Teja *et al*, 1971; Murphy *et al*, 1967).

In-patient studies show wide variations in rates for neuroses and personality disorders which nevertheless remain consistently less frequent than the psychoses (Cochrane, 1977; Dean *et al*, 1981; Carpenter & Brockington, 1980; Shaikh, 1985; Pinto, 1970). Clinical features of neuroses differ from those of British-born patients. Ndeti & Vadher (1984b) analysed the case notes of a mixed group of immigrants using the syndrome check list and CATEGO programme. In studying anxiety and related syndromes they found that the tension category, which comprises mainly physical symptoms, was significantly increased in African, Asian and Caribbean patients. In addition to this theme of somatisation, Rack (1982b) has also drawn attention to the effect of dissociation in hysteria. A pseudo-psychosis characterised by hallucinations is not uncommon in young Asian women and can be regarded as a means of communicating distress akin to the overdose in English patients.

Attempted suicide has increased following migration but remains less than the British rate (Burke 1976b). The principal method used is similar to that of English patients, having changed from corrosives and insecticides to psychotropic drugs. Surveys of childhood disorders using Rutter's method have shown a reduced rate of conduct disorder in Indian and Pakistani school-children (Kallarackal & Herbert, 1976; Cochrane, 1979). Cochrane attributed this to better psychological health among Asian immigrants, while Kallarackal & Herbert felt that the quality of Indian family life helped reduce any disturbance. There was an indication of increasing maladjustment with longer residence in the United Kingdom, but this failed to reach statistical significance.

Findings based on hospital admissions may be influenced by differences in utilisation of the psychiatric services. Some patients may avoid the service entirely and seek help from traditional healers. Aslam (Rack, 1982b) found that Hakims, practitioners using an Islamic system of medicine, diagnosed mental problems in a quarter of their patients. Many of these Asian immigrants complained of psychosexual difficulties. Brewin (1980) showed that Asians visited their general practitioner as frequently as British-born controls, yet their admission rate for psychiatric disorder was reduced. He concluded that general practitioners had not detected these disorders because of a failure of communication. The

unproven assumption behind this reasoning is that the distribution of psychiatric and physical illnesses were the same in each group. Although studies of mixed groups of immigrants show high rates of formal admissions (Hitch & Clegg, 1980), Shaikh (1985) reported no significant differences among Asians living in Leicester, even though schizophrenia was more commonly diagnosed. Pinto (1970) used the Camberwell Case Register to compare 49 Asian admissions, born in the Indian subcontinent, with an English control group matched for diagnosis. There was no significant differences in compulsory admissions, nor in the number of referrals by general practitioners. There was a higher rate of self-referrals by Asian patients and Asians did show a slightly longer duration of symptoms prior to admission. Pinto inferred from this that Asians may avoid seeking medical help, whether through ignorance or difficulties in communication. However, these conclusions are based on a small number of patients.

Cochrane undertook a series of community surveys using the Langner 22 Item Index to detect minor neurotic symptoms. He compared two samples of Indians and Pakistanis, selected to represent their respective communities by age and sex, with a matched British-born control group (Cochrane & Stopes Roe, 1977). Pakistani men scored significantly fewer psychological symptoms than the British controls, while all other scores were equivalent. The symptoms derived by the Langner index did not constitute psychiatric cases, and they were also elicited by lay research workers. A larger multi-centre study compared Indian and Pakistani-born with Irish-born subjects (Stopes Roe & Cochrane, 1980). Unlike the previous study this survey was randomised by the research worker choosing houses at random to visit. However, the sample was still limited to high density immigrant areas. The Pakistanis now achieved very high scores, while the Indians scored less than the Irish. The low score for Indians was supported by a similar study comparing Indian, Pakistani and British-born subjects (Cochrane & Stopes Roe, 1981).

On the basis of both his in-patient and community studies Cochrane inferred that Indians have less psychological ill-health than Pakistanis and that the two communities together suffer less psychiatric morbidity than the general British population. It is arguable whether the two types of studies can be used in support of one another, as they measure different phenomena. He speculated that the complex and restrictive nature of migration from the Indian subcontinent deterred all but the most determined and psychologically robust, resulting in a reduced morbidity among Asian immigrants. This lowered

morbidity has not been consistently demonstrated, and the discrepancies found between first time and total admissions may imply a low readmission rate. Asian patients may either receive greater support from their relatives than their British counterparts or they may return to the Indian subcontinent to seek treatment. Cochrane (1983) further speculated that improved socioeconomic status and upward mobility contributed to the low Langner scores among Indians (Stopes Roe & Cochrane, 1980). Pakistanis have lower socioeconomic origins and have experienced little upward mobility since arrival in this country. Nevertheless, he failed to demonstrate a correlation between social class and Langner scores among Pakistanis (Cochrane & Stopes Roe, 1981). A further consideration has been the degree of acculturation and exposure to the indigenous British culture. An insular lifestyle may protect against such stress and any resultant psychological problems (Westermeyer *et al.*, 1983). However, Pakistani women showed high Langner scores despite only 10% being at work (Stopes Roe & Cochrane, 1980).

The apparent contradictions between the studies discussed above may be lessened by a closer attention to the hidden variables. Where possible, samples should be large enough to take into account factors such as regional origin and religion. Research may also be valuably directed towards the areas of initial contact between the Health Service and the Asian patient.

Africans

Bagley (1971a), using a case register, analysed all contacts with the psychiatric services in Camberwell between 1966 and 1968. Of all the immigrant minorities the Africans showed the highest prevalence rate for all disorders, it being six times that of the indigenous British population. Using a different method Dean *et al.* (1981) confirmed this greater frequency among hospital admissions, although their sample included East African Asians.

The incidence of schizophrenia is high among African immigrants (Dean *et al.*, 1981; Bagley, 1971a; Rwegellera, 1977; Littlewood & Lipsedge, 1981a), and Copeland (1968) found that two thirds of West African in-patients suffered from schizophrenia or schizoaffective disorders, although research bias may have influenced the admission procedure (Rwegellera, 1980).

At the time these studies were carried out there was comparative freedom of movement for foreign students to enter the United Kingdom to study. Young Africans with a liability to schizophrenia and poorly adjusted within their own community may

have found it relatively easy to enter the United Kingdom as students with Commonwealth citizenship. Both Rwegellera (1977) and Littlewood & Lipsedge (1981a) found that the onset of symptoms occurred much earlier in Africans than in other immigrant groups. Characteristic features of the psychotic breakdown were paranoid delusions, magico-religious ideas and hypochondriasis (Ndeti & Vadher, 1984c, 1985b; Rwegellera, 1970; Littlewood & Lipsedge, 1981a). Paranoid ideas involving witchcraft or poisoning and directed towards fellow Africans were often found to be sub-cultural and to carry less weight when diagnosing schizophrenia. More typical of schizophrenia were delusions concerning Europeans and incorporating twentieth century technology (Copeland, 1968; Lambo, 1955; Littlewood & Lipsedge, 1981a). Grandiose delusions occur commonly in all types of psychoses among Africans and Jamaicans, and correlate with religious fervour (Ndeti & Vadher, 1985a; Lambo, 1955; Copeland, 1968). Disturbed behaviour is frequent and has been attributed to either learnt cultural role models or to gross anxiety engendered by magical beliefs (Rwegellera, 1980, 1970). Excluding first rank symptoms, Ndeti & Vadher (1984e) found that hallucinations of all types occurred more frequently in African, Asian and Caribbean patients compared with English-born controls.

In recent years Ndeti & Vadher have published a series of studies on individual symptoms based on a retrospective analysis of case notes using the Syndrome Check List. The retrospective nature of their work limits its value, but there are few comparable studies in the field. They found that first rank symptoms are present in only a third of schizophrenias diagnosed according to the CATGO programme (Ndeti & Vadher, 1984e). This agrees closely with Chandrasena (1980), who used the same instrument on a different sample. This goes some way towards confirming the reliability of these studies. In Nigeria 63% of patients with schizophrenia were found to have first rank symptoms (Carpenter & Strauss, 1974). An unusual feature in the United Kingdom is the distortion of the sex ratio such that men exceed women among those admitted for schizophrenia (Dean *et al.*, 1981; Rwegellera, 1977) although women do appear to have an increased admission rate for atypical psychoses (Dean *et al.*, 1981). Although the acute psychotic reaction is recognised in Africa (German, 1972), British studies dealing with this subject have been limited by sample size (Littlewood & Lipsedge, 1981b).

Affective and neurotic disorders in Africans share many of the features discernable in other New

Commonwealth groups. Ndeti & Vadher (1984a) noted that depression was rarely diagnosed by hospital clinicians, but when they reanalysed using the Syndrome Check List they found an excess of psychotic and retarded depressions. However, other studies suggest that affective disorders are well recognised and not under-represented (Dean *et al*, 1981; Rwegellera, 1977; Bagley, 1971a). Studies of first admissions and case register contacts (Dean *et al*, 1981; Bagley, 1971a) have shown a reduced rate for neuroses and personality disorders compared with the British-born. By contrast Rwegellera (1977), examining roughly the same data as Bagley, found that these disorders were increased to the extent of being the commonest diagnoses.

In a large, well-matched sample Rwegellera (1980) found that West Africans exceeded both West Indians and English controls in the rate of compulsory admissions and disturbed behaviour. Elsewhere he demonstrated a correlation between this disturbed behaviour preceding admission and social isolation (Rwegellera, 1970). Many were living on their own or with strangers, but there is no information available on the duration of their symptoms.

Epidemiological research carried out in Africa has shown the pattern of psychiatric morbidity to be similar to that of Western countries (Giel & van Luijk, 1969; Olatawura, 1982; German, 1972). Binitie (1981) found that less than 16% of new cases attending a clinic in Benin suffered from schizophrenia while up to 50% were treated for a psychoneurotic disorder. This is consistent with Rwegellera's findings, yet the bulk of British research has shown a marked excess of major psychoses.

As students Africans are exposed to stresses such as academic inadequacy, loneliness, career choice restrictions and accommodation difficulties (Anumonye, 1967). Nevertheless, of all immigrant groups African patients seem to conform most to the selection model. Now that the British government has ceased to fund universities for the training of overseas students, the nature of the African community in this country may be expected to change. This may in turn influence the future pattern of psychiatric morbidity.

Conclusion

Although 25 years have passed since the arrival of the first immigrants from the New Commonwealth, migration should not be regarded as a discrete event.

Adaptation to a new environment may be undermined in later life by crises of career, family or impending old age. Afro-Caribbeans have an increased rate of first admissions in the 35-45 year age group, a range which is atypical for both schizophrenia and affective psychosis (Carpenter & Brockington, 1980). Persistence of morbidity has also been demonstrated in earlier migrations (Hitch & Rack, 1980). It is not certain to what extent acculturation may cause pathology such as hysteria, somatization and the acute psychotic reaction to be relinquished in favour of symptoms more characteristic of the host population. That this morbidity is not static has been underscored by evidence of a shift away from psychosis towards depression in individual patients (Littlewood & Lipsedge, 1981b). Longitudinal studies would help clarify this, but research is hampered by problems of phenomenology. The theoretical perspective of psychiatry in this country will not readily accept concepts such as the reactive psychosis (Strommgren, 1974) and this should be borne in mind when interpreting epidemiological results. On the other hand, overcaution in assessing symptoms in culturally distinct patients may lead to an emphasis on the unusual or atypical disorder when a diagnosis such as schizophrenia may be more appropriate. A prospective study that did not rely on case records would help to improve our understanding.

The evidence suggests that earlier recognition and treatment of psychiatric disorder is needed. Education of health professionals may go some way towards facilitating access and improving the psychiatric care available. Accurate assessment of the immigrant patient demands adequate communication and an understanding of cultural influences (Cox, 1976, 1982). The emphasis on somatic complaints may delay diagnosis or direct patients to inappropriate hospital departments. Two areas where research may be of value are general practice and liaison psychiatry. The immigrant minorities are a factor which cuts across all psychiatric research, ranging from social psychiatry to clinical drug trials. An altered response to psychotropic drugs may arise out of diet, genetic differences in metabolism and cultural factors (Lewis *et al*, 1980; Allen *et al*, 1977). A wider acknowledgement of these issues would increase awareness and lead ultimately to improvements in health care. It also affords an opportunity to re-examine psychiatric concepts that have evolved within our own cultural frame of reference.

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Depression in Mentally Handicapped Patients: Diagnostic and Neuroendocrine Evaluation

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The dexamethasone suppression test was administered to 12 mentally handicapped depressed patients. One of the four patients with major depressive disorder, but none of those with other diagnoses, failed to suppress cortisol production. The International Classification of Diseases and the Newcastle Diagnostic Index were found to be unreliable for use with severely mentally handicapped patients. Modifications are proposed which would allow the Research Diagnostic Criteria and Hamilton Rating Scale for Depression to be applied to patients with any degree of mental handicap.

The simultaneous presence of mental handicap and depression is a great challenge to diagnostic skills, particularly when communication is exclusively non-verbal (Reid, 1972; Wright, 1982). A specific laboratory marker for depression would therefore be particularly welcome to psychiatrists working in the field of mental handicap. Evidence has steadily been accumulating of a disturbance of the hypothalamic-pituitary-adrenal axis in depressive illness (Schlessler *et al*, 1980): many depressed patients do not suppress cortisol production following the administration of a synthetic glucocorticoid, and the dexamethasone suppression test (DST) has been proposed for the diagnosis of endogenous depression (Carroll *et al*, 1981). The diagnostic problems of mentally handicapped patients are somewhat analogous to those of pre-pubertal children, in whom the DST has proved a useful diagnostic aid (Poznanski *et al*, 1982). This paper is the first report of the use of the DST in mentally handicapped patients.

Laboratory tests must be validated against clinical criteria, but the research instruments widely used for this purpose in affective disorders research—such as the Hamilton Rating Scale for Depression (HAMD) (Hamilton, 1967) and the Newcastle Diagnostic Index (NDI) (Carney *et al*, 1965)—have themselves rarely, if ever, been applied to mentally handicapped subjects (Matson, 1982; Kazdin *et al*, 1983; Russell & Tanguay, 1981; Ballinger *et al*, 1975). The same is true of the more recently introduced operationalised diagnostic criteria, notably the Research Diagnostic Criteria (RDC) (Spitzer *et al*, 1978): a recent literature review (Sovner & Hurley, 1983) could find no reports of the RDC being applied to mentally handicapped depressives. Thus idiosyncratic methodology may help to account for the disparate results of psychiatric

surveys in this field (Wright, 1982). The applicability of these research instruments and of the widely used International Classification of Diseases (ICD) (World Health Organisation, 1978) is also evaluated in this paper.

Method

The group studied was a consecutive series of patients referred for assessment of possible depression to psychiatrists attached to the Section of Psychiatry of Mental Handicap, St George's Hospital Medical School, which serves four Health Districts. Referrals were mostly from nursing and adult training centre staff: none were from general practitioners. There were 12 patients eligible for the DST, nine males and three females; eight were resident in hospital and the rest in the community. None required psychiatric admission as a result of the depression.

The DST was administered as part of the standard psychiatric assessment of depression. Informed consent was obtained from the patient where possible; otherwise, it was obtained from the next-of-kin, or from the care-giver when the next-of-kin was not in contact with the patient.

Exclusion criteria were as set out by Carroll *et al* (1981, 1982): temporal lobe epilepsy, hepatic enzyme induction by phenytoin and barbiturates, and high doses of benzodiazepines were of particular importance in mentally handicapped patients. Following a report of the induction of hepatic enzymes by carbamazepine (Privitera *et al*, 1982), patients on this drug were also excluded.

The dosage and timing of the test were in accordance with the procedure described by Coppen *et al* (1983): dexamethasone (1 mg) was given orally to the patient at 2000 hours, the author telephoning to confirm this before 2030 hours. A blood sample was obtained by 1530 hours the following day, and plasma cortisol estimated by radioimmunoassay. Laboratory staff were blind to the clinical ratings. A cortisol level of greater than 5 µg/dl was taken as an abnormal result.

All patients were classified according to the RDC and the glossary of mental disorders in the ninth revision of the ICD. Ratings were also made on the HAMD and the NDI. The ratings were made exclusively on data obtained directly from the patient and from a close informant; this was sometimes a parent and sometimes nursing or hostel staff. Historical data were obtained from clinical records.

Results

Patients

Some characteristics of the subjects are shown in Table I. The degree of handicap varied, from five patients at one extreme (cases 1, 7, 8, 9 and 11) who had not acquired verbal communication to three at the other (cases 5, 6 and 12) whose IQs were within the normal range, but who were included as they had been referred to psychiatrists in the mental handicap services, were resident in a mental handicap hospital (case 6) or hostel (cases 5 and 12), and had always functioned at levels much lower than might have been suggested by their IQs. Cases 3 and 11 had Down's syndrome, and birth trauma was probably responsible for the mental handicap of case 4, who had been born after a long labour and forceps delivery and also suffered from athetoid cerebral palsy. Only one patient (case 9) showed autistic features.

DST

Eleven of the twelve patients were effective suppressors of cortisol production, with post-dexamethasone cortisol levels below 5 µg/dl. One of the four major depressives (case 4) was the only non-suppressor, with a level of 13.5 µg/dl (Table I). There was no correlation of cortisol levels with severity of depression or endogeneity, as measured by the HAMD and NDI respectively.

The importance of the exclusion criteria was highlighted by one patient on carbamazepine, who was given the DST but was withdrawn from the study when a paper appeared demonstrating that carbamazepine produces false positive DST results (Privitera *et al*, 1982): he had a plasma cortisol of 15.1 µg/dl.

RDC

Four of the twelve patients had major depressive disorder, one of these carrying a 'definite' diagnosis. Seven further patients had minor depressive disorder, six of these being 'definite' and one 'probable'. The remaining patient fulfilled the criteria for 'probably currently not mentally ill'.

Two of the symptoms in the checklist for major depressive disorder occurred in only one patient (case 2); these were 'feelings of self-reproach or excessive or inappropriate guilt' and 'recurrent thoughts of death or suicide or any suicidal behaviour'. Apart from suicidal behaviour, these symptoms were found impossible to rate with any degree of conviction in the non-verbal patients. Four symptoms from the checklist are required for the diagnosis of 'probable' major depressive disorder and five for 'definite'; as it was feasible

to rate only six out of the eight possible symptoms in the non-verbal patients, their diagnoses were reviewed using the more lenient criteria allowed by the RDC for past episodes, with only three and four symptoms required to achieve probable and definite diagnoses respectively. The consequence was to convert cases 7 and 9 from definite minor to probable major depressive disorder, bringing the total of major depressives to six—a number which might have been expected to yield several positive DSTs if results in non-handicapped depressives had been applicable.

The RDC allows distinction between various subtypes of major depressive disorder: all four major depressives had primary rather than secondary disorder, three of them having had no signs of other psychiatric disturbance in the past year ('simple' subtype) as far as could be ascertained from the case notes: two were classified as probably endogenous and one as retarded. None of the depressions were secondary, recurrent, incapacitating, agitated, or situational.

ICD

Eight patients were classified as having a manic depressive psychosis category (296), two had adjustment reactions (309) to bereavement, and one could only be classified as 'depressive disorder not elsewhere coded' (311). The remaining patient had no affective disorder, but on the basis of the case history was thought to have an explosive personality disorder (301.3). No cases of neurotic depression (300.4) were found.

NDI

The mean diagnostic index was 5.3, and three out of the twelve scored above the 'endogenous' threshold of 5. The items of the index were then analysed individually. No patients scored positively on the items 'nihilistic delusions' or 'blames others'.

HAMD

Scores were calculated for the sum of the first 17 items of the scale, producing a mean score of 11.3 (Table I). The individual items with the lowest scores were psychic and somatic anxiety, gastrointestinal and genital symptoms, and hypochondriasis. It is possible that the lower scores were due to a genuinely lower level of symptomatology, but positive scores on these particular symptoms rely heavily on verbal report, and it seems more likely that the low verbal ability of this group of patients has artificially depressed the scores. Elimination of these symptoms (items 10, 11, 12, 14 and 15), together with two wholly verbal symptoms, 'guilt' and 'insight' (items 2 and 17), leaves ten items applicable to patients with any degree of mental handicap. It should be noted, however, that low scores on two of the remaining items ('suicide' and 'work and activities') also rely on verbal report and cannot be elicited by observation. Doubts must also exist as to the inclusion in the full rating scale of 'depersonalisation and derealisation', on which no mentally handicapped patient scored positively; this item is not required for the 17 item total.

TABLE
Clinical assessments and cortisol concentrations

Case No.	Age, years	Sex	Intelligence (most recent test), IQ	RDC ²	Diagnosis of affective disorder ICD-9	Raw	HAMD Adjusted	NDI	Plasma cortisol: $\mu\text{g/dl}$
1	17	M	40	Major (D)	Manic-depressive psychosis, depressed type (296.1)	13	19	8	1.5
2	19	M	58	Major (P)	Manic-depressive psychosis, depressed type (296.1)	15	17	5	0.9
3	30	M	49	Major (P)	Manic-depressive psychosis, depressed type (296.1)	8	14	5	<0.4
4	37	M	65	Major (P)	Manic-depressive psychosis, depressed type (296.1)	12	19	5	13.5
5	24	M	79	Minor (P)	Manic-depressive psychosis, circular type but currently depressed (296.3)	9	12	7	<0.4
6	26	M	78	Minor (D)	Manic-depressive psychosis, depressed type (296.1)	10	16	5	1.5
7	30	F	(MA < 1 year) ¹	Minor (D)	Depressive disorder, not elsewhere coded (311)	17	26	8	2.5
8	30	F	(MA = 2½ years)	Minor (D)	Adjustment reaction with mixed disturbance of emotion and conduct (309.4)	12	21	5	<0.4
9	35	M	(MA = 1½ years)	Minor (D)	Manic-depressive psychosis, depressed type (296.1)	8	12	5	1.3
10	37	F	43	Minor (D)	Manic-depressive psychosis, depressed type (296.1)	12	14	5	3.8
11	49	M	(MA = 3 years)	Minor (D)	Brief adjustment reaction (309.0)	10	12	4	0.9
12	20	M	84	CNMI (P)	Explosive personality disorder (301.3)	9	10	1	0.8

1 MA = Mental age

2. Major = Major depressive disorder; minor = minor depressive disorder; D = definite; P = probable; CNMI = currently not mentally ill

To obtain a Hamilton total comparable to that using the standard 17 items, the sum of the scores on the ten remaining items (items 1, 3-9, 13 and 16) must be scaled up by a factor of 1.73. The adjusted total scores are given in Table I: the corrected mean total is 16.0, nearly 5 points higher than the raw mean total. Correlation of the corrected totals with plasma cortisol levels was poor ($r=0.26$, $P=0.4$), but correlation with the NDI was high ($r=0.62$, $P<0.05$).

Discussion

As the patterns of care of mentally handicapped people change, more referrals to psychiatrists now originate from community nurses and other paramedical personnel on community mental handicap teams; without the general practitioner 'filter' between the community and the out-patient psychiatric clinic it would seem likely that milder and less intractable cases will be seen by the psychiatrist. This is confirmed by the present data, which reveal a generally mild level of depression, comparable with that treated in general practice (Sireling *et al.*, 1985).

The only positive DST was found in one of the patients with major depression. The sample size was small, but more non-suppressors might have been expected: giving the DST to patients with senile dementia, Spar & Gerner (1982) found that nearly half of those without major depression were non-suppressors. Although there were no false positives and the test was acceptable to patients, families, and care-givers, the DST seems to have little to offer in the differential diagnosis of depression in mentally handicapped patients. The reasons for this finding are not clear, and replication on a larger sample is required.

So far as the RDC are concerned, the excess of minor depression compared with major depression in this cohort is probably an artefact, resulting from the nature of several of the symptoms in the checklist for major depressive disorder. The requirements appear particularly stringent for non-verbal patients, and application of the 'past episode' criteria may give diagnoses more equivalent to those in non-handicapped subjects.

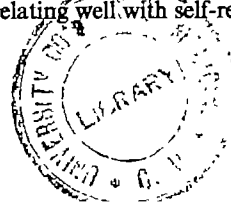
Rating the subtypes of major depressive disorder also presents difficulties in the mentally handicapped patient: in the present study, neither the memories of the patients nor the case notes were adequate to make decisions about the primary, secondary, or recurrent nature of the disorder. The criterion for a rating of 'incapacitated' is "inability to carry out any relatively complex goal-directed activity, because of severity of depressive symptoms", and this clearly is inapplicable to the more handicapped patient. Similarly, the subtype 'psychotic' requires the presence of delusions

or hallucinations—both hard to elicit in non-verbal patients—and four of the five manifestations of 'retarded' major depression depend on the presence of speech: this accounts for the discrepancy between the rarity of this subtype in the present cohort and the significantly greater degree of retardation, rated on the HAMD, and of depressive psychomotor activity rated on the NDI. Similar criticisms apply to the other subtypes of major depressive disorder, so that in general it is not feasible to rate any subtype except 'situational' in the non-verbal mentally handicapped patient.

The ICD glossary defining the rubric 'manic depressive psychosis, depressed type' (296.1) can be applied to patients with any level of mental handicap, as almost all the features described can be observed rather than elicited verbally. In contrast, the glossary for 'neurotic depression' (300.4), which was not identified in this cohort, is more difficult to apply, as it relies on terms such as 'disproportionate depression' and 'preoccupation with the psychic trauma', which are difficult enough to assess even in the absence of mental handicap.

The high proportion of 'endogenous' patients on the NDI could be due to selective bias on the part of those referring mentally handicapped patients to psychiatrists: such patients rarely present with complaints of dysphoria, and suspicion of a depressive condition may only be aroused when symptoms such as depressive psychomotor activity occur. Certain items of the NDI present problems of scoring in mentally handicapped patients, e.g. 'nihilistic delusions', 'guilt', and 'blames others for illness'; its validity must be doubted under these circumstances.

The more objective items of the HAMD such as sleep, weight loss, and effect on work and activities were readily rated, as was depressed mood, and this supports the clinical relevance of the adjusted Hamilton total score with the items dependent on verbal ability omitted. However, the raw score may be preferable for mentally handicapped patients with adequate communication skills, because items such as 'guilt' which are excluded from the adjusted form may be found frequently among mentally handicapped patients who are able to report such feelings (Heaton-Ward, 1977). The problems relating to individual items have been discussed, and the probable effect of selective referral bias can be seen here, as in the NDI, in the excess of retardation and the relatively low level of reported anxiety, somatic, and genital symptoms. Kazdin *et al.* (1983), who asked informants to complete the HAMD on 110 mentally handicapped adults (almost all mildly or moderately handicapped), also found it a satisfactory measure, correlating well with self-report measures.



Adequate tests of the validity of the adjusted score must await the collection of a further sample.

It is therefore suggested that widely used research tools such as the RDC and HAMD can, with modifications, be applied to the entire range of mental handicap in the diagnosis of affective disorder and the measurement of its severity. However, the modifications must be specified and preferably standardised, so that studies will be comparable: this paper proposes certain modifications. Particular problems with the ICD-9 and the NDI render these instruments unreliable for use with mentally handicapped patients.

The possibility of applying such sensitive instruments to mentally handicapped patients would aid the evaluation of new psychiatric techniques such as the DST, and would facilitate the evaluation of treatment methods in mentally handicapped patients with affective disorders.

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Emotional Attitudes and Direct Communication in the Families of Schizophrenics: A Cross-National Replication

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To examine how the measure of expressed emotion relates to family life, 30 relatives of schizophrenic patients were assessed for EE and then observed in ten-minute discussions with the patients. It was found that high-EE relatives express more negative emotional statements than low-EE relatives when face-to-face with the patient; they also talk more rapidly, and this speech rate is correlated with the patient speech rate. Whereas low-EE relatives expressed few criticisms or intrusive statements, high-EE over-involved relatives were more intrusive, and high-EE critical relatives were more critical as well as more intrusive in direct interaction. These findings emphasise the importance of understanding divergent EE sub-styles and the complementary behaviour of patients. The findings of an earlier Los Angeles study were replicated.

Over the last decade, evidence has been accumulating that the family emotional atmosphere to which schizophrenic patients return from hospital, as indexed by expressed emotion (EE), critically affects the likelihood of relapse from schizophrenia (Vaughn & Leff, 1976a; Leff & Vaughn, 1981; Vaughn *et al.*, 1984), and that interventions directed at changing EE reduce this risk (Leff *et al.*, 1982, 1985).

In another experimental trial, Falloon *et al.* (1985) have shown that psychosocial interventions, aimed at improving communication skills and problem-solving behaviour in families at risk, reduce the likelihood of relapse and improve the patient's social functioning. These changes are significantly related to the style of direct affective expression, as indexed by affective style (AS) (Doane *et al.*, 1985, 1986).

Most recently, Hogarty *et al.* (in press) examined the additive effect of two different treatment methods to maintenance antipsychotic medication: psycho-educational family treatment and individual social skills training. Of the 90 patients who stayed in treatment, the medication comparison group had a relapse rate of 41%, whereas the group that received both family and social skills training had a relapse rate of 0%, at one year follow-up. Furthermore, from measurements of EE at intake and one year follow-up, Hogarty *et al.* found that there were no patient relapses in any household that changed from high to low EE independent of treatment; in households that stayed high EE, only the combination of family therapy and social skills training prevented relapse.

Thus, both naturalistic and treatment studies suggest the importance of EE and AS as key indices of family processes. The primary aim of the current study was to examine the relationship between these two measures, to see how EE attitudes were expressed in direct interaction with the patient. It focused particularly on the differences between high-EE relatives with critical and over-involved attitudes.

Previous studies of the interactional correlates of EE attitudes in relatives of schizophrenics have assessed direct communication by a relative to a patient, but only in the presence of an interviewer (Kuipers *et al.*, 1983) or a second relative (Miklowitz *et al.*, 1984). It is possible, however, that the presence of others can suppress the expression of high-EE attitudes. For instance, one parent in a triadic discussion may dominate the discussion such that the other parent says very little, thus making it difficult to assess their individual styles of emotional expression. Alternatively, the presence of another relative can modify the way they express themselves to the patient. For instance, Valone *et al.* (1983) studied the correlates of EE in dyadic and triadic interactions between parents and their non-schizophrenic adolescent offspring. They found that parents often altered the number and harshness of their criticisms when interacting with their child in the presence of their spouse compared with their behaviour when alone with the offspring. The current study overcomes such problems, by studying dyadic communication between patient and relative.

A second aim of the current study was to compare

the expression of emotional attitudes in Britain and the United States. The recent cross-national replication of the British relapse studies (Vaughn *et al*, 1984) demonstrated that relapse in a US sample was predicted by similar levels of criticism and emotional over-involvement as found in the earlier British studies, suggesting that the attitudes of relatives have a similar impact on patients in the two countries.

However, this still leaves the question of whether the crucial mediating mechanism of intrafamilial behaviour is comparable in samples from the two countries. Miklowitz *et al* (1984) demonstrated a correspondence in a US sample between EE and AS, finding that relatives with critical attitudes were more critical in direct interaction and relatives with over-involved attitudes were more intrusive. However, because of different cultural norms in the expression of emotion, it may be that the overt expression of attitudes, particularly negative ones towards one's offspring or spouse, varies from country to country. We aimed to compare results from this British sample with data from previous US studies of EE correlates, to determine whether such cross-national variations exist.

Method

Participants

The sample was selected from recent admissions to Friern Hospital, a psychiatric hospital in North London. All files of patients admitted to acute admission wards were reviewed regularly for two years. Patients were selected for screening if: (a) they had lived with a partner or one or both parents for at least one month out of the previous three, (b) they were likely to be diagnosed as having schizophrenia, and (c) they had been admitted for the first time within two years of the current admission date. Patients were excluded if they showed evidence of an organic brain syndrome, or significant alcohol or drug abuse.

Prospective participants were given the Present State Examination (PSE). The clinicians had been previously trained to reliability on the rating of symptoms. All patients were interviewed within the first few weeks of admission (median = 12 days). If patients met criteria for a positive diagnosis of schizophrenia according to the PSE, the relatives were asked if they would consent to participate in the study. There were two exceptions, each of whom had one first-rank symptom which would have led to a positive diagnosis of schizophrenia according to the CATEGO program, but who presented a clinical picture of a manic psychosis; these were excluded from the trial. Of the sample of 24 patients who passed these initial screening procedures, one set of parents refused, and one family was not approached because it was already too involved with mental health services. The remaining 22 families agreed to participate. There were no further drop-outs from the study.

Of the 22 patients with a positive diagnosis of schizophrenia who were included, 21 met CATEGO criteria for schizophrenia on the basis of the presence of first-rank symptoms from Schneider's 'nuclear syndrome' of schizophrenia. The other participant showed delusions and auditory hallucinations, which fit the CATEGO criteria for 'paranoid psychosis.'

Additional historical data, independently collected, enabled diagnoses to be made on the basis of DSM-III (American Psychiatric Association, 1980). All of the patients satisfied criteria for a diagnosis of schizophrenia except one, whose symptom pattern suggested the possibility of a schizo-affective disorder.

The sample was from a relatively acute psychiatric population. This was the first admission for 59% of the sample, and the mean number of previous admissions was 0.73 (*s.d.* = 1.24), all of which had occurred within the previous two years. Most had lived continuously with their close relatives for the previous three months: a mean of 85 days out of 90 (*s.d.* = 16 days).

The sample was heterogeneous for age, gender, ethnicity, and socio-economic status: 8 were female and 14 male; 12 were British caucasians, 5 West Indian, 3 continental European, and 2 of mixed ethnic origin. The mean socio-economic status was 'unskilled' according to the UK Registrar General's classification (Office of Population Censuses and Surveys, 1980). Some had been students or had semi-skilled jobs prior to admission, but most were unemployed: the mean length of unemployment was 12 months out of the previous 24 (*s.d.* = 10 months). The mean age was 24.6 years, ranging from 18 to 51. The two oldest patients had experienced long-standing encapsulated delusions, but the families and patients had adapted to these with few problems, and they had not sought psychiatric treatment. They were admitted to hospital only when other psychotic symptoms appeared.

Medication levels were obtained from charts and converted to chlorpromazine dose equivalents (Davis, 1976). The mean dose at the time of the initial PSE was 371 mg, and at the time of the direct interaction task was 328 mg.

The 30 relatives had a wide range of socio-economic statuses: the mean occupational level was social class III (M), 'skilled manual workers', which was higher than that of the patients. Most of the relatives were parents: eight patients lived with both parents, eight lived alone with their mothers, and two alone with their fathers. Four patients lived with a partner: three with husbands and one with a wife. The mean age of the relatives was 50.2 years, ranging from 28-71 years.

Camberwell Family Interview

To elicit and measure EE attitudes, key relatives were administered the modified Camberwell Family Interview (CFI) according to the procedures of Vaughn & Leff (1976b). This interview was conducted at home, while the patient was in hospital, it was administered separately to each relative, a median of 26 days after intake.

The tapes were rated by clinicians extensively trained at the Institute of Psychiatry to rate EE; most were rated by two people. Five aspects of the relative's attitude were assessed: the number of critical comments (CC), the number

of positive remarks, the presence or absence of hostility, and ratings of emotional over-involvement (EOI) and warmth on six-point scales. A sample group of tapes classified by two raters into high and low EE showed agreement for 12 of the 13 tapes. Individual scale Pearson product-moment correlations were as follows: CC, $r=0.86$ ($P<0.01$); EOI, $r=0.94$ ($P<0.01$); hostility, $r=0.67$ ($P<0.02$). A further seven tapes which were rated by two raters for warmth and positive remarks had Pearson correlations of 0.74 ($P<0.05$) and 0.86 ($P<0.01$) respectively.

Classification

Relatives were classified as high EE if they made six or more critical comments or had a rating of three (moderately high) or more on emotional over-involvement. All other relatives were classified as low EE. These criteria are the same as used in recent EE research (Leff *et al.*, 1985; Miklowitz *et al.*, 1984). Using these criteria, 23 relatives were classified as high EE and 7 as low EE. This is a higher proportion of high-EE relatives than was reported in Camberwell (Vaughn & Leff, 1976a) and may reflect subculture variation.

The high-EE group was further divided into three subgroups:

- (a) critical group ($n=14$): those relatives scoring high on CC but low on EOI (using the same criteria as above)
- (b) emotionally over-involved group ($n=5$): those relatives high on EOI but low on CC
- (c) critical and emotionally over-involved group ($n=4$): those relatives high on both CC and EOI.

Ten relatives were rated as showing hostility; in every case they also made six or more CCs, confirming previous work (Vaughn & Leff, 1976a).

Relatives' EE status was examined in relation to family type. There were no significant differences in the percentages of low and high-EE relatives among single-parent, dual-parent, and spousal family units, and none in the percentages of low and high-EE relatives between mothers and fathers. There were also no significant differences across the EE subgroups. It was observed, however, that seven out of nine relatives with high EOI scores were mothers. The distribution of the four spouses across the EE categories did not differ markedly from the distribution of parents: one was low EE and three were high EE, two of these being critical and one emotionally over-involved.

Because eight patients were living with more than one key relative, a second classification was used for analyses by family unit rather than by individual relatives. If both parents were low EE, the family was considered dual low EE ($n=4$); if one was high EE and the others low EE, the family was considered mixed EE ($n=3$); if both were high EE, the family was considered dual high EE ($n=15$). All family units, whether of one or two relatives, could thus be classified as uniformly low, uniformly high, or mixed EE.

To determine whether there were differences in EE which related to characteristics of the sample, demographic variables were compared between high EE and low combined with mixed EE families. There were no differences for any of these variables.

Direct family interaction procedures

After the individual assessment of patient and relatives, a meeting was convened to assess face-to-face family interactions. This usually took place shortly before the patient was discharged, and was usually in the hospital, but sometimes at home. The median length of hospital stay was 42 days, and the median time to the interaction meeting 40 days.

The direct interactional task was a discussion between the patient and each relative separately about an issue of conflict, further details of the procedure are described in Miklowitz *et al.* (1984). The relative and patient were left to discuss the issue on their own, while the research worker monitored the conversation from another room and returned after ten minutes. The researcher would only return before that time if conversation stopped. Another topic would then be introduced, until a total of ten minutes' discussion had taken place. Whether the relative or patient cue was used as the first stimulus was randomised.

Immediately after these discussions, the clinician held a short debriefing session to answer questions, and to defuse any conflicts which might have arisen.

Direct interaction coding

Transcripts of the interactions were made and checked carefully before being sent to the UCLA Family Project for coding. A critical incident system of coding—the affective style (AS) coding scheme (Doane *et al.*, 1981)—was used. These codes are defined briefly below.

Benign criticisms are circumscribed, matter-of-fact, or directed towards specific incidents or sets of behaviour.

Harsh criticisms is a category made up of several sub-categories: 'Personal criticisms', in which criticism is made of broad classes of behaviour, or the patient's basic negative character; 'Guilt induction', in which the relative conveys that the patient is to blame for some event which has distressed the relative, and 'Critical intrusiveness', in which the relative implies knowledge of the patient's internal states and is critical of his intentions.

Neutrally-intrusive statements imply knowledge of the patient's emotional states, ideas, or preferences when there is no apparent basis for such knowledge (e.g. "You went out for a drink because you were upset."). These statements are neutral in tone, and do not imply criticism, but might be experienced by patients as intrusive and stressful.

Support the relative conveys that he or she feels genuine concern for the patient or about the patient's problems or behaviour, or supports the patient in other ways (e.g. "I know of nobody who makes more of an impression on others than you do.").

Reliability

The interaction transcripts were coded by two coders, blind to EE status. Inter-rater reliability was established prior to the present study on a sample of 27 transcripts from an earlier study of families containing a nonpsychotic adolescent, it was highly significant for benign criticisms ($k=0.85$, $P<0.0001$), harsh criticisms ($k=0.89$, $P<0.0001$),

neutral-intrusive statements ($k=0.85$, $P<0.0001$), and support ($k=0.86$, $P<0.0001$).

AS profile patterns

In addition to analyses of means, analyses were made on a case-by-case basis, and a profile method was designed to parallel the EE profiles. Previous studies have assigned a negative AS profile to relatives who express six or more neutral intrusions or one or more personal criticisms, guilt-inductions, or critical intrusions (e.g. Doane *et al*, 1985). However, intrusive profiles and critical profiles have not been separately defined. Since Valone *et al* (1983) showed that benign criticisms also were related to high-EE attitudes, relatives were assigned a 'critical' AS profile if they had at least one personal criticism or an excessive use (six or more) of the less impactful code of benign criticisms. They were assigned as 'intrusive' AS profile if they had at least one guilt induction code (which has an intrusive quality) or an excessive use (six or more) of the less impactful code of neutral intrusions. Some relatives met both criteria; those who met neither were assigned a 'good' AS profile.

Referrals

Eleven high-EE families were subsequently included in a randomised trial of educational, pharmacological, and psycho-social interventions, comparing a group for relatives with in-home family therapy. However, all data for the current study were collected prior to any intervention.

Results

Expressed emotion and affective style

The main aim of this study was to examine the relationship between emotional attitudes expressed about a patient to a neutral interviewer and direct emotional communication with that patient. It was hypothesised that high-EE relatives would be more emotionally negative than low-EE relatives—critical, intrusive, or both—when face-to-face with the patient. Table I shows the number of different

TABLE I
Means and standard deviations for high and low-EE interactions: affective style scores and speech rates

Affective style	Low EE		High EE	
	Mean	s.d.	Mean	s.d.
Criticism	0.86	1.86	5.00	5.21
Neutral intrusions	3.86	3.76	6.26	4.95
Total negative AS	4.71	5.47	11.26	7.71
Support	2.71	3.55	0.65	1.15
Relative speech rate	43.0	20.9	70.8	35.0
Patient speech rate	22.0	12.8	35.7	19.5
Total speech rate	65.0	27.0	106.5	46.7
Speech turnovers	72.0	45.3	94.5	44.2

affective style codes, as well as the total number of negative codes, used in ten minutes of interaction. For each analysis a two-tailed *t*-test was used. Variances were pooled unless they were significantly different, in which case a separate variance estimate was used.

High-EE relatives expressed significantly more negative AS statements in total than low-EE relatives ($n=30$, $t=2.08$, $P<0.05$). Breaking down the negative AS total score into criticisms and intrusions, high-EE relatives made significantly more criticisms than low-EE relatives ($n=30$, $t=3.20$, $P<0.003$). This was so regardless of whether the low frequency harsh criticisms were included in the total score. There was a trend for high-EE relatives to make more neutrally intrusive statements and fewer supportive statements, but neither trend reached statistical significance.

Expressed emotion and speech patterns

The relationship between EE and speech patterns during the interaction was examined. The number of words used by relatives during the ten minutes of interaction was measured from the number of lines on the verbatim transcript. High-EE relatives used a significantly greater number of words (65% more) than low-EE relatives ($n=30$, $t=1.98$, $P<0.05$) (Table I). This could be due to a generally more heated interaction, or to the high-EE relative being more dominant and lecturing. Therefore, the number of words used by the patient was added into the analysis. A two-way analysis of variance was conducted with EE (high or low) as one factor and relationship (relative or patient) as the other (this factor was correlated because patient and relative were in the same interaction). There were significant effects of EE ($F=4.94$, d.f. = 1,28, $P<0.03$) and of relationship ($F=18.57$, d.f. = 1,28, $P<0.0002$), but the interaction was not significant. Relatives thus use significantly more words than patients (twice as many), and the total number of lines used in interactions which included high-EE relatives was significantly greater (64% more) than for low-EE relatives. Since all conversations were exactly ten minutes long, it can be concluded that the average rate of speech was significantly greater over the course of the conversation. Patients tended to use more words speaking to high-EE relatives than to low-EE relatives (62% more), the difference being comparable to that found between high and low-EE relatives. Furthermore, there was a significant and positive correlation between the relatives' word count and the patient's word count (Pearson's $r=0.47$, $P<0.004$). Interaction with a high-EE relative is thus characterised by high verbal activity on the part of both relative and patient.

To investigate the length of individual speeches, a count was also made of the number of turnovers (i.e. changes from one speaker to another) during the ten minutes. Although the mean for high-EE interactions was 31% larger, the difference was not significant. This suggests that high-EE interactions were not characterised by longer speeches, but had at least as many changes of speaker as low-EE interactions.

EE sub-categories and affective style

A second aim of this study was to examine how different

TABLE II
Mean AS scores for low-EE and high-EE subgroups of relatives

Affective Style	Low EE	Critical	EOI	Critical and EOI
Criticism				
Mean	0.86	6.14	2.40	4.75
s.d.	1.86	5.79	1.52	6.18
Neutral intrusions				
Mean	3.86	5.57	6.20	8.75
s.d.	3.76	4.72	4.97	6.07

kinds of high-EE attitudes (criticism and EOI) would be expressed in direct interactional behaviour with the patient. It was hypothesised that a critical attitude would be seen as direct criticism of the patient and an emotionally over-involved attitude would correlate with neutral intrusions in the interaction. To examine this, an analysis of variance (ANOVA) was performed on the four sub-categories of EE: low, high on critical comments only, high on EOI only, and high on both EOI and critical comments.

It was found, as expected, that differences among the three high-EE sub-groups would not be significant for total AS, support, and rate of speech. Thus, all three high-EE sub-groups of relatives were consistently higher than low-EE relatives on total AS and rate of speech. Previous analysis had shown no mean differences between high and low-EE relatives on weekly face-to-face contact. However, there were found to be significant differences between sub-groups ($n=30$, d.f.=3, $F=2.96$, $P<0.05$), with the pure high-EOI relatives spending far more time (61 hours) with the patients than other kinds of relatives (26 hours).

As predicted, when the negative AS codes were broken down into their components of criticisms and neutrally intrusive statements, a more complex pattern emerged (Table II). All relatives who scored highly on CC (i.e. the high-EE critical and the high-EE critical + EOI subgroups combined) were compared in planned contrasts (on the basis of Miklowitz *et al.*, 1984) with the other two subgroups. The combined high-CC group made significantly more criticisms in direct interaction than both the low-EE group ($n=25$, $t=3.27$, $P<0.01$) and the purely high-EOI group ($n=23$, $t=2.27$, $P<0.05$).

Similarly, for the analysis of neutrally intrusive statements, the combined high-EOI groups were compared in planned contrasts with the other two sub-groups. The combined high-EOI group did not make significantly more neutral intrusions than the low-EE group ($n=16$, $F=2.25$, NS) or the purely high-CC group ($n=23$, $F=0.87$, NS), although the pattern of means was as expected.

The pattern of means for criticisms and intrusive statements in this British sample (Fig. 1) closely follows that seen in the American sample reported by Miklowitz *et al.* (1984), and replicates their findings.

However, although the findings for total criticisms are quite clear-cut, the pattern of means for neutrally-intrusive

statements is not so clear. Specifically, the mean number of neutral intrusions made by the high-EE critical subgroup is about the same as the mean number made by the high-EE EOI subgroup. In order to assess what this means about the way critical attitudes are displayed in direct interaction, correlational and case-by-case profile analyses were performed.

Firstly, the correlation between the number of critical statements and the number of neutrally intrusive statements was not significant ($n=30$, Pearson's $r=0.20$, NS). Secondly, the correlation between EOI (scored dichotomously as high or low) and neutral intrusions was marginally significant ($n=30$, Pearson's $r=0.24$, $P<0.10$) as predicted, whereas the correlation with total criticisms was not. However, the correlations between critical attitudes shown in the CFI interview and critical comments and neutral intrusions in direct interaction were both significant (criticism: $r=0.32$, $P<0.05$, neutral intrusions: $r=0.31$; $n=30$, $P<0.05$). This suggests that critical attitudes may be displayed in direct interaction as both criticism and intrusive statements, whereas emotional over-involvement may be displayed as neutral intrusions only.

This hypothesis is confirmed by case-by-case analyses, using the profile analysis described under 'Method'. Six of the seven low-EE relatives had good AS profiles, and 18 of the 23 high-EE relatives had critical or intrusive profiles—a highly significant difference (Fisher's exact $P<0.004$). Examining these data in greater detail, there is a significant overall skew in the data ($\chi^2=19.5$, d.f.=9, $P<0.05$), because relatives who are EOI in attitude (regardless of whether they are also critical) tend to be intrusive in interaction (six out of nine), and the majority of relatives who are critical in attitude tend to be either critical in behaviour or critical and intrusive in behaviour (8 out of 14).

Analysis of means, correlations, and case-by-case profiles therefore point to the same conclusion: that emotionally over-involved relatives display neutrally intrusive statements in face-to-face interactions, whereas critical relatives display both criticisms and intrusions.

Patient measures and EE

To determine whether the differences in relatives' attitudes towards the patient are related to differences in the

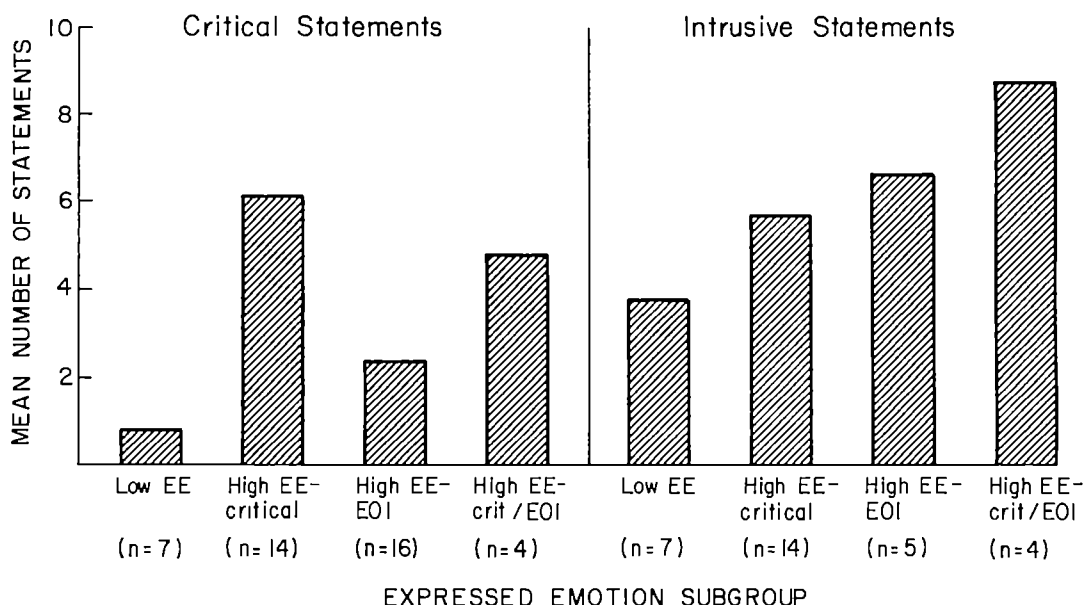


FIG. 1. Mean number of critical and intrusive statements made by relatives of schizophrenics with various EE attitudes

patient, we examined the relationship between relatives' EE and measures of premorbid socio-sexual functioning, medication levels and psychiatric state at intake.

The UCLA Social Attainment Survey was used as a measure of social-sexual competence prior to the onset of symptoms. Scores for patients from pure high-EE families were compared with those of patients from low and mixed-EE families. There were no significant differences, using either raw scores or the sex-specific criteria for defining good and poor premorbid status (Goldstein, 1978). There were also no significant differences in medication levels at the time of the PSE or at the direct interaction.

The PSE symptoms were grouped into the 38 CATEGO syndrome clusters (Wing *et al.*, 1974). A syndrome was considered present when the patient received a positive rating (1 or 2) on any symptom which was part of the cluster. The presence or absence of syndromes was examined in relation to the family EE status, comparing high-EE families with low and mixed-EE families combined. There were no significant differences in the proportion of patients who had any one syndrome present, but there were trends for syndromes related to depression. The four syndromes related to depression were therefore examined: depressive delusions and hallucinations, simple depression, special features of depression, and other symptoms of depression. It was found that only two out of the seven patients from the low or mixed-EE families had more than one syndrome of depression, whereas 11 out of the 15 patients from high-EE families had more than one syndrome of depression. This was a significant difference (Fisher's exact $P=0.05$). Furthermore, the presence of irritability was related to depression ($P<0.05$), although it did not reach significance

in relation to EE. These data may reflect a greater emotional reactivity to high-EE relatives in recent-onset schizophrenic patients, or stronger emotional responses by relatives to patients with a depressive aspect to their condition.

Family type

To investigate whether parents behaved differently from spouses, two-way ANOVAs were performed with family type as one factor, level of EE as the other, and EE and AS scales as dependent variables. The only significant differences were for spouses to show greater warmth in their attitude to relatives ($n=30$, d.f.=1, $F=7.71$, $P<0.01$) and more face-to-face contact ($n=30$, d.f.=1, $F=4.09$, $P<0.05$). There were no significant interactions, and thus it can be concluded that the relationship between emotional attitudes and behaviour is consistent across family types.

Discussion

This study examined how relatives of schizophrenics display EE attitudes in direct interaction with patients in a dyadic situation without the confounding presence of other relatives or clinicians. During a ten minute behavioural sample of dyadic communication between relative and patient about a conflictual issue, high-EE relatives were more affectively negative than low-EE relatives, being more critical, guilt-inducing, or intrusive. Such a negative emotional family climate is likely to be stressful, and appears to increase the risk of the development of schizophrenia-spectrum

disorders (Doane *et al*, 1981; Goldstein, 1985) as well as the risk of a relapse in someone recovering from an episode of schizophrenia (Doane *et al*, 1985; Vaughn & Leff, 1976a; Vaughn *et al*, 1984). Furthermore, changes in the affective climate of the family may decrease the risk of a relapse, as several recent family intervention studies have suggested (Strachan, 1985; in press).

Relatives who had EOI attitudes tended to be intrusive when face-to-face with the patient, either by making an excessive number of statements implying knowledge of what was going on in the patient's mind, or by making one or more guilt-inducing statements which blamed the patient for causing problems to the relative and implied that the patient had malicious or at least uncaring intentions. The picture was more complicated for relatives with highly critical attitudes. They were clearly more critical when face-to-face with the patient, by making either an excessive number of relatively benign criticisms or one or more statements which were harshly critical and attacked the character of the patient. However, it also appeared that relatives with a critical attitude were more intrusive as well. That high-EE critical and high-EE EOI relatives both use neutral intrusions in direct interaction raises the question as to whether the neutral intrusions used by high-EE critical relatives are different in type and impact from neutral intrusions used by high-EE EOI relatives or low-EE relatives. Research into this question is in progress.

Overall, these data closely parallel the pattern of findings of the connection between EE and interactional behaviour described by Miklowitz *et al* (1984), despite several differences in procedure and analysis between the two studies: we examined ten minutes of dyadic discussion without adjustment for line-length, whereas Miklowitz examined longer triadic discussions but adjusted them to control for word length. There is thus a comparability between intrafamilial behaviours in the UK and the USA, suggesting that family mechanisms mediating EE are comparable in the two countries.

A special aspect of the sample was that it was composed of recently admitted schizophrenics. Although most had suffered a recent onset of their symptoms, two patients had had longer-standing delusions. To assess whether these data were representative of a recent-onset sample, the main analyses were repeated excluding these two patients. Essentially similar results were obtained. Since the Miklowitz data were obtained from a more chronic population, this data thus appears to suggest that EE-AS relationships in a recent onset sample are similar to relationships in a more chronic sample.

It is important to note that while EE relates to the AS measure of interactional behaviour, the two measures are not identical in meaning. The EE measure taps emotional attitudes of the relative, whereas the AS measure taps direct interactional behaviour. The EOI measure in particular takes account of over-protective behaviour and excessive self-sacrifice, both of which are long term aspects of relatives' behaviour. That intrusiveness is associated with EOI does not mean they are identical, but the two are consistent in that intrusive remarks involve speaking for the patient, or making assumptions about his or her inner state.

Another important finding was that in high-EE pairs both relatives and patients used more words than was the case in low-EE pairs. Thus, in high-EE dyads, both participants are actively involved in the interaction, contradicting the thesis that high-EE relatives are dominant and the patients submissive. These data also argue against the idea that a negative affective style is merely a result of the high-EE relative being more dominant and speaking a higher proportion of time, thus producing more criticisms and intrusions. Furthermore, these data contradict the idea that a higher rate of negative behaviour is an artifact of word count: although high-EE relatives used 65% more words than low-EE relatives, they used more than twice as many intrusions and criticisms. It should be noted, however, that this pattern may change over the years if the patient becomes more chronic and withdrawn.

These findings relate to the study by Kuipers *et al* (1983), who observed relatives and patients talking to an interviewer, and found that high-EE relatives spent 57% more time talking than low-EE relatives—a figure close to the 64% higher word count observed here. They also found that the amount of silence was significantly greater in low-EE pairs than in high-EE pairs. They did not find that differences in the patients' behaviour were related to EE, but this may have been because an interviewer was present—this could inhibit the development of a reciprocal interaction pattern between the other two.

Do low-EE relatives show more support than high-EE relatives? The above studies showed no differences, but this may have been because the discussions were emotionally charged, thus eliciting critical and intrusive statements. There is evidence that with more neutral issues, low-EE relatives show more positive behaviours such as approval, agreement, smiling and humour (Greenwald *et al*, cited in Strachan *et al*, 1986).

Together, these studies suggest that interactions with high-EE relatives can be characterised as lively and heated, with a great deal of excitability, criti-

cisms, intrusiveness, interruptions, and simultaneous speech. Low-EE interaction can be characterised as calmer, more affectively neutral, more positive and supportive, and less verbal, with more pauses.

These findings also pose intriguing questions about patients' responses to, or provocation of, the criticisms and intrusions of the high-EE relative. Is the patient's style of coping one of counter-attack, withdrawal, or more differentiated expressions of autonomy? Are there repeated patterns of patients' behaviour which precede relatives' criticisms or intrusions? While the present study did not examine such issues, the word counts suggest that patients are actively involved in interactions with high-EE relatives, rather than being withdrawn.

The finding that patients with high-EE relatives reported more depression and irritability suggests hypotheses about the cyclical nature of the interaction. For example, Vaughn (1986) reported that the majority of critical comments are about long-standing attributes of the patient, rather than about recent changes in behaviour. As the current study suggests, this criticism of characteristics of the patient is probably expressed over the years and may, if internalised by the patient, contribute to the depressive symptomatology. Alternatively, as Coyne (1976a,b) has argued, the behaviour of depressed individuals may elicit alternating messages of rejection and reassurance. It is possible that patients who make self-deprecating remarks or statements of hopelessness elicit or perpetuate relatives' criticism and intrusiveness.

Thus, both the expression of high-EE attitudes

and the patients' depression and irritability may interact in a cyclical pattern. Further examination of patients' behaviour using sequential analyses could help to explain processes which might maintain and perpetuate a cycle of mutual antagonism or which might lead to a de-escalation of tensions. Such exploration might help to explain why, despite the general congruency between attitudes and behaviour shown in this study, there are discrepancies.

In conclusion, the establishment of a basic congruence between emotional attitudes and behaviour shown by these data in a British sample confirms the findings of Miklowitz and Valone in American samples. Taken together, the reports provide cross-national replications which parallel those of the relationship between EE and relapse in both Britain and the USA (Vaughn & Leff, 1976a; Vaughn *et al*, 1984), thus building a solid body of evidence on the nature of EE and suggesting that there are comparable behavioural mediating mechanisms in direct behaviour in the two countries.

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Monitoring of Long-Term Motor Activity in Depressed Patients

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Wrist motor activity was continuously recorded by a solid-state monitor in 12 patients with major depression throughout their stay in hospital; their clinical state was evaluated using three scales. During the day, activity troughs and immobility peaks occurred before noon and around 1500 hrs. Activity level progressively increased, while the duration of immobility decreased, with clinical improvement. The immobility measurement appeared to be a valuable index of depression and is more sensitive than the activity level, mainly for agitated depressed patients. Immobility episodes during the day could suggest the release of an ultradian sleep-wake rhythm in depression.

Changes in motor behaviour accompanying depressive illness have long been recognised. Psychomotor retardation or agitation are part of the criteria used for the diagnosis of major depression, together with other symptoms including anorexia, insomnia, lack of interest, and guilt. A distinction has been made between agitated and retarded depressions, but this is usually dependent on the presence or absence of motor retardation and does not take into account psychic retardation. It has also been maintained that retardation is almost always a sign of major depressive illness, and the classical description of melancholia has retained marked psychomotor retardation, which in some extreme cases may mimic stupor.

A global clinical scale to assess retardation has been proposed by Widlöcher (1983a, b), and includes both mental and motor components. There have been attempts to measure retardation using specific techniques such as the "speech pause time" test for verbal flow (Hardy *et al*, 1984), and other authors have tried to measure the motor component by means of telemetry (Kupfer *et al*, 1974; Post *et al*, 1977), or by using a small activity monitor (Kripke *et al*, 1978; Wehr *et al*, 1980, 1982; Borbely, 1983).

The main purpose of our study was to evaluate the extent to which objectively recorded motor activity parameters are correlated with the clinical state, as assessed by rating scales, during pharmacological treatment of depression.

Method

Patients

Wrist motor activity was continuously monitored in 12 depressed patients during their stay in a psychiatric ward; seven were also recorded for an additional week at home,

after discharge. All depressive patients admitted between 15 April and 15 May 1983 were considered for the study. To be included, they had to give informed consent and agree to the requirements of the study (principally the continuous wearing of a wrist monitor). The two who did not consent to these conditions were excluded. The remaining patients (ten females, two males) were classified as major unipolar depressives, according to DSM-III (American Psychiatric Association, 1980); by the Newcastle criteria (Gurney, 1971), six were considered endogenous depressives, four non-endogenous, and two 'doubtful'. In one patient a manic period occurred several months after the end of the study; this patient had been a night worker one month prior to her admission. The mean age was 55 (range 40-76 years), and the mean duration of hospital stay 31 days (range 15-71). Prior to admission, five patients had received antidepressive treatment for several weeks; in eight cases this included benzodiazepines. During admission, all the subjects received specific antidepressive therapy (the tricyclic compounds, mianserine and clenbuterol, alone or in combination) as well as benzodiazepines.

The activity monitor

Motor activity was recorded by a solid-state motor activity monitor (Borbely *et al*, 1981) which was worn like a wrist-watch on the non-dominant arm; this is small (30 × 51 × 17 mm) and light-weight (87 g). Movements were recorded by a piezo-electric transducer, which generates a pulse whenever acceleration exceeds a preset threshold (0.1 g). The pulses were counted during a preset time interval (15 min), and their number stored in a memory (storage capacity 256 h). The power was provided by three 1.5 V batteries.

Clinical evaluation

During the recording period, the clinical state was evaluated on four occasions: at admission, before starting the antidepressive treatment (day 0) and after a one week washout of the previous antidepressive drugs; eight days

after the start of antidepressive treatment (day 8); on the day before discharge (day D); and after one week at home (day H). These clinical evaluations were based on two global depressive symptomatological scales—the Hamilton depressive rating scale (HAMD) (Hamilton 1967) and the Montgomery and Åsberg Depression Rating Scale (MADRS) (Montgomery and Åsberg, 1979)—and on the depressive retardation rating scale of Widlöcher (1983a, b). The sleep-wake schedule, the extent of nocturnal awakenings, and the occurrence of naps were estimated from the sleep diaries kept by the patients.

In addition, subjects were asked to rate their mood six times per day (every 3 hours) on a visual analogue scale, anchored by the words "I am not feeling sad" and "I am feeling sad". Four patients were unable to perform the self-rating (three among them were the most severely depressed, with HAMD scores of 31, 29, and 28).

Analysis of data

Clinical improvement was expressed relative to the score obtained on admission. The criteria for remission were an improvement resulting both in a decrease of 10 points or more on the HAMD scale and in a score under 10: these criteria correspond roughly to an improvement of at least 60% relative to the entrance score.

The level of motor activity was analysed by 15-minute periods; the mean of the 15-minute values was calculated for each 24-hour period. On the days of clinical evaluation the data were averaged with those of the preceding and the following days to obtain more stable values. The number of 15-minute immobility epochs was calculated separately both for the day and night together and for the night alone (0000–0060 hrs). The hourly distribution of levels of both activity and immobility were calculated for the days of clinical evaluation.

The data were analysed by means of one way analysis of variance. Spearman rank correlations between clinical scores and motor parameters were computed.

Results

Table I gives the clinical evolution of the patients as a group. There was a relationship between improvement and a progressive decrease in the three clinical scores. A 17-day recording of motor activity is illustrated in Fig. 1, showing the nocturnal awakenings and occasional naps. However, daytime immobility periods did not always correspond to naps. Although no sleep recording was performed, observation confirmed that some patients lay on their bed completely immobile, for minutes or even hours, without sleeping. At day 0, six of the patients had immobility periods in the morning, but only one subject reported having slept. In the afternoon eleven patients had immobility periods, with seven reporting a short episode of sleep.

Group results

The hourly distribution of activity parameters is illustrated in Fig. 2. At day 0 the activity curve was characterised by several troughs (<70 movements/h), which occurred late in the morning, in the early afternoon, and around 1800 hrs. At the end of the hospital stay (day D), the activity remained high throughout the day. These changes in activity were paralleled by modifications in the number of immobility epochs. The number of minutes/hour without movement during the night decreased from day 0 to day D. At day 0, the main diurnal peak of immobility was observed around 1500 hrs, with a secondary peak before noon. These immobility peaks did not correspond to any restraint due to hospital schedule or treatment commitments; they tended to disappear in each patient as the clinical state improved. At day 8, the activity level was intermediate between those of day 0 and day D, with a trough of activity and a peak of immobility still found at 1500 hrs.

We analysed the changes of motor activity parameters in relation to clinical improvement. The number of immobility epochs significantly decreased from day 0 to day D: this change was more significant for the night (day 0, 11.4 ± 4.6 ;

TABLE I
Mean values of clinical scores during hospital stay ($n=12$) and after one week at home ($n=7$)

Day	HAMD score		MADRS score		Retardation score	
	Mean	s.d.	Mean	s.d.	Mean	s.d.
0	21	6.5	27	9.4	20.3	10.6
8	14.7	9.3	17.3	11	17.1	10.9
D	6.1	5.1	6.6	5.3	8.9	4.2
H	6.4	5.5	8.4	7.9	10.1	7.1
Variance analysis						
F3, 39	11.4		12.5		4.26	
P	<0.001		<0.001		0.01	

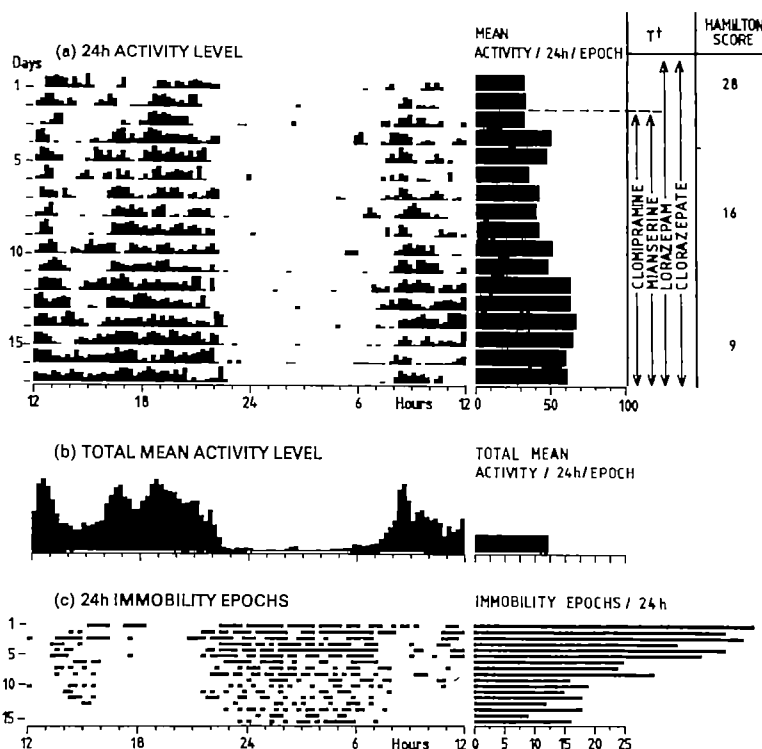


FIG 1. A 17-day recording of motor activity of an endogenous depressive 40 year old female. (a) On the left, each line illustrates the distribution of motor activity levels recorded every 15 minutes over a 24-hour period. On the right, each bar indicates the mean activity level by 15 minute epochs and by 24 hours. Treatment and clinical state estimated by the HAMD score are also given. (b) The mean level of the same parameter for the entire duration of hospitalization. (c) On the left are the immobility epochs during 24 hours; on the right, the total number of immobility epochs in each 24 hour period.

day 8, 9.1 ± 3.7 ; day D, 7.2 ± 2.8 ; $F_{2,33} = 5$, $P < 0.02$) than for the combined day and night period (day 0, 23 ± 10.4 ; day 8, 19 ± 6.5 ; day D, 14.5 ± 6.4 ; $F_{2,33} = 3.8$, $P < 0.05$). Although the 24-hour mean level of activity increased, this increase was not significant (day 0, 47.6 ± 20.5 ; day 8, 61.9 ± 22.9 ; day D, 64.3 ± 13.4). Although the relationship between activity level and immobility was not a reciprocal one, we found a significantly negative correlation between them (day 0, -0.66 , $P < 0.05$; day 8, -0.50 , $P < 0.05$; day D, 0.55 , $P < 0.05$).

In general no significant correlation was obtained between the clinical scales and individual levels of immobility or activity at day 0 and day D. At day 8, significant correlations were found: a positive one between retardation scores and 24 h immobility levels ($\rho = 0.58$, $P < 0.05$), and a negative one between retardation scores and activity levels ($\rho = 0.61$, $P < 0.05$). When improvement at day D was expressed as a percentage of the initial value of a given scale, only one significant positive correlation was found between

the improvement of the self-ratings of depression given on visual analogue scales and the 24-hour immobility epochs ($\rho = 0.64$, $P < 0.05$).

Individual cases

The number of immobility epochs appeared to be more closely related to the clinical state than the activity level. Fig. 3 illustrates three individual cases.

Patient A exhibited a mean level of activity which transiently increased as a result of the treatment (clenbuterol), but without a clear change in her depressive state, while the immobility index remained high. The immobility index started to decrease only when the clinical state had improved.

Patient B did not present true retardation. In his case, as well as in two other patients who had a retardation score at the limit of normal (< 14) on admission, clinical

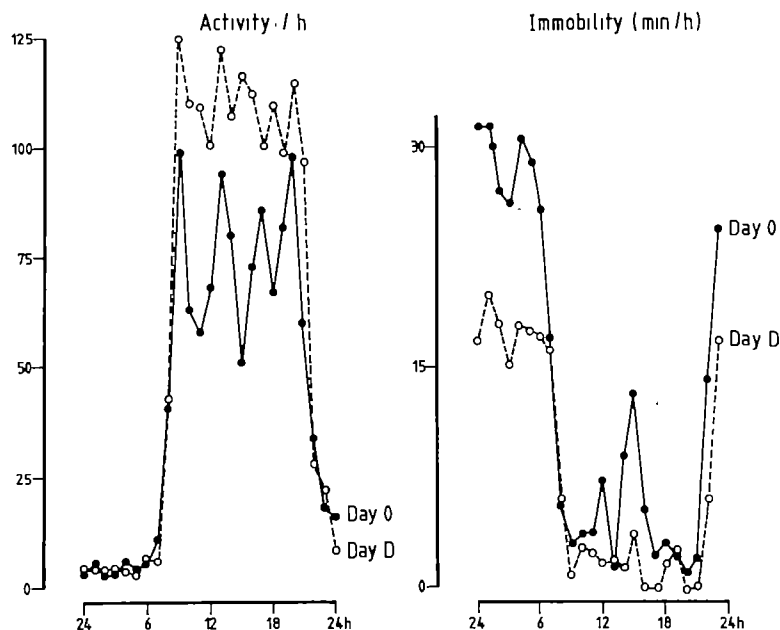


FIG 2. Hourly distribution of activity and immobility levels at the beginning of hospitalization (day 0) and at the end of hospitalization (day D).

improvement was reflected only by a decrease of immobility parameters, without changes in the activity levels

Two patients whose clinical improvement was insufficient to reach the criteria described above left hospital while still depressed. One of them (C) showed activity and immobility levels which had fluctuated from day to day without clearly increasing or decreasing trends.

The values of clinical scales obtained on the same days are given in Table I.

Discussion

The wrist activity monitor was readily accepted by the subjects, and is a good way to collect, in a relatively unobtrusive fashion, continuous longitudinal data for long periods in depressed patients. Wrist activity in unipolar depressed patients has rarely been monitored, and the results obtained previously with a similar device (Wehr *et al*, 1982) cannot be directly compared with the present data—mainly because of differences in characteristics of the respective patients. We found that motor activity in the depressed state was significantly lower than that found in the euthymic state in the same subjects; this is in agreement with several studies (Foster & Kupfer, 1975; Post *et al*, 1977; Wehr *et al*, 1980; Wolff *et al*, 1985). While a correlation between motor

activity and clinical state has been observed in 17 unipolar or bipolar depressed patients (Foster & Kupfer, 1975) as well as in one manic-depressive patient (Post *et al*, 1977), other authors (Kupfer *et al*, 1974) have failed to obtain a significant correlation. The latter authors reported that there were unimproved patients with high anxiety scores and high levels of activity, which could have been a possible cause of the lack of correlation. Absence of correlation between activity and self-report of mood is in agreement with the results of Godfrey & Knight (1984).

However, the results concerning the immobility index are more interesting; both for the entire 24 hours and for the night alone, they progressively decreased as the clinical state improved. This observation was surprising, since sleep disorders with nocturnal awakenings are frequent symptoms of depression. To our knowledge, this study underlines for the first time the importance of immobility as a function of the mood state, but the lack of correlation between immobility and HAMD score indicates that this immobility cannot be used as an absolute index of depression. However, in agreement with the significant correlation between individual feeling of improvement and improvement of immobility, this

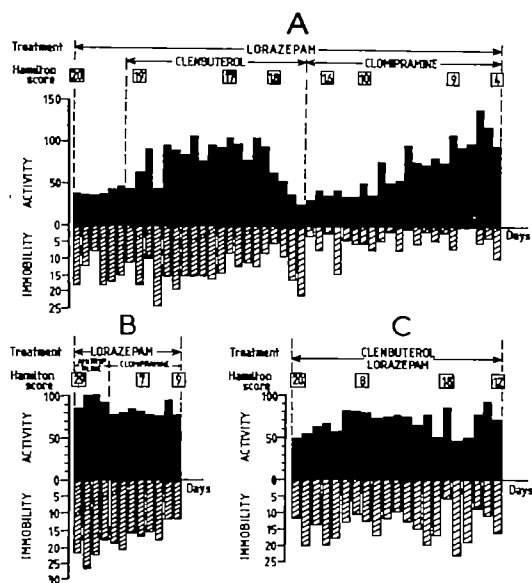


FIG. 3. Evolution of the activity parameters in three patients. The black bars show the mean activity level in a 15 minute period, averaged over 24 hours. The shaded bars show the number of immobility epochs in each 24 hour period. Patient A was a 48 year-old female (non-endogenous depression); patient B was a 76 year-old male (doubtful endogenous depression, anxious-agitated); patient C was a 51 year-old female (non-endogenous depression, non-responder).

immobility index can be used as a measurement of the evolution of depressive illness in a given patient. It could also be used to judge the efficacy of the treatment. Immobility therefore seems to be a better index of depression than the activity level; while motor activity may be reduced in depression, the lack of initiation of spontaneous movements appears to be a more fundamental deficiency, and is similarly observed in non-retarded patients.

The activity troughs and immobility peaks occurring before noon and around 1500 hrs were partly

related to naps. The circadian fluctuations of activity parameters in our patients have been described elsewhere (Benoit *et al*, 1985). Kupfer *et al* (1972) have noted a strong correlation between a low level of activity at night and sleep. In the first few days after admission our subjects reported frequent naps around 1500 hrs, but not in the morning. The episodic occurrence of immobility over the 24-hour period suggests the release of an ultradian sleep-wake rhythm in depression. An ultradian rhythm of prolactin secretion (Mendlewicz, 1983) and body temperature (Nikitopoulou & Crammer, 1976) have been reported in depressed patients.

The great number and long duration of immobility episodes observed in our patients raise the question of whether pharmacological treatment, particularly by benzodiazepines, may have been a cause of the immobility. Borbely *et al* (1983, 1984) have shown that a single hypnotic dose of a benzodiazepine taken before bedtime enhances night-time immobility. While the precise role of hypnotics in the present study cannot be reliably assessed, there are some indications that they did not exert a major influence. Although medication with benzodiazepines in eight of the patients had begun prior to admission, the level of immobility was high for all patients on day 0. It was thus felt that the continuation of this treatment did not prevent the decrease of immobility in relation to the clinical improvement. Furthermore, in the two patients who did not respond the immobility index did not change, although all patients received similar drug treatment. Even though medication does not appear to have been the major factor in inducing immobility, the problem needs to be further investigated; it is possible that frequent episodes without movement constitute a specific feature of depressive illness. If this is confirmed in further studies, immobility could be a valuable index of depression.

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Patients Who Break Their Fast Before ECT

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Of 81 patients receiving ECT during a period of 24 months, 21 ate or drank something, against medical advice, shortly before ECT was due. Compared with controls, the fast-breakers were less often married; were more likely to be detained under a section of the Mental Health Act; had an unfavourable view of ECT; and had often not given consent for ECT. Fast-breaking was more likely to occur after four or more ECTs. Amongst the fast-breakers there was a significant correlation between age and (a) number of previous ECTs; (b) treatment at which the fast-break occurred. The more drugs the patient was taking the more likely it was that the fast would be broken early in the course of ECT.

Little is known of the clinical characteristics of patients who refuse ECT or those who, while not overtly refusing, avoid receiving it by breaking their fast. One may speculate that the latter group may be constituted of at least two sub-groups: patients who forget that they must not eat and patients who wilfully eat as a form of passive resistance. However, this distinction is based on the ascertainment of motives and so is difficult to make in clinical practice.

This paper reports on a prospective study into the more objective clinical characteristics of a cohort of fast-breakers.

Method

The study was carried out in a 30-bed acute unit (Friends Ward) in Fulbourn Hospital, Cambridge. All patients receiving ECT during the period of 24 months starting on 13 July 1983 were included. There were 617 admissions during the period in question: 76 patients received ECT, of whom 21 broke their fast at some time.

Fast-breaking was defined as eating or drinking, against clear medical advice, during the 8 hours prior to ECT. Due to a ruling by the consultant anaesthetist in charge, such fast-breaking led automatically to a cancellation of that particular treatment. In emergency cases the patient was kept fasting from the time of the incident and the treatment was postponed until the afternoon.

Information was collected by means of specially formatted data sheets which were completed immediately after the incident. These requested data on the patient's age, sex, civil status and legal status under the Mental Health Act; whether he gave consent to ECT, the treatment at which the fast was broken; the patient's and family's attitude to ECT; the patient's previous experience with ECT, medication, and ICD-9 diagnosis; the psychosocial state of the ward; the number of nurses working on the particular shift; and the number of empty beds (an indirect index of overcrowding).

The fast-breakers ($n=21$) constituted the index sample. There were 55 other patients who received ECT and these constituted the control sample. Five control subjects

received ECT twice during the period in question: in each case only the first course was included in our analysis.

In the first stage of our analysis the index sample was considered separately and the correlations between all its variables were examined. In the second stage the index and control samples were compared on those variables for which they were not matched. Statistical analysis was carried out by means of the Minitab Statistical Package (available in the Computer Laboratory of the University of Cambridge).

Results

There was a predominance of females amongst the fast-breakers (18 out of 21) but this reflected a bias in the hospital population. Significant correlations (Pearson correlation coefficient) were found in the index sample between age and (a) the number of previous ECTs ($r=0.43$; $P<0.02$); (b) the ECT at which the fast was broken ($r=-0.51$; $P<0.008$); and (c) the drug score ($r=-0.53$; $P<0.006$). There was a tendency for the fast to be broken after four or more ECTs, and the ECT at which the fast was broken was significantly correlated with the drug score ($r=-0.61$; $P<0.01$). No other correlations were found between any of the following variables: number of previous courses of ECT; the number of nurses on the ward; the number of empty beds; drug score; psychosocial climate of the ward.

The index and the control groups were not different in terms of age, sex, diagnosis, drug score, number of previous ECTs or family attitude towards ECT (Table I). There were however some statistically significant differences: the fast-breakers included more subjects who (a) were divorced, single or widowed; (b) were detained under a section of the Mental Health Act; (c) held an unfavourable view of ECT; and (d) had not given consent for ECT.

Discussion

The identification of potential fast-breakers is of some clinical importance. This form of behaviour interrupts treatment; it may prolong the hospital

TABLE I
Comparison of patients who broke their fast before ECT (index sample) with a control group

	Index sample (n = 21)	Controls (n = 55)	Statistic	Significance
Age (years): mean, s.d.	47.7; s.d. 19.7	46.9; s.d. 15.8	$t = 0.159$	NS
Sex	3 M/18 F	21 M/34 F	$\chi^2 = 2.98$	NS
Civil status: married/other	8/13	39/16	$\chi^2 = 5.61$	$P < 0.05$
Legal status: informal admission/MHA	11/10	45/10	$\chi^2 = 6.58$	$P < 0.01$
Consent for ECT	12 Yes/9 No	48 Yes/7 No	$\chi^2 = 6.59$	$P < 0.01$
Diagnosis (ICD9)				
Manic-depressive depressed (296.1)	14	39	$\chi^2 = 0.31$	NS
Manic-depressive, manic (296)	4	8		
Schizophrenic psychosis (295)	2	6		
Schizoaffective (291.1)	1	2		
Attitude to ECT (favourable/neutral/unfavourable)				
Family	11/9/1	24/28/3	$\chi^2 = 0.46$	NS
Patient	7/6/8	12/35/8	$\chi^2 = 8.27$	$P < 0.01$
Drug score: mean, s.d.	2.52; s.d. 1.36	2.60; s.d. 1.57	$t = -0.209$	NS
Previous courses of ECT: mean, s.d.	0.85; s.d. 1.06	0.76; s.d. 0.94	$t = 0.35$	NS
Duration of admission (days): mean, s.d.	40.6; s.d. 25	35.8; s.d. 21	$t = 0.78$	NS

stay and so increase costs; and it may damage the relationship between the patient and the staff.

This paper has identified a cluster of features which may help the clinician to identify such patients. The fast-breaker can be male or female and of any age but is likely to be single, divorced or widowed rather than married; to be detained under a section of the Mental Health Act; not to have given consent to ECT (even if able to do so); and to hold unfavourable views on ECT. The fast tends to be broken after four or more ECTs. Confusion, memory impairment and the total amount of medication taken by the patient while receiving ECT do not seem to be important factors. Indeed, in our study only one patient claimed to have forgotten the instruction.

Tea- and coffee-drinking were the most common method of fast-breaking and the action tended to occur within the three hours prior to ECT. The subjects were very rarely willing to provide an account of their motives.

Our initial hypothesis was that environmental factors such as the psychosocial state of the ward, the number of nurses working on the particular shift, and the number of empty beds might be important. This was not borne out by our findings.

The fact that there were more fast-breakers than controls detained under a section of the MHA sug-

gests that the condition of the fast-breakers was more severe, or their behaviour more disordered. In fact there was a tendency for fast-breakers to stay in hospital longer than controls, but the difference did not reach statistical significance. There was no difference in outcome between the two groups, nor did the index sample receive more psychotropic medication than the controls. Since we did not include specific measures of severity, this issue remains unresolved.

We conclude that wilful fast-breaking was more common than forgetfulness or confusion in our sample. It is hypothesised that this represents some form of passive resistance which may be related to increased experience of side-effects as the course of ECT progresses, since fast-breaking tends to occur during the later stages of the course. The more drugs the patient was taking the earlier he tended to break his/her fast, which suggests that medication may amplify any unpleasant side-effects of ECT. Another hypothesis is that ECT produces increased arousal and psychomotor reactivation, leading to disinhibition and increased self-assertion.

It is recommended that patients meeting the criteria mentioned above are subject to special observation and given special counselling before and during any course of ECT.

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Psychosocial Effects of Mastectomy: Is it Due to Mastectomy or to the Diagnosis of Malignancy?

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Twenty-three consecutive married and sexually active Chinese women treated for breast cancer were compared with a randomly selected group of 34 female patients suffering from different kinds of malignancies. A structured interview was used to study both groups. The breast cancer group were found to be less depressed and more emotionally stable than those suffering from other malignancies, suggesting that the diagnosis of malignancy was the more important factor in the psychosocial morbidity of these patients.

Many clinical studies have shown that mastectomy for breast cancer is associated with considerable emotional trauma. These studies have usually centred around the immediate pre- and post-operative periods of their treatment, and there have been few long term follow-up studies (Morris *et al*, 1977, two years; Craig *et al*, 1974, five years) of patients surviving breast cancer. Furthermore, those studies were carried out on women living in a Western society with cultural backgrounds very different from those of Oriental women. The aims of the present study were to ascertain

- (a) the long-term psychosocial sequelae of mastectomy
- (b) whether these sequelae in Oriental women were any different from those observed in women living in Western society
- (c) whether the emotional morbidity of women undergoing mastectomy was any different from that of survivors of other cancers.

Method

Over a period of six months, 23 consecutive married Chinese females who were long term survivors of breast cancer were recruited for the study. They were compared with a control group of 34 Chinese female patients who were long-term survivors of other types of cancer: thyroid ($n=10$), stomach ($n=6$), colon and rectum ($n=13$), liver ($n=2$) and oesophagus ($n=3$). The breast cancer patients were all under the care of one surgeon; those in the control group were patients undergoing treatment in the same institution but under the care of different surgeons. Both groups were comparable as regards age, time since commencement of treatment, and the different modalities of treatment received (Table I). None of the patients in the control group had any major postoperative complications which could have prolonged their stay in hospital. Patients with rectal cancer were those who underwent sphincter-conserving resections of the rectum, they did not have open perineal

wounds or colostomies. Patients with breast cancer were those who had operable tumours ($T_{1,2}N_{0,1}M_0$); they underwent mastectomy and subsequently received adjuvant radiotherapy, chemotherapy, or both. None of these patients underwent any form of breast reconstruction. They all wore padded brassieres to conceal their deformity; none of the breast cancer patients had lymphoedema of the arm. Patients in both groups were fully aware of the natural outcome of their respective illnesses; no patient in either group had a past history of mental illness or had sought psychiatric treatment before the onset of their present illness. No formal preoperative psychiatric assessment was made in any of these patients.

A structured interview was used to compare the groups; and responses were graded as mild, moderate or severe. All patients were interviewed by a single assessor. Particular attention was direct to

- (a) assessment of depression by the Beck Depression Inventory (DI); cut-off score of 12 (which we have found from our experience with the local population to be valid) was used in the assessment of patients. Inquiries were also made regarding addiction to alcohol and excessive use of tranquilizers and sedatives
- (b) changes in sexual activity and any special sexual problems that patients experienced
- (c) changes in interpersonal relationships within the family and among friends and neighbours.

In addition, both groups were tested with the Eysenck Personality Inventory (EPI) for neuroticism. Chi-squared analysis was used to detect any significant differences in the data between the two groups.

Results

All women who were approached agreed to participate in the study, and co-operated fully in answering the detailed questions put to them. The majority of the patients studied were able to cope with the domestic tasks of housework, washing, cooking and shopping, and there were no significant differences in this respect between the two groups.

TABLE I
Clinical data of sample and control groups

	<i>Breast cancer (n = 23)</i>	<i>Other cancers (n = 34)</i>
Mean age: years	45.2 (s.d. 11.4)	41.8 (s.d. 9.7)
Mean time since start of treatment: years	6.9 (s.d. 1.8)	6.5 (s.d. 3.0)
Treatment received		
Surgery alone	5	15
Surgery and drugs	5	8
Surgery, radiotherapy and drugs	13	11

TABLE II
Depression and loss of femininity

	<i>Breast cancer</i>	<i>Other cancers</i>
Depression	11	21
Reasons for depression		
No reason	5	7
Financial matters	1	4
Diagnosis of malignancy	3	6
Changes in interpersonal relationships	2	4
Loss of femininity and changes in maternal role	5	6
Beck score	12.6 (s.d. 10.4)	20.8 (s.d. 8.9)

Depression was found in 47% of the breast cancer patients and 61% of the control group. On further questioning, well over half of these patients felt that fears arising from a diagnosis of malignancy, anxiety over financial matters and the loss of a close relationship were the causes of their depression. None of the breast cancer patients mentioned mastectomy as a cause of their depression. When specifically asked about feelings of loss of femininity or a sense of unworthiness in continuing the maternal role in the family, only 23% of the breast cancer and 17% of the control group reported such feeling. The breast cancer patients had a significantly lower DI score ($P < 0.01$) than those in the control group (Table II). In neither group was there any instance of increase alcohol consumption or addiction; nor were there any patients who were excessively dependent on tranquilizers and sedatives.

Only three (14%) of the breast cancer patients and nine (26%) of the control group felt that their husbands were less affectionate or were involved in extramarital affairs. Both groups experienced a decrease in frequency of sexual intercourse (moderate or marked decrease: breast cancer group 50%, controls 35%) and sexual satisfaction (moderate or

severe: breast cancer group 61%, controls 32%, $P < 0.05$) when compared with their pre-illness levels.

In the sphere of family and social life, there were many differences. Women in the control group experienced greater difficulty in adjusting to family life; there were greater parent-child tensions and more numerous instances of conflict with children (56%) than in the breast cancer group (34%). A similar finding was recorded in relationships with friends and social acquaintances; the control group (62%) found greater difficulty in social adjustment than women with breast cancer (14%). Furthermore, a greater number (56%) of women in the control group had suffered the loss of a close personal relationship over the past five years than those suffering from breast cancer (39%), but the difference was not statistically significant.

There was a significantly lower ($P < 0.01$) EPI neuroticism score in the breast cancer patients.

Discussion

The results show that even several years after the diagnosis and treatment of breast cancer, there still

remain a proportion of patients who never recover from the psychosocial morbidity of their illness. Of breast cancer patients, 47% were found to be depressed at the end of a mean period of seven years. This figure is much higher than that quoted in similar studies on Western women (Morris *et al*, 1977: 20% at two years; Craig *et al*, 1974: 20% at five years). It is unlikely that this high figure is due to the longer interval between completion of treatment and assessment in the present study; Morris *et al* (1977) showed that there is no great difference in the proportion of women remaining depressed at one year, and at two years following mastectomy.

A further point of interest lay in the reasons elicited for the depression. Loss of femininity and physical disfigurement did not appear to be the most important reasons for depression. Rather, fears arising from a diagnosis of malignancy, with its implications of incurability, recurrent disease and premature death, worries over family income, and fears regarding social acceptability appeared to be more important factors. This finding is reinforced by the fact that women in the control group (none of whom had undergone mastectomy) fared worse. It is unlikely that this difference in the two groups could be attributed to differences in the modes of treatment received. Lyon (1977) had observed that radiotherapy is an important factor in post-mastectomy depression, and one would have expected, considering the larger number of breast cancer patients receiving radiotherapy, to observe a greater degree of depression in these patients than in the control group. It would appear that the emotional trauma of mastectomy plays a secondary role in the aetiology of depression, and it is more than likely it has a similar role in the overall psychosocial morbidity of patients with breast cancer. Further evidence for this view comes from the studies of Sanger & Reznikoff (1981), who compared patients undergoing mastectomy with those having the more recent breast-conserving surgical procedures for breast cancer. No significant differences could be found in body anxiety, general psychological adjustment, and marital satisfaction between these groups.

Maguire *et al* (1978) and Morris *et al* (1977) showed that 18% of patients undergoing mastectomy experienced moderate or severe sexual difficulties three months after treatment; at two years following treatment this figure had increased to 32%. It was further observed (Morris *et al*, 1977) that patients who deteriorated within the first three months after surgery were unlikely ever to return to their preexisting levels of adjustment. The high figures recorded in the present study for sexual difficulties (as evidenced by a decrease in frequency of

sexual intercourse and sexual satisfaction) experienced by both groups of patients appeared to support this view. These difficulties had occurred despite the fact that the majority of women had received the understanding, care, and emotional support of their husband. Only 4% of breast cancer patients and 15% of the control group felt that their husbands were less affectionate than before their illness. Further study is necessary to ascertain the factors involved in the deterioration in sexual relationships with time. Deterioration in physical health was certainly not a factor, as the majority of women in both groups were able to cope with their daily household chores. It is more likely that the conservative attitudes of Asian women to sex, and their natural reluctance to talk openly on sexual matters, were more important factors in their sexual maladjustment. None of the women had ever sought help from marriage counsellors, psychotherapists or even their general practitioners. A noteworthy finding was that there was no significant difference between the groups in the number of women having sexual difficulties; which again would suggest that the emotional sequelae of mastectomy may not be the most important factor in the sexual morbidity of breast cancer patients.

The study by Morris *et al* (1977) has shown that post-mastectomy women may also have difficulties in making social adjustments. However, these difficulties lessened with time. It was surprising, therefore, to note in the present study that even after a mean period of seven years following treatment, there still remained women in both groups who had difficulty in making adjustments in family and social life. However, it was women in the control group who had experienced significantly greater difficulties in adjustment, with 56% of them admitting to have lost a close personal relationship over the preceding five years.

Perhaps the most important finding in the present study is that post-mastectomy patients were at no greater risk of developing psychosocial morbidity than patients with other malignancies. It may be argued that the long interval of time between treatment and assessment permitted the breast cancer patients to adjust themselves more easily than those with other malignancies; this may be especially relevant as mastectomy in Oriental women may not cause the same degree of emotional distress it does in Western women. The breast, in an Asian environment, does not have the same sexual symbolism it has in the West. However, it is unlikely that these are valid reasons for the differences seen in the two groups. Worden & Weisman (1977) have shown, in a similar study of Western women, that post-mastectomy women were no worse off than women

suffering from Hodgkin's disease, malignant melanoma and cancer of the colon. A more likely explanation is that the diagnosis of malignancy (which was common to both groups) was a powerful factor in the psychosocial morbidity of these patients and that this overshadowed the emotional trauma of mastectomy in patients with breast cancer. It would appear from the present study that women with malignancies other than breast cancer could also develop serious psychosocial problems, and in many respects the need for therapy could be greater in these women than in women with breast cancer.

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Inter-generational Conflict and Psychiatric Symptoms

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Secondary school pupils and their parents were investigated using the scaled version of the General Health Questionnaire (GHQ-28) and by a questionnaire designed to study attitudes involved in inter-generational conflict in psychiatric patients. Parent-pupil and interparental conflict in answers to the attitude questionnaires were taken as measures of inter-generational and intra-generational conflicts respectively. The former significantly exceeded the latter. Parent-student conflict was higher when the students involved were females, Kuwaiti, or had less educated fathers. The tendency of the number of reported GHQ symptoms to be higher in members of families with higher inter-generational conflict did not reach statistical significance. There is an apparent discrepancy between this finding and the prominence of inter-generational conflict in clinical material.

Inter-generational conflict has been known since the days of Socrates (Ness, 1959), although Mead (1970) stated that 'the breakdown between generations is wholly new: it is planetary and universal'. Western literature on inter-generational differences or the 'generation gap' reports a spectrum of findings. These extend from inevitability (Davis, 1940) and dramatised descriptions of active warring conflicts or attempts of the young to exist in the present without any linkage to the past (Michael, 1965; Feuer, 1969) at one extreme to denial of the existence of a generation gap (Lerner & Weinstock, 1972; Lauer, 1973; Barclay & Sharp, 1982) at the other.

Reports on inter-generational conflict from the Arab world relate it to several factors: parental attempts to direct children in accordance with parents' aspirations rather than their own interests (Hamza, 1959); exposure to Western influences (Nagati, 1963); adoption of more progressive values by the young (Sultan *et al*, 1972); rigidity of parental control (Torky, 1974); or loosening of parental control (Hassan, 1977). The conflict centres mainly around patterns of relationships in the Arabian family, methods of marriage, and the emancipation of women (El-Islam, 1983). Clinical research in the Arabian Gulf area revealed that inter-generational conflict was involved in the presentations of 57% of cases of attempted suicide, 20% of neurotic and depressive illnesses, and 17% of schizophrenic illnesses (El-Islam, 1974, 1976, 1979; El-Islam *et al*, 1983). Alcohol consumption to the extent of dependence may be motivated by anti-parental and anti-cultural attitudes of some youths in Gulf countries where the Islamic code prohibits the use of alcohol (Demerdash *et al*, 1981).

The present study examines the relationships

between inter-generational conflict, psychiatric symptoms, and some demographic variables of members of two generations in Kuwait, where major socio-economic and cultural changes have taken place since the discovery of oil, e.g. expansion of urban areas and shrinkage of Bedouin (nomadic) life. It was hypothesised that conflict differs according to nationality, sex, and upbringing (urban versus Bedouin) and that age and educational differences between parents and students correlate positively with conflict. The association of inter-generational conflict with case presentation in clinical material led to the hypothesis that greater conflict is likely to be associated with more reported symptoms.

Method

A stratified sample of secondary school pupils was obtained. One district was randomly selected from each province, and the schools in each selected district stratified according to sex; two government schools (one for males and one for females) were randomly drawn from each district. Age was controlled by including only third-year pupils (11th standard) in secondary schools (age range 15–19). The number of pupils drawn from each school was in proportion to the population density in the school's province. Selection procedures were based on a full directory of schools and pupils, provided by the Department of School Social Services. To give unbiased estimates, every unit in the final stage of sampling had a chance of being drawn.

Eight schools (four for girls and four for boys) were selected. The proportions of Kuwaiti pupils were in the region of 40%–60% in each school; non-Kuwaiti pupils in government schools are Mediterranean Arabs whose fathers have been employed for long periods in Kuwait. Third-year rather than final (fourth-year) pupils were selected because the latter are likely to be anxious on

account of the decisive nature of the final year in relation to future university education and career prospects.

The age and nationality of pupils and their parents were recorded. The level of education of parents was determined according to the highest level reached at school, and an education score given: 0=illiterate; 1=standards 1-4, 2=standards 5-8; 3=standards 9-12; and 4=higher standards. Since the pupils were in standard 11, their education score was 3. The education gap is the difference in education scores between family members, being negative if the parent's score exceeded 3. The locality where pupils spent their first ten years of life was recorded as Kuwait city (urban), Kuwaiti Bedouin (nomadic) areas, or outside the country of Kuwait.

For this investigation, inter-generational conflict was defined as disagreements between pupils and their parents (Jacobsen *et al.*, 1975). The former often seem to reject traditional attitudes to patterns of family relationships, methods of marriage, and the emancipation of women; these three areas were chosen for study on the basis of their prominence in clinical material. Such disagreement carries the potential for strife, although it is not necessarily associated with overt clashes as an expression of the ideational conflict.

An attitude questionnaire was designed for this study, consisting of 36 statements about attitudes to family relationships, methods of marriage, and the emancipation of women. (Both Arabic and English versions of the questionnaire are available from Professor El-Islam on application). Statements were derived from accounts of young patients whose illness involved conflict in these areas, from their parents, and from volunteers (mostly healthy relatives of patients), who were asked to describe current attitudes of members of both generations in these areas. Sixteen statements centred around family relationships (area 1), twelve around methods of marriage (area 2), and eight around the emancipation of women (area 3). Half the statements in each area expressed the traditional attitude, while the other half conveyed the non-traditional attitude, their order was randomised. Within each area traditional and non-traditional scores derived equally from agreement and disagreement answers to the statements provided.

Pupils were only included if they had lived with both genetic parents since childhood. Linear one-to-one parent-child differences, rather than differences between a parents' group and a children's group, were sought. The questionnaire was administered directly to parents and pupils, unlike the studies of Al-Aasser (1978) and of Melikian (1981), who relied on members of the younger generation to obtain information about their parents' value-attitude system as perceived by the youths themselves. For every pupil and parent, a score for non-traditional (liberal) answers was obtained in each area (maximums of 16, 12 and eight in areas one, two and three respectively). Conflicts in answers to statements were counted, and scores obtained for father-pupil, mother-pupil and father-mother attitude conflict in the areas examined.

To detect the presence of psychiatric symptoms in pupils and their parents, we produced an Arabic form of Goldberg & Hillier's (1979) scaled version of the General Health Questionnaire (GHQ-28). It was found necessary to modify

the form and style of the English test before it could be applied to Arabs; for example, a less condensed layout of alternatives facilitated the choice of answers, and items which did not translate well had to be rephrased to convey the meaning in common Arabic methods of parlance. GHQ-28 has four seven-item scales: somatic symptoms, anxiety and insomnia, social dysfunction, and severe depression. The answer to each question was chosen from four alternatives, related to the present state of health. For example, when enquiring about a symptom 'S', alternatives from which a candidate chose an answer would be: A. I never experienced S; B. My experience of S is no more than usual; C. My experience of S is a little more than usual; D. My experience of S is definitely more than usual. The scoring method 0, 0, 1, 1 was used for the A, B, C, and D alternatives respectively; the range of possible scores therefore extended from 0 to 28.

Both questionnaires (Attitude and GHQ-28) were administered at the same time by school social workers, who received prior coaching in the method of administration and in explanation of instructions and statements in case they should be asked to clarify them by pupils or parents. For illiterate parents, the social worker had to read the instructions and statements, and mark the answers on the questionnaire sheets.

Non-parametric methods were used; Kruskal-Wallis one-way analysis of variance by rank was used to test whether more than two groups derived from the same population. This test is more useful than extension of the median test, because it utilises more of the observed data. Pairwise comparisons of medians were made using the Mann-Whitney test; since it considers the rank value of each observation this test is more useful in deciding on rejection of the null hypothesis than the 'median' test, which depends on the location of observations in relation to the combined median.

Results

Initially, 479 pupils were asked to participate—equivalent to 5% of all 11th standard students in government schools—but only 414 agreed to have their parents involved. They were studied during the period December 1982 to February 1983. Thirty-three pupils living in one-parent families (30 Kuwaiti and three non-Kuwaiti) were excluded; the missing parent was often the dead father. In the remaining 381 subjects the nationality distribution was similar among both sexes: 123 male and 100 female Kuwaitis, 88 male and 70 female non-Kuwaitis.

Pupils had significantly higher median liberal attitude scores than parents, the difference between parents was significant only in the area of emancipation of women (Table 1). Examination of the median number of differences (i.e. the degree of attitude conflict) between pupils and fathers, pupils and mothers, and between the parents themselves in responses to the attitude questionnaire revealed significant differences between father-mother attitude conflict and both father-pupil and mother-pupil attitude conflicts. Differences between father-pupil and mother-pupil attitude conflicts were not significant for either male or female pupils.

TABLE I
*Liberal attitudes questionnaire. median scores for 381 families**

<i>Attitude area</i>	<i>No. of questions</i>	<i>Fathers</i>	<i>Pupils</i>	<i>Mothers</i>
1 Family relationships	16	6.4	7.4	6.4
2. Marriage	12	6.7	7.8	6.8
3 Women's role	8	3.1	3.9	3.5
Total	36	16.4	19.4	16.8

* $P < 0.001$ for differences between fathers, mothers and pupils in all areas, between fathers and pupils in all areas, and between mothers and pupils in areas 1 and 2.
 $P < 0.005$ between mothers and pupils in area 3.

TABLE II
Liberal attitudes, attitude conflicts and GHQ median scores: differences between male and female pupils

	<i>Male (n = 211)</i>	<i>Female (n = 170)</i>	<i>P</i>
Liberal Attitudes			
Area 1	7.5	7.4	NS
Area 2	7.7	8.0	0.007
Area 3	3.2	4.7	<0.001
Total	18.5	20.2	<0.001
Attitude Conflict with father			
Area 1	4.0	4.4	NS
Area 2	3.4	4.9	<0.001
Area 3	2.9	3.3	<0.001
Total	10.5	12.8	<0.001
Attitude Conflict with mother			
Area 1	4.0	4.4	NS
Area 2	3.2	4.4	<0.001
Area 3	2.5	3.2	0.002
Total	10.5	12.2	<0.001
GHQ Total	6.0	8.4	<0.001

Pupils reported more symptoms on GHQ (median 7.2) than fathers (median 3.0) and mothers (median 3.5); the differences between these groups were statistically significant ($P < 0.001$). Pairwise differences between pupils and either parent were statistically significant ($P < 0.001$), but the difference between the parents was not.

Differences between male and female pupils

When male and female pupils were compared, significant differences were found in both Kuwaitis and non-Kuwaitis

TABLE III
Liberal attitudes: differences in scores between Kuwaiti and non-Kuwaiti families

	<i>Kuwaiti families (n = 223)</i>	<i>Non-Kuwaiti families (n = 158)</i>	<i>P</i>
Father	15.4	17.5	<0.001
Mother	15.7	18.2	<0.001
Pupil	18.9	20.0	<0.002

in their attitudes, in their parent-pupil attitude conflict, and in their responses to GHQ (Table II). Female pupils in general had significantly more liberal attitudes than males, and significantly more parent-pupil attitude conflict in areas two and three. GHQ-reported symptoms among female pupils significantly exceeded those in males.

Differences related to nationality and childhood upbringing

Non-Kuwaiti pupils of both sexes were significantly more liberal than their Kuwaiti counterparts (Table III). Only 19 pupils spent their childhood outside Kuwait; they were excluded from this analysis. Pupils brought up in Kuwait city (urban area) were compared with those brought up in Bedouin (nomadic) areas outside the city; they did not differ significantly from each other in their median liberal attitude scores.

Age and education gaps

The ages of pupils ranged from 15–19 years (mean = 16.3). Pupils with father-student age gaps of less than 30 years were compared with those with age gaps of 30 years or more. Younger parents, especially fathers, tended to have higher median scores on liberal attitudes, but their parent-pupil conflict was not significantly different from that of older parents.

Compared with the less educated fathers, those with equal

TABLE IV
Median GHQ scores for above-median and below-median scorers on attitude conflict

Group	n	Median GHQ scores		
		Fathers	Mothers	Pupils
Father-pupil attitude conflict				
Above median	198	3	4	8
Below median	183	3	3	7
Mother-pupil attitude conflict				
Above median	174	3	4	8
Below median	207	3	3	7
Father-mother attitude conflict				
Above median	200	4*	4**	7
Below median	181	2*	2**	7

* $P=0.003$

** $P=0.014$

or higher education (education gap ≤ 0) had significantly higher non-traditional scores and tended to have lower parent-pupil attitude conflict scores in their families. Mothers were all younger and, with the exception of three Kuwaitis, less educated than fathers. Mothers whose education was less than their children's were significantly less liberal and had significantly more conflict with their children.

Differences in GHQ in relation to attitude conflict

We compared the GHQ scores of pupils with attitude conflict scores above and below the median. Higher conflicts tended to be consistently associated with higher GHQ symptom scores in all family members; the increase in symptoms with increase in conflict was statistically significant only in parents in relation to their own attitude conflict (Table IV). On the other hand, GHQ scores for subjects with liberal scores on the attitude questionnaire above and below the median did not show any consistent trends. Pupils who failed their final examinations in May/June 1983 (73) were examined as a separate group; their scores on the various parameters, including GHQ, did not differ from those of the sample population as a whole.

Discussion

The finding of significant differences between parent-pupil and father-mother attitude conflicts confirms that the idea of parent-pupil conflict has some basis in fact. That female pupils have more liberal attitudes than males seems to be a reaction to their socio-culturally disadvantaged position (El-Islam, 1982), for which non-traditional attitudes seem to offer a remedy. Hence, the differences in liberal attitude scores between male and female pupils are most prominent in the area of emancipation of women.

Our estimate of parent-pupil conflict, which derived from differences in each parent-child unit, is quite revealing in contrast with studies that looked for differences between pooled attitudes of parents and of pupil cohorts, where pooling masked linear differences and no generation gap was found (e.g. Freeman, 1972). The findings of Thurnher *et al* (1974) that women are less likely than men to conflict with those adopting different values and attitudes cannot be exemplified by our finding that female pupils have more conflict with their parents than males in the studied attitudes. Same-sex parent-child conflict did not differ significantly from different-sex parent-child conflict; this finding contrasts with the conclusion of Aldous & Hill (1965) that more inter-generational continuity exists in same-sex family lineages, and with the contrary findings of Jacobsen *et al* (1975) that more differences are encountered between same-sex parents and their children.

Greater attitude conflicts exist in Kuwaiti families at both the inter-generational and parental intra-generational levels. The roles of coexisting indigenous and imported value orientations are important in this respect. Non-Kuwaiti Arabs in Kuwait come from more Westernised Arab countries; they bring to Kuwait not only their expertise, but also their attitudes and life styles which are at variance with local tradition (Melikian, 1981). Moreover, the wealth of Kuwaitis following the discovery of oil has made it possible for them not only to obtain Western commodities, but also to be exposed to European and American attitudes and values, through the mass media and tourism.

The failure to find significant differences in liberal

attitudes between pupils with urban or Bedouin upbringing may point to the greater importance of the common current social conditions and social institutions in shaping attitudes. However, inter-generational conflicts in attitudes were greater in families of pupils who had also been exposed to greater Westernisation during their Kuwait city upbringing, as compared with families of students with Bedouin (nomadic) childhood. The difference was significant only in mother-pupil conflict, possibly because of the poorer education of mothers.

The scope of liberal attitudes adopted by our pupils was generally similar to that described by Mehryar & Tashakkori (1978) in Iranian students who had a similar educational standard: they rebel against traditional parental authority, arranged marriage systems, and the restriction of the role of women to marriage and mothering. The challenge involves attitudes related to methods of marriage and to the emancipation of women more than those related to the area of family relationships. Conflict in the latter area accounted for a relatively small portion of the total, although it was probed by 16 of the 36 items in the attitude questionnaire. There may be a core of inter-generationally agreed family relationships that forms a 'point of strength' in the Gulf family (Melikian, 1981).

Since older parents are likely to be also less educated, disentanglement of the correlates of parental age and education may be difficult (Meidan, 1981). Barakat (1960) and Al-Eassa (1979), in their studies of other Gulf communities, pointed to the excess of non-traditional attitudes among the educated, but did not study the conflicts arising therefrom. Since Kuwaiti men are more likely to remarry at an older age than non-Kuwaiti men (El-Islam, 1982, 1983), they tend to have larger father-pupil age gaps. Non-Kuwaiti men whose children are admissible to the government schools which were studied are mostly professionals or technicians, i.e. they are relatively better educated than Kuwaiti fathers, and hence educationally closer to their children.

The tendency to have a greater number of GHQ symptoms in association with above-median attitude conflicts and the absence of such a relationship between GHQ symptoms and liberal attitudes suggest that it is conflict of attitudes, rather than the adoption of non-traditional attitudes as such, that is likely to be associated with symptoms. Inkeles & Smith (1974), rather than discovering adversity, found better psychological adjustment in association with modernisation of social attitudes. What is important for the development of clinical symptoms seems to be the perceived inadequacy of supportive relationships in the face of stress (Henderson, 1981).

Since the area of family relationships is the least conflicting in the families studied, family relationships may absorb the tension of disagreements in other areas (Lowy, 1983), e.g. methods of marriage and emancipation of women.

We did not assess the mental health of pupils and parents by psychiatric interviews. Therefore, it is not implied that the observed attitude conflict and reported GHQ symptoms are necessarily indications of poor mental health. Moreover, the similarity of attitude conflict and GHQ scores in pupils who failed and those who passed their final school year examinations does not support a case for inter-dependence of attitude conflict, GHQ symptoms, and scholastic achievement. Kim *et al* (1983), however, attributed both symptoms and poor school achievement, at least partly, to a decline in traditional attitudes among adolescent students.

How can the frequent encounter of inter-generational conflict in clinical psychiatric practice be reconciled with the much less noticeable and statistically insignificant association between inter-generational conflict and GHQ-reported symptoms in this community sample? Masterson (1967) found that feelings of anxiety and depression were as common in adolescents who have never had treatment for psychiatric disorder as in a sample of adolescent patients. In their investigation of the generation gap, Rutter *et al* (1976) found that a large proportion of the adolescents examined by questionnaire reported insomnia and feelings of depression, yet their psychiatric examination often failed to detect any psychiatric disorder. They differentiated the common adolescent experience of turmoil associated with parent-adolescent 'alienation' from psychiatric disorder that associates with parent-adolescent 'stresses'. Our adolescents' feelings of turmoil were detected by the GHQ, although probably they were neither spontaneously reported as complaints nor noticed by adult family members. Only in a minority of adolescents does inter-generational conflict make it difficult for the family to cope with adolescent problems using its intrinsic resources; in these cases the conflict is instrumental in provoking a search for psychiatric help.

The clinical psychiatric significance of inter-generational conflict is therefore likely to be over-estimated if we rely solely on data from our clinical material. Moreover, since inter-parental conflict is significantly more likely to associate with GHQ symptoms in either parent than is inter-generational conflict with symptoms in pupils or parents (Table IV) no specific pathogenic effects could be attributed to the conflict being inter-generational rather than intra-generational. Finally, though conflict or

alienation may lead to psychiatric disorder, the reverse may also be true (Rutter *et al.*, 1976).

Since our study attempted to find group differences in the reported prevalence of symptoms, we used GHQ-28 solely for this purpose without attempting to demarcate illness and well-being, or to define 'cases' with cut-off points for sensitivity or specificity at this stage. The groups we studied cannot claim to be representative of the total youth population they derive from: obvious constraints include the requisites for admission of non-Kuwaitis to the government schools studied and the inability to investigate the paternal social class correlates of our findings. In Kuwait, social classes defined by standards of living are not consistently dependent on educational and occupational achievements (Kurtz, 1981).

Thus, our hypothesis that the magnitude of inter-

generational conflict varies with differences in nationality and sex was confirmed, but the hypothesis about relationship of conflict to locality of childhood upbringing was only partly confirmed. Inter-generational educational differences, rather than age differences as such, showed a positive association with inter-generational conflict. Our hypothesis that greater inter-generational conflict associates with more symptoms did not receive statistical confirmation.

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Psychosocial Risk Factors and Urban/Rural Differences in the Prevalence of Major Depression

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The Piedmont Health Survey interviewed 3798 adult community residents in a region of North Carolina. Current major depression was nearly three times more common in the urban than in the rural counties; rural residence decreased the risk of major depression for some, but not all, demographic subgroups. The risk of major depression was decreased for young rural residents, compared with their urban age peers; rural residence was more protective for young women than for young men. Rural residence appears to be a buffer against major depression.

Despite growing emphasis on the genetic and biological components of psychiatric disorders, the social origins of mental illness continue to receive attention. Urban or rural residence is one social characteristic that has a long history of consideration as a risk factor for psychiatric morbidity (Schwab *et al*, 1974; Neff, 1983; Dohrenwend & Dohrenwend, 1974); the usual hypothesis is that urban living places individuals at greater risk for psychiatric disorder—especially the more reactive or stress-induced disorders such as depression or anxiety. This is compatible with public images of urban and rural environments, and is based on the assumption that urban areas are characterised by change, interpersonal estrangement, anonymity, and conflicting values, whereas rural areas are characterised by social stability and integration, supportive interpersonal networks, and consensus about moral and political issues. Rural areas are also aesthetically superior to large cities—a skyline of high-rises and smokestacks cannot compare to the pastoral beauty of a pond surrounded by open fields or sheltering forests. Indeed, although results have not been unanimous, the majority of previous studies support the hypothesis of increased prevalence of psychiatric disorder in urban areas (Dohrenwend & Dohrenwend, 1974).

However, there are several reasons for continued exploration of this issue. Firstly, most previous studies of urban/rural differences in the prevalence of psychiatric illness used global scales of psychological well-being or numbers of psychiatric symptoms, rather than specific psychiatric disorders defined by reliable diagnostic standards. Secondly, urban/rural comparisons have often been made across rather than within studies, thereby obscuring urban/rural differences with regional, cultural, or methodological variations between studies. Thirdly,

most previous studies failed to take into account compositional differences between urban and rural areas: if urban/rural residence is a risk factor for psychiatric morbidity, that relationship should be observed once compositional differences (e.g. age, sex, socio-economic status) between urban and rural settings are statistically controlled.

However, there are other reasons to suspect the usual hypothesis that rural residents are advantaged in terms of the risk of psychiatric disorder—or at least to question its generalisability across time and space. Several authors (e.g. Flax *et al*, 1979; Srole, 1978) contend that the images of cities as evil and rural areas as peaceful enclaves have long been based more on romantic ideals than on realistic assessments of the two environments. Urban areas have been defended by those who point out that although cities may not be characterised by integration and cohesion, neighbourhoods within them are frequently stable and supportive, and may be the more appropriate unit of analysis (Srole, 1978). Others have pointed out that rural residents are more conservative and less flexible than their urban counterparts (Rogers & Burdge 1972; Hassinger, 1976), and that rural areas are not without social problems—isolation, lack of social and medical resources, outwards migration of the young, and relatively high rates of poverty and unemployment (Flax *et al*, 1979; Srole, 1978).

The potential effects of social change also may be relevant. Areas characterised by low population density may be considerably less 'rural' than they were 10–30 years ago. Mass communications (especially television), motorways, inwards migration of people who commute to urban jobs, etc., may have resulted in a significant degree of urbanisation of areas that remain sparsely populated. Thus, even if there has been an historical pattern of urban/

rural differences in the distribution of psychiatric disorder, that pattern may have been eliminated or substantially reduced by the homogenisation of society.

This paper examines urban/rural differences in the prevalence of current major depression. Previously (Blazer *et al*, 1985) we reported urban/rural differences in the prevalence of eight of the most common psychiatric disorders. Bivariate analyses suggested that the psychiatric diagnosis most strongly related to urban/rural residence was depressive disorder; current major depressive disorder was nearly three times more common among urban than among rural residents. Moreover, this difference persisted when the effects of standard demographic variables (i.e. age, sex, race, marital status, and five-year residential mobility) were statistically controlled (Blazer *et al*, 1985). The present paper builds on our previous work in two ways. Firstly, the degree to which the urban/rural difference in the prevalence of major depression is consistent across demographic sub-groups is analysed: the results suggest that rural residence significantly buffers the risk of major depression only among certain demographic sub-groups. Secondly, a multivariate logistic regression procedure is used to predict major depression on the basis of an expanded set of demographic variables and selected social characteristics which previous research suggests are risk factors for major depression. The multivariate analysis is important because it permits us to determine whether place of residence remains a significant predictor of major depression, after controlling for a variety of other demographic and social risk factors.

Compared with previous studies, these analyses have a number of distinct advantages: the dependent variable is a specific psychiatric disorder, rather than a global measure of symptom level or amount of impairment; the urban and rural subsamples are from the same geographical region and share a common history, cultural traditions, and political structure; and use of multivariate statistical procedures permits us to determine whether place of residence affects the risk of current major depression after normalising for urban/rural differences in the social demographic compositions of the two sub-groups.

Method

Sample and Data

The Epidemiologic Catchment Area (ECA) Program is a collaborative research project, funded by the National Institute of Mental Health. Its major goals are to examine

the prevalence of psychiatric disorders, risk factors associated with the onset of mental illness, and health service utilisation for psychiatric morbidity (Regier *et al*, 1984). Five universities participated, surveying defined local populations: Yale University (Greater New Haven, Connecticut), Johns Hopkins University (East Baltimore, Maryland), Washington University (Greater St. Louis, Missouri), Duke University (the Piedmont region of North Carolina), and the University of California at Los Angeles (East Los Angeles/Venice, California). The data reported here are from the Duke University ECA Project, locally called the Piedmont Health Survey (PHS).

Each ECA site randomly sampled its defined population to generate stratified random samples of approximately 3000 community residents and 500 institutional residents. Three sites, including the PHS, also obtained oversamples of older non-institutionalised adults. The sampling frame for the community sample and elderly oversample for the PHS consisted of a five-county area, encompassing two Community Mental Health Center catchment areas: Durham County, an urban county (based on size of residence codes used by the US Bureau of the Census); and Franklin, Granville, Vance, and Warren Counties, which are rural. The two catchment areas are approximately equal in total population, although the rural one is much larger in area. The findings in this paper are based on reports from community residents (including the elderly oversample); the institutional sample of the PHS will not be described.

Within the sampling frame, three-stage probability sampling was used to select respondents. In the first stage, small geographical segments were selected randomly, after being stratified to reflect the racial, socio-economic, and urban/rural compositions of the five counties; in the second, housing units within segments were randomly selected; in the third, once a selected household was rostered for usual residents, the Kish (1965) method was used to identify the designated household respondent. Households for community sample and elderly oversample were drawn from the same segments. The only difference in selection of respondents for these two subsamples was age: household members aged 18 and over were eligible for the community sample, while household members aged 60 and over were eligible for the elderly oversample. For the community sample, 3911 households were screened to generate a sample of 3015 respondents. In the elderly oversample, 3371 households were screened, generating a sample of 906 respondents. The combined response rate for the community and elderly samples was 79%. Reasons for non-response included inability to obtain a roster of household members (10%) and refusal of a designated respondent to participate in the interview (11%).

The analyses reported in this paper are based on a sample of 3798 respondents. Reasons that certain interviews were not used included physical or mental impairment too severe to permit a reliable interview ($n=80$), failure to complete the interview ($n=37$), and determination by the research team that the interview data were unreliable ($n=6$).

Respondents completed an interview lasting approximately two hours. Information about psychiatric disorders

was obtained using the Diagnostic Interview Schedule (DIS)—a highly structured interview designed to be administered by lay interviewers and capable of generating computer-based diagnoses for selected DSM-III disorders (Robins *et al*, 1981). The DIS elicits the elements necessary for a diagnosis: symptoms; their severity, frequency, and distribution over time; and whether they can be attributed to physical illness, alcohol or drug use, or are psychogenic in origin. Both the questions and the probes are fully specified, and the interview is pre-coded so that data can be computer-entered after editing. The DIS generates information about both current and lifetime prevalence of selected DSM-III disorders.

This paper focuses on one DIS/DSM-III diagnosis—major depression. A respondent was diagnosed as having major depression if he or she experienced symptoms sufficient to meet criteria for a major depressive episode and had no history of manic episodes. Other DSM-III exclusion criteria (American Psychiatric Association, 1980) were not used, largely because of recent data questioning their validity (Boyd *et al*, 1984). This paper reports only current major depression, defined as depression experienced during the six months prior to the interview. For the sample in this paper the prevalence of current major depression was 1.9% (based on weighted data), which is slightly lower than those reported by three other ECA sites (Myers *et al*, 1984).

Other variables included in analysis were respondents' demographic characteristics and selected social characteristics that previous literature suggest are risk factors for major depression. The demographic factors examined were age, race (essentially white or black in this sample), sex, education, place of residence (urban or rural), socioeconomic status measured in quartiles, and marital status. The other social characteristics examined have been closely linked to depression or to depressive symptomatology in previous research: the experience of one or more stressful life events in the past year (Markush & Favero, 1974; Brown & Harris, 1978), whether or not the respondent is the mother of pre-school children (Brown & Harris, 1978; Weissman & Paykel, 1974) and the degree to which a confidant is available, which appears to be an especially important dimension of social support (Lowenthal & Haven, 1968).

Methods of analysis

As reported previously (Blazer *et al*, 1985), current major depression was nearly three times more common among urban than among rural Piedmont residents. In order to obtain more detailed information about urban/rural differences in the prevalence of major depression, chi-squared tests were used to compare urban and rural residents within subgroups stratified on the basis of age, sex, and race.

In later analyses, logistic regression was used to examine the effects of urban/rural residence on major depression, controlling for demographic and social characteristics that previous literature and our earlier findings indicated are risk factors for major depression. Logistic regression is the best technique when the dependent variable is dichot-

omous, as is the case in predicting absence or presence of current major depression. Using logistic regression, independent variables may be either discrete or continuous; in this paper, all independent variables were coded in discrete categories. For each independent variable a set of two or more dichotomous dummy variables was created, $n-1$ variables were estimated in the logistic regression equation, where n is the number of dummy variables used to represent the total set of categories comprising the independent variable. Logistic regression analysis generates a coefficient for each variable in the equation; these coefficients equal the natural logarithms of the odds ratios for those predictors. The coefficients are presented here as odd ratios. Each omitted or reference category of the independent variable was assigned an odds ratio of 1.00; the odds ratios for the estimated categories represent the odds having major depression relative to the reference category. Similarly, tests of statistical significance were based on a comparison of the estimated categories with their reference categories (Kleinbaum *et al*, 1982).

Most of the analyses reported here, and all tests of statistical significance, were based on weighted data. The design weights used correct for non-response, adjust for household probability selection, and generate distributions that match the age, sex, and race profiles of the adult population of the five county area generated by the 1980 US Census. Although the distributions remain adjusted for these factors, sample size was downweighted to the original number of respondents for tests of statistical significance.

Results

Table I shows the demographic and social characteristics of the PHS respondents, based on both unweighted and weighted data; distributions are presented separately for urban and rural residents. The unweighted distributions are greatly skewed by the large proportion of older respondents; e.g. in addition to affecting the age distribution, inclusion of the elderly oversample in the unweighted data also inflates the proportions of the sample who are female, white, widowed, and have low levels of education and socio-economic status. The weighted distributions, however, are corrected for the elderly oversample and other biases, resulting in demographic distributions that are representative of the adult population of the five counties.

As indicated by the weighted distributions, the PHS sample has age and sex distributions characteristic of both North Carolina and the USA generally. The sample has a large proportion of non-whites, the vast majority of whom are black; the rural counties have higher proportions of non-whites than the urban county. The distributions for educational attainment and socio-economic status (SES) are somewhat lower than comparable national figures—especially for the rural PHS respondents. This reflects the generally lower educational and occupational attainments of Southerners in general and of rural Southerners in particular. In terms of the social risk factors, about one-third of the sample had experienced one or more stressful life events in the past year, about 10% of the sample consisted of women with small children at home, and three-quarters

TABLE I
Demographic characteristics of Piedmont Health Survey respondents by place of residence

	<i>Rural (n=1936)</i>		<i>Urban (n=1862)</i>	
	%	Weighted %	%	Weighted %
Age				
18-24	8	17	12	18
25-44	29	37	35	43
45-64	29	29	27	26
65 or older	34	17	26	13
Sex				
Male	37	44	41	47
Female	63	56	59	53
Race				
White	59	53	68	69
Non-white	41	47	32	31
Marital status				
Married	54	61	50	57
Widowed	24	11	16	8
Separated/divorced	10	9	14	11
Never married	12	19	20	24
Education				
Less than High School	50	45	33	25
High School grad/GED	42	48	42	48
College grad	8	7	25	27
Socio-economic status				
Quartile 1	36	27	21	14
Quartile 2	35	41	28	28
Quartile 3	22	24	31	35
Quartile 4	7	8	20	23
Number of stressful life events				
None	64	62	65	64
One or more	36	38	35	36
Mother with small children				
No	93	90	93	91
Yes	7	10	7	9
Availability of a confidant				
Seldom	8	8	7	7
Sometimes	16	16	19	18
Usually	76	76	74	75

TABLE II
Urban/rural differences in the prevalence of major depression, by age, sex and race (weighted data; weighted cell sizes in parentheses)

	<i>Male</i>			<i>Female</i>			<i>Total</i>
	<i>White</i>	<i>Non-white</i>	<i>Total</i>	<i>White</i>	<i>Non-white</i>	<i>Total</i>	
Age 18-44							
Urban	1.2 (448)	0.8 (187)	1.1 (637)	3.0 (417)	6.5 (257)	4.3 (675)	2.7 (1312)
Rural	0 (204)	0 (222)	0* (426)	0** (245)	2.2* (233)	1.1** (478)	0.6** (904)
Age ≥45							
Urban	1.8 (272)	0 (96)	1.3 (368)	2.3 (322)	2.3 (126)	2.3 (449)	1.8 (817)
Rural	1.4 (174)	0.4 (128)	1.0 (303)	1.3 (267)	1.3 (194)	1.3 (461)	1.2 (764)

* $P < 0.05$

** $P < 0.01$

of the respondents reported that they usually had a confidant available. Interestingly, the social characteristics—unlike the demographic variables—did not differ between urban and rural residents.

Table II shows the prevalence of six-month major depression by urban/rural residence, stratified for age, sex, and race. The significance tests refer to the difference between urban and rural prevalences within the demographic subgroups. The data strongly suggest that rural residence buffers young adults, rather than middle-aged and older adults, from major depression. Moreover, although this pattern holds true for both sexes, the urban/rural difference is more striking and consistent for young women. In contrast, the relationship between place of residence and the prevalence of current major depression does not appear to differ by race.

The distribution of prevalences in Table II strongly suggests an interaction between age and urban/rural residence affecting the prevalence of major depression—and perhaps a three-way interaction involving age, sex, and place of residence. Unfortunately, the small number of cases of current major depression in the PHS sample precludes stable estimates of these and other potential interaction effects in both the cross-tabular and logistic regression analyses.

Table III gives the results of the logistic regression analysis in which demographic variables and selected social characteristics are used to predict major depression. Six of the nine independent variables exhibit significant odds ratios. Most importantly, from the perspective of this paper, place of residence remains a significant predictor of major depression, normalising for other demographic and social factors. As was true in the bivariate case, the odds of having major depression are nearly three times greater among urban than among rural residents. As expected on the basis of previous literature, the likelihood of having major depression also is significantly related to sex, SES, being a mother with small children, experiencing recent stressful life events, and the availability of a confidant. In contrast, normalising for other variables in the equation, marital status, race, and age were not significantly related to current major depression.

The six statistically significant predictors of current major depression differ in magnitude. Absence of a confidant is an especially strong predictor of depression (odds ratio = 3.79). Stressful life events and sex are also relatively powerful predictors, as would be expected on the basis of previous research (odds ratios of 2.31 and 2.93, respectively). Only one of the levels of SES is significantly different to the reference category; nonetheless, two of the estimated coefficients exceed 2.0, and we conclude that SES is an important predictor of major depression. More troubling than the size of the coefficients for SES is their direction. Normalising for other variables in the equation, SES exhibits a non-linear relationship with major depression. Moreover, all three higher levels of SES increase the odds of having major depression, compared with the lowest category, and upper middle class respondents are at greater risk. These findings are contrary to the more usual pattern of increased symptomatology among lower SES persons (Dohrenwend & Dohrenwend, 1969),

and may reflect the fact that in this study other demographic correlates of SES have been statistically controlled. Finally, as expected, mothers with pre-school children at home are at greater risk of current major depression than other respondents. This odds ratio is somewhat smaller than the others, but is significant.

The logistic regression for which results are given in Table III is our choice of a 'final model'. A series of logistic regression equations were estimated, the models differing slightly in the specific combinations of independent variables included. This equation was chosen as the most useful model, i.e. it includes those variables that proved to be most relevant in terms of their predictive power or importance as control variables. Education was not included in

TABLE III
Multiple logistic regression equation predicting current (six-month) major depression (weighted data)

<i>Independent variable</i>	<i>Odds ratio</i>
Place of residence	
Rural	1.00 ¹
Urban	2.99**
Age	
18–24	1.00 ¹
25–44	0.83
45–64	1.43
65 or older	0.59
Sex	
Male	1.00 ¹
Female	2.93**
Race	
White	1.00 ¹
Non-white	1.44
Socio-economic status	
Quartile 1	1.00 ¹
Quartile 2	2.02
Quartile 3	2.50*
Quartile 4	1.65
Mother with small children	
No	1.00 ¹
Yes	1.90*
Stressful life events	
None	1.00 ¹
One or more	2.31**
Availability of confidant	
Usually	1.00 ¹
Sometimes	1.45
Seldom	3.79**
Marital status	
Married	1.00 ¹
Widowed	0.88
Separated/divorced	0.70
Never married	0.80

¹Reference category

* $P < 0.05$

** $P < 0.01$

the final model, because its direct effect was non-significant and because its inclusion was partially redundant, given that the SES scores are based on a weighted combination of education, occupational status, and income. Employment status was included in some earlier models, but its predictive power and effect as a control variable were negligible—again, perhaps, because of the inclusion of SES. Death of a parent during childhood (a variable suggested as a risk factor for depression in previous research), residential relocation during the past five years, and household size were also tested, but eliminated because they served no purpose in terms of predictive power or control variables.

The results in Table II suggest an interaction between age and place of residence in the prevalence of current major depression. Although the low overall prevalence of major depression precluded testing this possible interaction, separate logistic equations were estimated for relevant subgroups to explore this possibility further. In all of these subgroup analyses, the ratio of independent variables to number of cases in the dependent variable was unfavourable. Consequently, the results of these analyses must be interpreted cautiously; although they have not been tabulated, the findings will be briefly described.

Firstly, the equation in Table III (with the exception of place of residence) was estimated separately for urban and rural residents. As expected, age had substantially different effects in these two models. In the equation for urban residents, younger respondents were at the greatest risk of major depression, normalising for other variables. The opposite was true in the equation for rural residents: younger respondents were at the lowest risk of major depression. Secondly, the logistical equation in Table III (minus age) was estimated separately for respondents aged 18–44 and for those aged 45 and older. Again, the findings support the pattern of subgroup differences in Table II. Place of residence was a significant predictor of major depression for younger respondents (with urban residence increasing the risk of depression), but was not significant for older respondents.

Discussion

Two findings are of particular importance to our understanding of the role of urban or rural residence as a risk factor for current major depression. Firstly, the results suggest that rural residence is a more important buffer against major depression for some demographic subgroups than for others. Rural residence significantly decreases the risk of major depressive disorder only among young adults aged 44 and younger and is perhaps more of a buffer for young women than for young men; this finding is useful because it suggests that the protective qualities of rural life are not uniformly experienced by all rural residents. Secondly, the logistic regression analysis strongly suggests that place of residence is a significant predictor of major depressive illness, normalising for a variety of demographic and social

characteristics which have been suggested as risk factors for depression in previous research. This finding suggests that the advantage enjoyed by rural residents in terms of risk of depression is not due to compositional differences between urban and rural populations. This is especially relevant in that the demographic profiles indicated substantial demographic differences between the urban and rural subsamples.

Given these findings, it remains unclear why or how rural residence translates into a protective buffer against the risk of current major depression. Our expectation was that the bivariate relationship between place of residence and major depression would be eliminated, or at least substantially reduced, after demographic differences between the urban and rural subsamples were taken into account. At this point, we can only echo the sentiments of earlier studies which suggest that there is some elusive quality about rural environments that serves as a protector against psychiatric illness—in this case, major depression. Although we have not identified that quality yet, we have at least ruled out a number of potential contenders. Moreover, it appears that, at least for depressive disorder, the quality we are seeking must be one which has a stronger effect on young adults than on middle-aged and older persons.

The age-specificity of the relationship between place of residence and major depression complicates interpretation and the search for explanatory factors. It argues against the perspective that the uniqueness of rural living is being undermined by urbanisation and social change. Numerous investigators have demonstrated consistently that social trends have greater effects on younger members of the group experiencing change (Ryder, 1965; Riley, 1976). Thus, if rural environments are becoming more urban-like in terms of lifestyles and values, we would have expected the exact opposite of what we observed, i.e. that urban/rural differences would be greater among older adults. It is possible that social change in general is experienced first and most rapidly in urban areas and that, because young adults are most affected, the impact of social change is to elevate the prevalence of major depression in young urban residents compared with their rural age-peers. There are no empirical data supporting this hypothesis, however.

This study has several limitations. Firstly, like all self-report surveys, the DIS and other instruments included in the PHS are vulnerable to deliberate or inadvertent misreporting of information. This is especially pertinent to the DIS, where accurate reporting of sometimes sensitive and highly personal

symptoms is required for valid measures of psychiatric disorder. The effects of forgetting symptoms, however, should be minimised in this study because of the exclusive focus on current disorder. Moreover, there is no reason to believe that the reporting of symptoms would be unequally distributed across urban and rural residents.

Secondly, although the DIS is designed to elicit diagnoses compatible with DSM-III diagnostic criteria, the diagnoses generated by the DIS are not identical to those made by psychiatrists performing diagnostic assessments of clinic patients (Robins *et al*, 1981; Helzer *et al*, 1985). Nonetheless, the DIS generates reliable diagnoses, and appears to be superior to the instruments used in most previous studies of urban/rural differences in psychiatric disorder. Again, there is no reason to suspect that the validity of the DIS would be different for urban and rural residents.

A final limitation of the study is potentially more consequential. This issue is related to two characteristics of the data used in this study: the data are cross-sectional, and retrospective data about migration history were not collected. The issue of concern is selective migration, which has been a long-term concern in studies of urban/rural differences in the prevalence of psychiatric illness. Given our reliance upon cross-sectional data and the lack of a detailed migration history, we cannot know whether current residence as urban or rural preceded or followed the onset of depressive disorder. We believe that this is less of an issue because of our focus on current disorder—i.e. the more recent the disorder examined, the less likely that the respondent has subsequently moved. Moreover, although the PHS did not obtain a migration history, information was elicited about length of residence at the respondent's current address; this factor was not significantly related to major depression and was not useful as a control variable. Length of residence does not directly cover the issue of selective migration, in that we could not distinguish between moves between and within urban and rural settings. Additionally, the most recent move may not be the crucial piece of information; for example, Schwab *et al* (1974) found a significant relationship between

size of community of origin and psychiatric impairment as measured by the Health Opinion Survey, regardless of current residence. Thus, we cannot rule out selective migration or community of origin as possible explanations for the observed urban/rural differences in the prevalence of current major depression.

Because these findings raise a number of questions, they point to exciting areas for future research. The trend toward examining the epidemiology of specific psychiatric disorders (rather than symptom scales) should be continued. The PHS research team is currently examining urban/rural differences in the prevalences of anxiety disorders and alcohol abuse and dependence; although only preliminary analyses are available, it appears that rural residence does not invariably decrease the risk of psychiatric disorder. As other psychiatric disorders are examined, care should be taken to determine whether the effects of place of residence are invariant across demographic subgroups and remain statistically significant after normalising compositional differences between urban and rural residents. Such information refines our understanding of the effects of place of residence, and has the potential to identify some of the mechanisms underlying bivariate relationships between urban/rural residence and psychiatric disorder. Perhaps most importantly, effort should be devoted to specifying what it is about urban and rural environments that affects the risk of psychiatric disorder. As operationally defined, the distinction between urban and rural residence is based simply upon population density; although it is possible that the number of people per square mile has a direct effect on mental health, it is more likely that place of residence now serves as a proxy for one or more important social processes that affect the risk of psychiatric disorder. Identification of those processes could have important implications for our understanding of the aetiology of psychiatric disorder and for its treatment.

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Neurological Factors in Obsessive Compulsive Disorder Two Case Reports and a Review of the Literature

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Two patients are described in whom an obsessive-compulsive disorder began just after the onset of epilepsy. Neurological factors in OCD are then reviewed, and clinical reports of patients with OCD and co-existing brain pathology, EEG abnormalities, CT scan changes and electrical stimulation of the brain discussed. It is concluded that neurological factors are present in only a minority of cases of OCD. Successful behavioural treatment is independent of such factors.

Obsessive-compulsive disorder (OCD) has long been an enigma in psychiatry because of its bizarre presentation. We report two cases in which typical OCD developed in teenage patients shortly after the onset of epilepsy. One patient had temporal lobe fits, the other grand mal fits. Both patients were free of obsessive-compulsive symptoms before the onset of epilepsy.

Case Histories

Case 1

A 16 year-old student had been well until the age of 11, when he developed left-sided classical migraine headaches. During a migraine attack at the age of 12 he had a fit, and subsequent investigation after more fits revealed a temporal lobe seizure disorder. Six months after the first fit the patient developed obsessive-compulsive symptoms. He felt an irresistible urge to clean things with the sleeve of his shirt, and soon wore holes in his jacket. He also started smoothing the velvet fittings at home so that the pile ran all in one direction. He later developed other compulsions, such as putting his handkerchief in his pocket in a certain way, or hanging up his trousers in an exact fashion with precise rituals which would take ten minutes or more to perform. The number three took on a magical significance, and the patient felt compelled to perform many acts three times; for example, he would dry his face three times after washing, or would cough three times when he had to cough. These tasks would have to be done in an exact manner, otherwise he would become consumed with worry. He feared that imperfection in these tasks would magically cause him to have a seizure, or to lose sleep at night.

There was no psychiatric illness in his family. One sister had suffered a febrile convulsion as an infant. The patient was raised by both parents at home, and had a warm relationship with them. They worked full time: the father was a policeman, the mother a secretary. He had one sibling, a sister one year younger. The patient was the

product of full term, normal delivery, and achieved normal developmental milestones. Early childhood was happy, and he had many friends and enjoyed sport. He began school at the age of five, and performance was normal until the onset of his obsessive-compulsive symptoms, after which his academic performance dropped.

The patient had migraine and temporal lobe epilepsy. Fits would occur with varying frequency, from two/day to one/week, and were treated with carbamazepine and phenytoin. An electroencephalogram (EEG) showed frequent isolated spikes and slow complexes of temporal lobe origin, more in evidence on the right side. A physical examination gave normal results.

On examination the patient had normal mood, with no thought disorder, hallucinations or delusions. Attention and concentration were relatively poor, because of interruption by his obsessions. His intelligence was slightly above average.

The patient had 3 months of in-patient treatment by exposure *in vivo* for his fears and self-imposed response prevention. His rituals improved by 50%, and he returned home to live with his family.

Case 2

A 21 year-old white male had a long history of mental subnormality and behavioral problems, following a head injury at the age of three. He had his first fit (grand mal) at the age of 15, and shortly after developed fears that he would be contaminated by dirt and began ritual handwashing to combat this. He became fastidious about the way he arranged his clothes or food, and would only sit down after performing a series of precise rituals. If these rituals were interrupted, or if his family did not co-operate with them, he would strike out at objects around him or at himself. Over the year before admission his rituals had increased, becoming more handicapping. He ritualised before almost every action, most of his day being taken up by the rituals. It took him an hour to drink a cup of tea, and two hours to eat a sandwich or to go to the toilet.

As an infant his birth, delivery, and development milestones had been normal. He had burns to both legs at the

age of one, and regressed temporarily. At the age of three he hit his teeth and head on a car, and all his teeth were removed under general anaesthesia. After this he was never again a normal child. He regressed permanently in development, and became a frequent behaviour problem. His behavioural outbursts and his mental subnormality resulted in his transfer to a school for the educationally subnormal. He attended a series of schools, but his outbursts made formal education difficult. After the onset of his epilepsy and rituals his education was further impaired.

Treatment with primidone and carbamazepine reduced the fit frequency from one per week to once every three weeks. His EEG showed spike and wave components, predominantly in the left anterior quadrant, against a background of a slow alpha rhythm. A computerised tomography (CT) scan showed an increase in ventricular size. He had out-patient psychotherapy between the ages of 7 and 12 for his behavioural symptoms, and was admitted in 1980 and 1981 in unsuccessful attempts to ameliorate his OCD.

On examination, he frequently ritualised. He spoke in a halting, staccato tone with short bursts of words, without thought disorder. His mood was normal, and he denied experiencing hallucinations or delusions. He was alert and oriented, but his memory was only fair: he could recall only two or three nouns after three minutes. His concentration was good, and he could perform the 'serial sevens' mental arithmetic test without error. His sight into his condition and judgement about social situations were limited, and his intelligence was subnormal (Wechsler Adult Intelligence Scale full scale IQ 65; verbal 72; performance 60).

He had in-patient behavioural treatment by exposure and response prevention and a token economy system. After more than 300 hours of treatment, few gains had been made.

Discussion

Modern ideas about OCD emerged in the 19th century. Charcot & Magnan coined the term 'onomatomania' for patients who were disabled by 'imperative ideas', and in 1890 Culerre noted a link between epilepsy and Charcot & Magnan's onomatomania (Tuke, 1894). Although Freud (1894) regarded obsessive states and neuroses, attributing their origin to repressed memories of sexual guilt, other writers continued to regard them as organic in origin. Tuke (1894) considered that they were the result of abnormal cortical function, a view supported by other authors in the early part of this century (Wexberg, 1938; Schilder, 1938).

Since these early reports a number of authors have detected 'organic' features in OCD, but there is marked disagreement about their frequency. Since the mind is the brain in action, psychopathology must at some level reflect dysfunction of brain activity. Our division of 'organic' from 'functional'

reflects our ignorance, as the two are at the ends of a continuum along which neurology and psychiatry merge into one another. Present tests of brain function are crude. As better indicators of brain activities are developed, so will dysfunctions become more reliably evident. Better attention to controls and to sampling would also reduce the current discrepancies of opinion.

Links with specific neurological conditions

Encephalitis and encephalopathy. There were many reports of obsessive-compulsive symptoms accompanying encephalitis lethargica in the years following the pandemicity of this condition after World War I (e.g. Bender, 1935; Brickner, 1940; Schilder, 1938). The phenomena included counting and other rituals, and obsessive thoughts. They often occurred in short bursts lasting a few minutes, and might be accompanied by oculogyric crises, anxiety and aggression. Many of these patients had developed Parkinson's disease, suggesting impairment of the basal ganglia and substantia nigra. There were no neuropathological differences between encephalitics with and without obsessive-compulsive symptoms. Compulsive counting without anxiety but with psychic akinesia was described in two patients with encephalopathies who had bilateral necrosis of the globus pallidus, one following a wasp sting and the other following carbon monoxide poisoning (Laplane *et al.*, 1984).

Parkinson's disease. Obsessive traits (not symptoms) are frequent in Parkinsonism (Trimble, 1981), but Parkinsonism is usually a disorder of the elderly, whereas OCD mainly affects young adults. Perhaps paradoxically, oral levodopa for Parkinsonism occasionally produces obsessive-compulsive symptoms as a side effect (Yaryura-Tobias & Neziroglu, 1983).

Diabetes insipidus. This is a rare association. A case of Parkinsonism, diabetes insipidus and compulsive behaviour was described by Courtney (1928). Five of nine cases described by Barton (1965) had OCD in addition to excess drinking; one had a pinealoma and another was post-encephalitic. According to Barton (1976) either the diabetes insipidus or the OCD could start long before the other began. Vasopressin did not relieve the obsessive-compulsive symptoms (Barton, 1976). One study found more history of *neurological disorder* in OCD than in other neuroses, but the label of 'neurological' was swelled by peripheral problems

such as diphtheria, herpe zoster and Bell's palsy (Grimshaw, 1964).

Head injury. Head injury was followed by OCD in 3% of 415 cases reported by Hillbom (1960), but in only 0.3% of 670 cases studied by Lishman (1968). Four cases of OCD were reported by McKeon *et al* (1984), in whom the symptoms began shortly after the head injury – in one case within 24 hours.

Birth injury. A history of birth injury was found in a third of 33 patients with OCD, more than in other psychiatric patients (Capstick & Seldrup, 1977). However, only one of the obsessive-compulsives had physical signs of brain damage. These authors noted that the patients with a birth injury tended to have more 'bizarre and magical' rituals.

Epilepsy. Links between OCD and epilepsy have been noted in a trickle of case reports. Napoleon had epilepsy and a compulsive ritual of counting the number of windows in the buildings he passed. This century has seen a few case reports. Two patients developed petit mal a few months after first showing obsessive-compulsive symptoms (Pacella *et al*, 1944). Another subject developed typical OCD when *grand mal* fits stopped suddenly after 15 years (Garmany, 1947). The present paper describes two cases of OCD which began just after the new onset of epilepsy, the fits in one coming from the temporal lobe. Gibson & Kennedy (1960) describe one woman in whom the spike and wave discharges on EEG would increase when her obsessive-compulsive symptoms became worse. Stereotyped thoughts or forced thinking have been described as part of an epileptic aura (Penfield & Jasper, 1954), and obsessive-compulsive symptoms may appear briefly during the aura of a fit associated with temporal lobe pathology or reappear transiently in the postictal phase (Brickner *et al*, 1940). Further cases of epilepsy with ictal or interictal obsessive-compulsive symptoms were noted by Yaryura-Tobias & Neziroglu (1983).

Obsessive personality traits are frequent in the general population, so it is hardly surprising that they are also found in some epileptics. Guerrant *et al* (1962) found that they occurred no more often than in other patients with chronic illnesses.

Other considerations

EEG abnormalities in OCD. Varying rates of EEG abnormalities in OCD have been reported, ranging

between 11% and 65% (Bingley & Persson, 1978; Ingram & McAdam, 1960; Inouye, 1973; Insel *et al*, 1983; Pacella *et al*, 1944; Rockwell & Simons, 1947; Sugiyama, 1974). The EEG abnormality may be most marked over the temporal lobes either in a waking state (Jenike & Brotman, 1984) or during sleep (Epstein & Bailine, 1971). Visual evoked potentials have been reported as abnormal (Ciesielski *et al*, 1981; Beech *et al*, 1983). According to Shagass *et al* (1984) the pattern of the evoked potential in 14 obsessive-compulsives could be distinguished from the pattern in other neurotics and schizophrenics. The sleep EEG in OCD sufferers differed from that of controls, but was similar to the EEG of age-matched depressives (Insel *et al*, 1982; 1984). Among adolescents who had OCD and a history of major depression, the sleep EEG also resembled that of young adults with primary depression (Rapoport *et al*, 1981). Flor-Henry *et al* (1979) described eleven patients with OCD who had left frontal lobe dysfunction shown by abnormal EEG variability and neuropsychological impairment. However, such abnormalities in the EEG and similar tests were not found by Insel *et al* (1984) in 18 cases of OCD. In children with OCD, EEG abnormalities were uncommon, mild and non-specific (Flament & Rapoport, 1984), although they had deficits on neuropsychological tests similar to those in frontal lobe lesions.

CT scan changes in OCD. Flament & Rapoport (1984) reported increased ventricular size in children with OCD. Sixteen adolescents with OCD had a higher mean ventricular:brain ratio (and more spatial perceptual deficits) of frontal type than did matched controls (Behar *et al*, 1984). Other studies (Rapoport *et al*, 1981; Insel *et al*, 1983) noted no changes in adults with OCD.

Psychosurgery. Relief of OCD after psycho-surgery indicates some of the pathways that can partly mediate the problem, though they may well be downstream from its origin. Benefit has followed surgical lesions the orbital frontal cortex, anterior cingulum and related areas. In 334 patients with OCD from 12 series (reviewed by Yaryura-Tobias & Neziroglu, 1983, p197) 54% improved markedly and a further 24% improved moderately after various psychosurgical operations. Further evidence of improvement in OCD after surgery was reviewed by Marks (1986).

Electrical stimulation of the brain. Relevant information has been yielded by electrical stimulation of

the brains of subjects without OCD. In one woman, area 6 on the medial side of the left hemisphere was stimulated repeatedly while she recited the alphabet (Brickner, 1940). Whenever she was being stimulated the patient would repeat the same letter over and over again. This perseveration ceased instantly when the stimulation was stopped. The areas were then excised, but pathological examination detected no abnormality. Different movements – sucking, palpation or nibbling – were evoked in 52 epileptics during electrical stimulation of certain sites in the anterior cingulum (Talairach *et al.*, 1975). The patients experienced these movements as involuntary and forced on them, as occurs with compulsive rituals. Unlike rituals, however, the movements were associated with euphoria and not anxiety, were not repetitive, and were less complex.

Conclusions

Rare cases of obsessive compulsive disorder have been reported after lesions of the hypothalamus, third ventricle and basal ganglia. A handful of obsessive-compensatives had diabetes insipidus. OCD occasionally accompanies epilepsy, as in the two cases described in the present report. Stimulation of area 6 on the medial side of the left hemisphere has led to perseveration in one case. Surgical lesions of

the anterior cingulum and frontal areas can improve severe and intractable OCD. Such data indicate pathways that can mediate occasional obsessive-compulsive symptoms. These pathways, however, could be well downstream from the usual mechanisms that initiate and maintain OCD, and precise understanding of them is still a long way off. Most cases of OCD have no organic pathology detectable by present methods; there is great disagreement about the proportion who have abnormal EEGs or psychological tests or a history of brain injury.

The evidence for neurological factors therefore remains 'tantalizing' (Elkins *et al.*, 1980) but inconclusive. Newer tools such as the positron emission tomography scan or neurotransmitter studies might unlock further secrets of this elusive disorder. However, aetiology and treatment need not go hand-in-hand. Despite the neurological factors seen in some obsessive-compulsive patients, most cases of OCD improve markedly and lastingly with behavioural treatments (Marks, 1986), although this was not true for the 2 cases of epilepsy we report – perhaps their anti-ictal drugs or their brain dysrhythmias disrupted learning.

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The Dexamethasone Suppression Test in Generalised Anxiety Disorder

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The dexamethasone suppression test was performed on 79 patients with a diagnosis of generalised anxiety disorder. A non-suppression rate of 27% was obtained, comparable to that found in out-patient major depression but notably higher than previous reports in panic disorder. No good clinical predictors of non-suppression were discovered, nor was the co-occurrence of depression sufficient to account for the finding.

The dexamethasone suppression test (DST) has become the most widely applied neuroendocrine measure in depression research. Non-suppression of cortisol has consistently been reported to occur in 40-50% of in-patients suffering from endogenous depression (Carroll, 1982). Reports of non-suppression rates for out-patients have been lower, ranging from 15-25% (Winokur *et al*, 1982; Amsterdam *et al*, 1982; Rabkin *et al*, 1983). Claims for the specificity of DST non-suppression range around 95% (Carroll, 1982), but recent reports suggest significant non-suppression in other psychiatrically ill patients (Dewan *et al*, 1982; Castro *et al*, 1983; Targum, 1983; Coppen *et al*, 1983; Raskin *et al*, 1982) and normal control subjects (Winokur *et al*, 1982; Amsterdam *et al*, 1982; Coppen *et al*, 1983; Hallstrom *et al*, 1983).

Anxiety states are the disorders most closely linked to depression. Recent studies of family history (Crowe *et al*, 1983) and of the course of illness (Leckman *et al*, 1983) suggest a partially shared diathesis. There is also a suggestion that antidepressant drugs may have a therapeutic effect in the treatment of certain types of anxiety states (Sheehan *et al*, 1980; Klein, 1984) and that anxiolytics may be efficacious in endogenous depression (Feighner *et al*, 1983).

Three studies (Curtis *et al*, 1982; Lieberman *et al*, 1983; Sheehan *et al*, 1983) have reported on cortisol response to dexamethasone challenge in anxious patients. All three studies were restricted to the DSM-III diagnosis of agoraphobia with panic disorder (American Psychiatric Association, 1980), and all reported rates of DST non-suppression that were comparable with normal controls. The present study examines the cortisol response to dexamethasone in a group of patients suffering from generalised anxiety disorder.

Method

A group of 47 women and 32 men with a diagnosis of generalised anxiety disorder was studied; their ages ranged from 21 to 61 years, with a mean of 41.2 (sem = 1.30). The patients were self-referred to the Psychopharmacology Research Unit at the Department of Psychiatry, University of Pennsylvania, for treatment in a double-blind drug trial. All patients were diagnosed by a trained research psychiatrist, employing a standardised psychiatric interview based on DSM-III criteria. Patients with depressive disorders or other Axis I diagnoses were excluded. Two research psychologists administered the Schedule for Affective Disorders and Schizophrenia, Lifetime version (SADS-L; Endicott & Spitzer, 1978) to a random sample of 31 of the 79 patients. The psychologists were blind both to clinical diagnosis and to whether the patients were to enter a depression or anxiety treatment protocol.

The Hamilton Anxiety Scale (HAS), Raskin-Covi Scale and the Hopkins Symptom Checklist-90 (HSCL) were completed on all patients. A physical examination was carried out, and patients excluded if they had acute medical problems, a history of alcohol or drug abuse, were on concurrent psychotropic medication, or had an HAS score of less than 18 or a Raskin Depression score greater than 8. No patient met the exclusion criteria for DST (Carroll *et al*, 1981). Freedom from psychoactive medication for seven days prior to entry was confirmed by a urine drug screen in all patients.

Each patient was instructed to take 1 mg of dexamethasone orally at 2300 hrs on the night before their next clinic appointment. Blood was taken for plasma cortisol determination between 1200 and 1700 hrs; plasma cortisol concentration was measured as described by Amsterdam (1982). Cortisol cut-off values of both 4.0 µg/100 cm³ and 5.0 µg/100 cm³ were used to determine non-suppression. Chi-squared, Mann-Whitney, and one-tailed and two-tailed *t*-tests were used in the analysis of the data.

Results

Twenty-one of the 79 patients (27%) who met the criteria for generalised anxiety disorder were non-suppressors,

using the $4.0 \mu\text{g}/100 \text{ cm}^3$ cut-off, and 19 patients (24%) were non-suppressors on the basis of a cut-off of $5.0 \mu\text{g}/100 \text{ cm}^3$. The mean post-dexamethasone cortisol value for the non-suppressor group was 9.1 (sem=1.4) compared with $2.5 (=0.5) \mu\text{g}/100 \text{ cm}^3$ for suppressors. A blind SADS-L evaluation of a random sample of 31 of the patients confirmed the clinical diagnosis in all but three of the DST suppression subjects (revised diagnoses were one panic disorder, one atypical anxiety, and one generalised anxiety disorder with concurrent major depression) and in all but one of the DST non-suppressors (revised diagnosis: panic disorder).

Examination of differences in patient characteristics between the non-suppressor and suppressor groups was undertaken; five of the 32 men (16%) and 16 of 47 women (34%) were non-suppressors ($\chi^2=2.43$, NS). Pooled *t*-tests revealed no statistically significant differences in the ages, heights, or weights of the two groups. The scores on the individual items of the HAS and the HSCL were analysed for significant differences between suppressors and non-suppressors using the Mann-Whitney and an $\alpha=0.05$ significance level. On the HSCL, non-suppressors differed from suppressors for poor appetite, fearfulness, feeling nervous when left alone, absence of paraesthesias, and absence of feeling tense or keyed-up. On the HAS, the item relating to somatic (muscular) symptoms was significantly higher for non-suppressors. HSCL items were then grouped into the five factors, and pooled *t*-tests used to test for significant differences in the means between the suppressor and non-suppressor groups. None of the factors achieved significance, although there was a trend toward significance for the somatic factor ($P=0.06$) and the depression factor ($P=0.08$). Raskin depression scores also revealed no significant differences.

Discussion

The main finding of this study is a 27% rate of cortisol non-suppression after dexamethasone administration in out-patients suffering from generalised anxiety disorder; this is comparable to rates for out-patient major depression (Winokur *et al*, 1982; Amsterdam *et al*, 1982; Rabkin *et al*, 1983). This rate of non-suppression contrasts sharply with the low rate of abnormal DST results reported in three studies (Curtis *et al*, 1982; Lieberman *et al*, 1983; Sheehan *et al*, 1983) of patients with panic disorder. Curtis *et al* tested 20 patients with panic disorder, both with and without agoraphobia, and found three out of thirteen (23%) abnormal results in patients with agoraphobia and panic; they noted that two of the DST non-suppressors were in an alcohol withdrawal state, and that the other had a concurrent major depression. None of the other seven panic disorder patients without phobic avoidance had abnormal results. Lieberman *et al* reported

no abnormal results in ten panic patients, though the patients in this study had been premedicated prior to the test with diazepam (2–10 mg) or lorazepam (2–6 mg). Sheehan *et al* (1983) reported 4 out of 51 (8%) abnormal results in a sample of agoraphobics with panic disorder. In agreement with these three studies, we have found only one case of DST non-suppression in 11 patients with panic disorder.

Some explanations for the relatively high rate of dexamethasone non-suppression in generalised anxiety disorder must be considered. Firstly, it is possible that some patients were non-compliant with regard to dexamethasone administration, but the high level of co-operation with the other aspects of the out-patients drug treatment argues against this. Time of blood sampling is known to affect cortisol results, but sampling was usually done earlier than the conventional 1600 hrs, and therefore may have reduced rather than increased the incidence of non-suppression. The co-occurrence of depression might be a diagnostic artifact and explain the high levels of non-suppression in this sample. Blind SADS-L confirmation of clinical diagnoses makes the covert presence of syndromic levels of depression unlikely. At a subsyndromic level, three indices of severity of depression can be applied: the depression item on the HAS and the Raskin score on the Raskin-Covi scale both yielded non-significant differences between suppressors and non-suppressors, while the self-report depression factor on the HSCL only achieved a significance of $P=0.08$. Overall, it is unlikely that depression accounted for a large measure of the DST non-suppression.

Lastly, the data were examined for predictors of non-suppression. All of the HSCL individual items, HSCL factors, and HAS items were analysed for significant differences. Not surprisingly, there were some significant predictors of non-suppression in the HSCL items, but no more than would be expected to occur randomly, and none that matched our predictions. Sex did seem to have some effect, since females showed a trend for a high rate of non-suppression ($P=0.07$).

In summary, a sample of patients with generalised anxiety disorder was found to have a rate of DST non-suppression that was comparable to that found in out-patient major depression. This was in contrast to a much lower rate of non-suppression observed in studies of patients with panic disorder. No good clinical predictors of non-suppression were discovered, nor was the co-occurrence of depression enough to account for the present finding. Further studies of DST in generalised anxiety disorder patients need to be undertaken, both to confirm this result and to shed more light on its implications.

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Psychotherapy for Victims of Torture

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Three groups of torture victims were studied with the aim of establishing a concept of psychotherapy for such victims. Analysis of the first group, consisting of 200 case-reports made by Amnesty International medical groups, resulted in a general outline of the psychological methods of torture and their main impact on the victims. A second group of 24 torture victims was examined by the authors, and a quantitative assessment of long-term neuropsychological complaints and a qualitative insight into these symptoms achieved. In-depth interviews with victims in the third group which, consisted of the victims from the second group and six others, together with the results from the other two groups, formed the basis of a concept of psychotherapy for torture victims.

Exposure to extreme situations may cause physical and psychological sequelae. This has been abundantly documented for former concentration camp prisoners (Kral *et al*, 1967; Strom, 1968; Nefzger, 1970; Thygesen *et al*, 1970; Beebe, 1975) and naval personnel (Askewold, 1980) after the Second World War. Similar findings have been reported with hostages (Eitinger & Weisaeth, 1980).

Amnesty International medical groups have described many such complaints in torture victims from a number of countries (Amnesty International, 1977, 1978, 1980, 1981; Randall *et al*, 1986). The initial aim was to provide proof, from the point of view of forensic medicine, of the existence of torture. The same reports also provide evidence that the sequelae of the clandestine torture of today are of the same magnitude as those reported for concentration camp victims. Furthermore, it can be inferred that there is little or no tendency to spontaneous recovery. This topic is therefore a concern of the medical and psychological professions.

Different views upon the aetiology of the sequelae of concentration camp survivors are reflected in the different terminology of the various syndromes (Strom, 1968; Thygesen *et al*, 1970; Trautman, 1964; Lederer, 1965; Krystal, 1968; Niederland, 1968; Hoppe, 1971). The discrepancy between the American psychodynamic views and the European more organic oriented explanations may be due to the fact that highly selected but very different groups were investigated (Eitinger 1986).

As pointed out by Lederer (1965), classical German psychiatry stated that life experiences, no matter how severe provided they do not inflict organic brain damage, can never cause lasting symptoms or alteration of personality. A similar

point of view was alleged by Wolff (1960) in his paper on the conduct of prisoners-of-war. The interpretation by laymen may be the same. Also, victims of torture may consider themselves responsible when mental disability results from the cruelty. According to this hypothesis, psychological sequelae arise from a constitutional susceptibility, and psychological complaints can be seen as wishful reactions with the aim of maximal gain. The admission of psychological sequelae may thus imply a stigma, and the symptoms may be denied for years without motivation for treatment. Klein (1974) described the same therapeutic paradox in his work with survivors of the concentration camps.

On the other hand, Trautman (1964) stated that human beings placed under extreme and prolonged existential stress, fear and anxiety undergo an extensive adaptive change of mental and biological functioning. This point of view is supported by Bettelheim (1943). Adaptive change in psychiatric patients submitted to an authoritarian institution is described by Goffman (1961). Trautman's conclusion was that psychological sequelae should be seen as meaningful conditioned reactions for a sound and forceful constitution that make survival possible in a very pathological situation.

In this study the various psychological methods of torture and the long-term neuropsychological symptoms are evaluated to assess the adaptation of torture victims and to establish a concept of psychotherapy for them.

Method

This study is based on an analysis of three different groups of torture victims. The term 'torture' is used in accordance

with the definition of the World Medical Assembly (The Declaration of Tokyo, 1975): the deliberate, systematic or wanton infliction of physical or mental suffering by one or more persons acting alone or on the orders of any authority, to force another person to yield information, to make a confession, or for any other reason.

Group 1. Two hundred case reports from Amnesty International medical groups, based on semi-structured interviews and medical examination of torture victims, formed the background for a systematic outline of the psychological methods of torture. All reported methods of torture were analysed with the aim of obtaining a more general knowledge of the actual procedures and their main impact on the victims. Ninety-five percent of the victims were males, and most of them were exiles at the time of study. The group consisted of victims from Latin-America, Europe, Africa and Asia.

Group 2. Since 1981 a large number of torture victims have been examined thoroughly at the Department of Neurology, Rigshospitalet, Copenhagen. Among them were 24 victims from different Latin-American countries. Long-term neuropsychological complaints from this group were analysed with regard to time since first exposure to torture. Ten of the victims had previously been examined by the Amnesty International Danish Medical Group using the same semi-structured interview as in the present study; the complaints of these victims were used as the basis for a follow-up comparison. All victims were males and exiles. All were in perfect health before exposure to torture.

Group 3. This group consisted of the 24 victims from group 2 plus four European and two African victims. Twenty-nine of the victims were males, and most of them were exiles at the time of study. The method of psychotherapy has been worked out from observations and systematic notes made by the authors during 425 in-depth interviews with these 30 torture victims since 1981.

All of the victims in this study were selected, and no control group was included. The victims examined by Amnesty International medical groups volunteered for medical investigations within the scope of forensic medicine, epidemiology, and clinical nosography. The victims examined by the authors volunteered for an in-depth investigation of physical and psychological sequelae to torture with the aim of establishing rehabilitation programmes.

Results

Psychological methods of torture

Although an over-simplification, the procedures can be divided into two types: the weakening techniques and the personality-destroying techniques. Common to the methods of the former is teaching the victims to be helpless and creating exhaustion. The latter methods are characterised by the induction of guilt, fear and loss of self-esteem.

Deprivation

Deprivation is defined according to Schultz (1965). A variety of procedures have been used to reduce environmental stimulation, and as a rule the victim is submitted to

different forms of deprivation at the same time. During deprivation the victim suffers from cognitive disturbances, disorientation in time and space, difficulty in concentration, poor memory, and in some cases psychotic reactions with hallucinations. Long-term sequelae may be weakening of identity and emotional instability.

Constraint techniques

These are defined as psychological techniques which force experiences and stimuli upon victims which are totally foreign and harmful to their personality and conduct. Victims are submitted to a detailed set of regulations and rules, resulting in close supervision where everything (including completely insignificant details) is controlled. Violation of the rules (either real or supposed) is used as an excuse to punish the 'offending' victim. The victims are exposed to all kinds of humiliations, which force them to see their dignity and identity completely destroyed.

Induction of incongruent actions, i.e. methods which bring the victims into situations of impossible choices and consequently to act or react against their ideology and ethics, seems central to all psychological torture. Included are methods with the classical aim of yielding information, but it should be emphasised that in many cases this is a sham-purpose, with the real goal being to destroy the personality of the victim. The technique may be slow and progressive, starting by letting the victim admit insignificant or diffuse items; gradually, more specific information is disclosed. When the victim eventually breaks down he enters a vicious circle of guilt followed by repentance, which cannot be expiated. Other methods are mock-executions and threats of torturing family or friends if the victim does not fulfil various demands. African victims have reported violation of taboos as a method of torture.

The effects of the constraint techniques are multiple and long-lasting. The methods represent deep attacks on the identity and dignity of the victim, and they are closely linked with guilt, fear, loss of self-esteem, and repentance. During torture and imprisonment the aggressions of the victim are by necessity turned inwards, and there is no possibility of adequately expressing emotions. As time goes by the events may to an extent be repressed, leaving a feeling of discontinuity in the victim's personality. Some of the experiences are remembered as profound humiliations. Other experiences become an unconscious source of suffering due to a feeling of guilt.

Communication techniques

There are a variety of verbal and non-verbal communication techniques, with varying aims. Counter-effect technique, for example, is defined as torture which seems meaningless to the victims and which continues whether or not the victim co-operates. Any response from the victim becomes the pretext for renewed or continuous torture. The victim's aggressiveness and ability to fight back gradually decrease, and thus the victim learns to be totally helpless.

Double-bind techniques are frequently abused as torture. The term 'double-bind' originates from Bateson's hypoth-

esis about schizophrenia. Victims are exposed to many ambiguous situations and contradictory messages, which induce confusion. Seemingly nice suggestions by the torturers soon turn out to be false, and promises to victims are broken in the next moment. A well-known technique involves the use of a friendly and a unfriendly interrogator. After violent torture carried out by torturers of low rank, the victim is interrogated by an officer of high rank in an ambience of tranquillity and polite conversation. Simultaneously, the officer intimates that he would hate very much to have to hand over the victim for further violence. In a situation of deep distress it is difficult for the victim to resist and not to grasp a 'helping' hand.

Conditioning technique was first described by Pavlov in his experiments with dogs, and the principle is to induce desired reactions or behaviour by certain conditions. As torture, the conditioning techniques are abused to adapt the victims to various demands.

Abuse of pharmacology and psychiatric institutions

There are many examples of these kinds of abuses as methods of torture. Such methods imply the abuse of the medical and psychological professions. The goals are various: profound mental destruction, punishment, creation of anxiety and psychological disturbances, causation of pain, immobilising victims, and yielding information. The psychopharmacological drugs abused either blunt the senses (neuroleptics or sedatives) or are psychotropics, including hallucinogens. Neuromuscular blocking agents (curare compounds) are abused to paralyse fully-awake subjects, which induces total panic due to the impossibility of breathing, and the victims are not offered oxygen until the point of suffocation. If victims are defined as 'mentally ill' they can be admitted to psychiatric institutions and submitted to various 'treatments' against their will, including psychopharmacological drugs.

Physical methods

Physical methods of torture are administered by physical means, but their main impact is of a psychological nature. Because of the acute and chronic pain evoked by such torture, one of the sequelae may be a distortion of the body image.

Long-term neuropsychological symptoms

The investigation of the 24 torture victims in group 2 was carried out a median of 9.5 years (range: 7.5–11.5) after the first exposure to torture. The median age of the victims was 38 years (range: 28–42). The previous examination of 10 of the victims had been performed a median of 3.9 years earlier (range: 2.5–5.5).

Table I shows the frequency of neuropsychological complaints, arranged in decreasing order. There are no obvious differences in symptoms at the two examinations; nor are there if the 10 victims previously examined are compared only with themselves (not shown in the table). The investigation thus provides evidence of a poor tendency to spontaneous recovery. The number of victims is inadequate for any statistical evaluation; the quantitative aspects of the symptoms will not be further described.

TABLE I
Neuro-psychological complaints

<i>Complaints¹</i>	<i>Present study (n=24)</i>		<i>Previous examination (n=10)</i>	
	<i>%</i>	<i>n</i>	<i>%</i>	<i>n</i>
Sleep disturbances	83	20	60	6
Headache	79	19	80	8
Impaired memory	79	19	60	6
Impaired concentration	75	18	60	6
Fatigue	75	18	60	6
Nightmares	71	17	60	6
Fear/anxiety	50	12	40	4
Impaired hearing	38	9	40	4
Social withdrawal	38	9	50	5
Vertigo	33	8	20	2
Sexual disturbances	21	5	20	2
Tremor/shaking	13	3	20	3

1 Five (21%) of the victims investigated in our study suffered from guilt. This complaint was not systematically asked for.

The complaints have rather particular qualitative characteristics. Perhaps surprisingly, spontaneous hate, hostility, and aggression have not been a common feature. The victim's feelings about the torturers are reflected by the typical statement that the torturers should be prosecuted in accordance with democratic laws. During interviews examiners can sense the victim's inward-turning aggression. In spite of all the suffering and sacrifice, victims often claim that the struggle for social justice will never cease. Much emphasis is laid upon the victim's further education and work and the welfare of the family. We have not in any single case observed that victims identify themselves with the aggressor (the 'Stockholm syndrome', Eitinger & Weisaeth, 1980).

Unlike neurosis, the anxiety of the victims is associated with conscious memories, tentatively but never really fully repressed. The anxiety is evoked by sensory, auditory or visual signals or experiences symbolising torture and persecution. The victims react immediately to the 'signal' and are overwhelmed by panic before they have had time to think. To the observer it is clear that these victims are living on an unconscious alert, as if the previous danger was still present. This panic reaction can be evoked by such things as an automobile door slamming, the sound of a siren, or the sight of a soldier or policeman in uniform. Evoking experiences may be such things as a visit to a small room or lift (the cell is recalled), admittance to hospital and confrontation with equipment or investigations resembling the torture situations (blood samples, ECG, X-ray, etc.), or taking an examination (the interrogation centre is recalled). The same may be the case when a therapist, social worker, employer or any 'authority' addresses the victim in a somewhat unfriendly manner (the person is immediately identified with the torturer).

Sleep disturbances are frequent. One of the main reasons

is a repetitive reliving of the same torture experiences during nightmares in which the subject is trying to escape but is helpless, without means of protecting himself and also without counter-aggression. In the conscious memories of the torture, the victim is aware that the aggression had by necessity to be repressed; in the nightmare, however, there is no attempt at defence, and this may be one of the reasons why these nightly experiences are so evocative of extreme anxiety.

Many victims allege impaired memory and difficulty in concentration. During in-depth interviews we have made no observations consistent with dementia, nor observed progression in the alleged cognitive malfunction. In some cases the behaviour may be marked by decreased vigilance and resistance to taking an interest in outside activities. In spite of the victim's 'poor' memory the experiences of torture are remembered to the smallest detail.

Depression is another common feature. Some victims are less anxious when more depressed and vice versa. Loss of self-esteem and a feeling of guilt are often linked, and during the in-depth interviews these complaints can be traced back to certain experiences during torture. Fatigue, emotional instability, and social withdrawal may be considered as consequences of the other symptoms. Sexual disturbances have not yet been studied in detail.

Psychosomatic complaints are frequent. Chronic diffuse headache, muscular tensions, fear of having heart disease, and dyspeptic symptoms are daily experiences for most victims.

Concept of psychotherapy

The course of psychotherapy for torture victims is divided into 3 phases. The first phase is mainly cognitive, with the aim of bringing about a detailed insight into what really happened and thereby generalising the personal suffering. The second phase is more emotive, with emphasis on a reconstruction of the most harmful experiences gradually bringing the victim in contact with repressed feelings. During the reliving of the feelings the victim has an opportunity to realise that his conduct was not unique, but predictable. In this context the victim must also work with feelings which were naturally present, but which could not be expressed at that time, especially the aggression. The third and final phase serves to re-establish reality by offering the victim a way out of the seemingly endless traumatic state induced by the torture. The principle is to interrupt different vicious circles and to transform much of the energy from the previous fixed role of a victim into creativity, enabling a more dynamic course of life.

To illustrate the method we present a case history. Certain details have been changed to avoid recognition.

Case History

The victim is a 27 year-old Latin-American male. He grew up in a safe and economically solid home together with four siblings. After school he soon qualified as a skilled worker. The victim was considered as a strong person by those who knew him, and he has always reacted against social injustice. There was no previous history of neuro-

psychological problems, and he was in perfect health before exposure to torture. His first imprisonment, at the age of 18, lasted 7 months. The second imprisonment took place 2 years later, and lasted 18 months. On no occasion was he under any charge or put on trial. The victim was exiled after release from the second imprisonment.

During the first imprisonment he was submitted to solitary confinement, placed in a small dark cell without a toilet and almost without food and drink. The warders were extremely brutal, and he was repeatedly beaten up, suspended, whipped and threatened. When he started bleeding, salt was poured on the wounds. His head was battered against the wall until he lost consciousness. This was repeated twice per day during the first 3 weeks. He was sexually abused twice.

During the second imprisonment he was placed in a small room in a cellar, without light or toilet, and with only a dirty blanket for cover. For one month he was exposed daily to electric torture. Current was applied to all parts of his body, but particularly to his testicles, teeth, tongue, and ear-lobes. Several times his head was held in a bucket with water containing vomit and urine until the point of suffocation. Three times he was exposed to mock-executions. Several times he was forced to see other prisoners being tortured, particularly by different kinds of sexual torture.

Five years after release the victim was still suffering from the same symptoms as immediately after torture. He was distrustful and despairing, showing severe depressive features with suicidal thoughts. Panic was evoked by abrupt sounds or when he was confronted with authorities. Chronic headache, impaired hearing in the left ear, poor memory and social withdrawal were other important features. Apathy and fatigue were the consequences of difficulty in falling asleep and interrupted sleep with frequent nightmares.

The victim was offered psychotherapy in combination with a simultaneous physical pain-relieving therapy. The cognitive phase took place during ten in-depth interviews, three per week. When mutual trust was established the therapy proceeded by cognitively reconstructing the harmful experiences. If the victim resisted a thorough description of the experiences in prison, they were approached instead by way of his nightmares. The cognitive context was stressed, consisting of information about the most important goal of the torture: to destroy the personality of the victim. During the conversations, otherwise inexplicable humiliations were viewed within a meaningful relationship, rendering it possible to generalise the personal suffering. The victim felt relief after every interview. There were emotional fluctuations at the end of the first phase. The victim was joyful for some days, making subsequent dives into depression even more painful.

The emotive phase consisted of ten in-depth interviews during a 3-week period. The victim was still suffering from violent nightmares, which provoked intense fear and made the victim depressed for a long time. The most frequent nightmare concerned a mock-execution to which he had been submitted during the second imprisonment. The torturer had placed a bottle on his head, whereupon the

bottle was shot to pieces. The victim awoke abruptly at the moment of dreaming of the shot, totally in panic and helpless. During the victim's detailed description of this nightmare he realised that the most panic-evoking part was his own lack of aggression. Three interviews later he related that the previous night he had experienced the same nightmare. This time, however, he had fixed the eyes of the torturer, who immediately looked at him with respect and was not laughing as usual. At that moment he felt a great strength. During the interview he remembered that for several days after the mock-execution he could hardly believe he was alive. He thus found himself in a situation where the following electric torture was a confirmation of life. Later he felt that he could no longer stand to be tortured constantly, and he begged the torturers to kill him. For some time great care was taken that the victim did not lose consciousness during torture. Thus he found himself in a situation where being forced to live was in itself torture. In subsequent in-depth interviews the method of impossible choices was thoroughly explained to him. It was a great relief for the victim to realise that no matter what he had done, he would have felt that he had made the wrong choice.

When the victim proceeded to the third phase, the re-establishment of reality, there were changing moods from almost euphoria one week to depression the next. On some occasions there were relapses of nightmares, which were evoked by bad news from his country. The interviews took place with intervals of 1–2 weeks. Gradually the victim developed insight into the various vicious circles, and was soon able to circumvent the mechanisms. The victim started helping friends and other victims. He was then, for the first time since release, able to deal constructively with the school problems of his children. Before the therapy his wife had justly accused him of being evasive when confronted with these problems. The victim started supplementary education. One result was that it was no longer necessary for his wife to shoulder a heavy burden, and she could then tell us that she was a torture victim herself with an urgent need for psychotherapy.

After 28 in-depth interviews over 4 months the therapy was stopped, with an agreement that the victim could apply again if he wanted further treatment. During a follow-up period of two years there has been no subjective need for resumption of psychotherapy. At the end of that period an in-depth interview confirmed that further treatment was unnecessary.

Other cases

The psychotherapy was performed according to the same principles in the other 29 cases. A median number of 22 in-depth interviews were needed (range: 10–52). The whole procedure was carried out during a median period of 12 weeks (range: 4–19). In our hands the method has been successful, with only a few relapses during a median follow-up period of 11 months (range: 6–36 months). When supplementary psychotherapy was needed, 2–3 sessions were generally sufficient. It must be emphasised that during the period of psychotherapy a pain-relieving physical therapy was simultaneously carried out.

Discussion

The psychological methods of torture listed above provide evidence that normal psychological mechanisms are distorted by torture and that torture is performed in a systematic and scientific way. Destruction of the personality of the individual victim is probably one of the most important goals of torture.

An important biological character of the brain is the great capacity for adaptation. One would expect the victims to adapt to the atrocious circumstances with the aim of preserving as much as possible of their personality, including norms, ideology and emotional reactions. There are two other 'possibilities': biological or psychological death—suicide or psychosis. Bettelheim (1943) observed the adaptation procedure of concentration camp prisoners, and Goffman (1961) described adaptation to an authoritarian institution. Between them they describe four coping strategies: intransigence, re-establishment, introversion, and conversion. Eitinger (1974) found during interviews with more than 2000 concentration camp survivors that one of the most important coping mechanisms was the conservation of the ability to make at least a few of one's own decisions. Those who were able to retain their system of values and personality more or less intact were the most fortunate, i.e. people who could practice and show interest in others (doctors, nurses, priests, social workers, etc.). Identification with the aggressor was used by some, but this mechanism led to deep pathological changes in the personality. A few had maintained an attitude of a detached observer, but this mechanism was unsuccessful due to the loss of contact with others and with reality. Eitinger's conclusion was that survivors who mobilised an active and more or less conscious coping mechanism had fewer psychiatric complications than survivors who were completely isolated, remained passive, or ascribed survival to mere luck.

Short-term neuropsychological complaints provide evidence that torture victims adapt to the inhuman conditions (Amnesty International, 1977, 1978, 1980, 1981). The 200 case reports from Amnesty International medical groups in the present study provide no evidence for the use of a strategy of conversion, and an intransigent attitude was rarely maintained for a significant time. This has been confirmed during the 425 in-depth interviews with the third group of torture victims studied, so presumably those mechanisms are rarely used by torture victims. On the other hand, the same two studies provide evidence for a frequent use of a strategy of introversion and re-establishment.

The various physical sequelae reveal the applied physical torture (Genefke & Aalund, 1982). Perhaps more surprisingly, the long-term neuropsychological symptoms (Table I), and particularly the qualitative aspects, are evidence of a long-term adaptation to a victim's role, where previous danger is still experienced or easily evoked. There is thus an element of a faulty accordance with reality. Furthermore, the tendency to spontaneous recovery from the neuropsychological complaints seems poor.

Out of this concept of the psychological sequelae as a result of specific and systematic psychological methods of torture, adaptation to atrocious circumstances, vicious circles of anxiety, guilt, repentance, atonement, and loss of self-esteem, we propose a psychotherapeutical approach to bring the subject out of this victim's role, thus enabling him to restore reality and re-establish a valuable course of life. Forgetting is not the answer; nor is atonement through obsessive remembering.

Psychotherapy for victims of torture requires a thorough reconstruction of the harmful experiences during in-depth interviews, where the methods of torture and the conduct of the victim are evaluated as cause-and-effect situations, not as phenomena. In contrast to classical psychotherapy, the therapy of victims can start as soon as a mutual trust is established, and a preceding period of analysis is unnecessary. In the cognitive phase the victim realises how the torture distorted normal psychological mechanisms, and that the torture was systematic with very particular goals (often different from what the victim thought were the goals). When he realises that these goals were obtained, not because of an inborn weakness, but as a predictable result, the heavy burden of responsibility is justifiably transferred to the torturers.

The cognitive phase is followed by a phase where the most harmful experiences are reconstructed in a more emotional context, enabling the victim to see that there were no options. It is of the utmost importance that the therapist is never identified with the aggressors. The ambience must be characterised by mutual and complete trust. The therapy takes place in an atmosphere of humanity, but with directness and professionalism without unnecessary sentimentality. All procedures which may cause uncontrollable pain and memories are systematically avoided. Precise information about all aspects of the examination and therapy must be given, and the subject must fully understand and accept the procedures. Finally, the therapy must be carried out at a tempo adjusted to the immediate emotional reactions of the subject, and enough space must be allowed between the sessions that the subjects cogni-

tive acceptance and emotional reactions correlate.

Implicitly, the victim realises during the first two phases of the therapy that there is no reason for a general mistrust, and when both a new cognitive and emotional context are achieved the re-establishment of reality is not troublesome.

If a victim has strong repressive mechanisms he is asked to tell his story in chronological order. The victim must describe precisely what he experienced during isolation and the time spent waiting for torture. If these topics (e.g. size of the cell, noises, light, food, toilet facilities, hallucinations) are not probed into, the victim will usually not tell much about them. The victim's reactive pattern often shows itself most clearly when these experiences are described, and this is thus a good starting point for a dialogue about a variety of the victim's psychological mechanisms. The principle of forcing a victim into a conduct totally opposed to his personality and thereby creating guilt and repentance is thoroughly expounded.

During discussions of the situations in which the victim was forced to make a choice, and feels that he made the wrong choice, it can often be proved that no matter what decision was taken the result would have been the same. As a rule the victim has not considered this, and it is a relief to understand how the method of impossible choices has been exploited. Furthermore, it is emphasised that a victim cannot have responsibility for what he did in a situation of utmost physical and psychological pain. The methods are elaborated with the purpose of creating a guilty conscience, and the responsibility lies completely with the repressive system. This principle should be recognised by international law.

By penetrating the most painful experiences the victim has an opportunity to work with them within a new context. During this kind of conversation the victim often realises that he has turned all aggression against himself, even though it should be directed at the torturers. Humiliations must be discussed point for point. Hereby the victim conceives that it is actually the torturers who are degraded by their inhuman conduct. The memories no longer cause the victim the same fear as before, when he eventually comes to understand what was done to him and that he was broken down in a predictable way by torture distorting normal psychological mechanisms.

There are many sources of guilt besides those already mentioned. Some victims consider that they did not help fellows enough during imprisonment, for example, the difficult situation of the family, the many years without education, the present social situation and the very fact of being alive are other examples. It is emphasised that it is the opposite of

egoism and very valuable to mankind that people are willing work for a better society even under very difficult conditions.

The results of the psychotherapy obtained so far have been encouraging. The present study provides evidence that the long-term neuropsychological symptoms may be reversible provided psychotherapy is applied rather than supportive treatment or teaching the victims to cope better. The concept of psychotherapy will be further evolved and evaluated at the International Rehabilitation and Research Center for Torture Victims in Copenhagen.

Rehabilitation of torture victims has to include spouses and children, and indeed the whole social

network of the victims must be taken into consideration. Psychological disability due to exile is also of great significance. However, all of these topics are outside the scope of this paper.

The rehabilitation of a torture victim cannot be considered to have succeeded until the term 'victim' no longer signifies the subject as adapted to this role and instead signifies only a historical event.

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Applied Relaxation Training for Generalised Anxiety and Panic Attacks:

The Efficacy of a Learnt Coping Strategy on Subjective Reports

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The results of applied relaxation training in patients with generalised anxiety and panic attacks are reported. ART was taught during one session, by means of participant demonstration, written instructions, taped instructions, or a combination of all three, with instructions to practise at home. All four methods proved superior to a waiting list control, but there were no differences between the treatment groups. There was some evidence for the non-specific effect of expectancy, but this did not completely explain the treatment effect.

The treatment method of progressive Relaxation Training was designed by Jacobson (1938). During the early development of behaviour therapy it became incorporated into the procedure of systematic desensitisation, as a response incompatible with anxiety (Wolpe, 1958). More recently, however, relaxation has been used (as originally suggested) as a treatment in its own right, and numerous conditions such as hypertension, headaches, insomnia, and anxiety disorders have been shown to be suitable for such treatment (Rachman & Wilson, 1980). Anxiety disorders have been investigated in most detail, and relaxation training is reported to be superior to control procedures. It was typically effective when taught for four or more sessions, when instruction was given in person, and when subjects had control over the progress of treatment (Borkovec & Sides, 1979; Hillenborg & Collins, 1982). Use of a counter-demand condition indicates that the treatment effect of relaxation cannot be explained purely by non-specific effects (Steinmark & Borkovec, 1974; Borkovec *et al*, 1978).

Relaxation training may facilitate fear reduction during exposure treatment of situational anxiety (Gillan & Rachman, 1974), though Glaister (1982) concluded that the fear-reducing effect of relaxation alone was less than that of exposure to the feared stimulus. However, in generalised anxiety states where no precipitating situation can be identified *in vivo* exposure is not possible, and relaxation training may then be indicated for reduction of anxiety. Suinn & Richardson (1971) developed anxiety management training (AMT) for such generalised anxiety conditions: patients were taught to control anxiety through muscle relaxation and mental imagery. Although two studies indicated positive results in

student volunteers (Edie, 1972; Nicholetti, 1972), generalisation to a clinical population may not be valid. However, a recent study (Jannoun *et al*, 1982) reports treatment of 27 out-patients using a modified version of AMT. Patients were allocated to three groups, with a four, six or eight week waiting time. Improvement occurred on all anxiety ratings over the treatment period, and these gains were maintained at three months: significantly greater changes for the groups with less waiting time indicated that the improvement was due to treatment. Furthermore, depression ratings also decreased over this period, and intake of anxiolytic drugs decreased by 60%.

In AMT the therapist assumes a didactic role in helping the patient to acquire a coping skill, and this is in accord with the recent direction of behaviour therapy. However, if the patient is to learn a skill, practice is required. Hillenborg & Collins (1983) demonstrated the superiority of home practice relaxation over no such practice in volunteers, but the same authors (1982) found that only 60% of studies reported any type of home practice, and only 19% assessed compliance. A number of studies indicate that subjects exaggerate compliance, although results correlating subjective and objective compliance with outcome are inconsistent (Hillenborg & Collins, 1983; Hoelscher *et al*, 1984; Barr Taylor *et al*, 1983).

The behaviour literature has been less concerned with aetiology than with the outcome of treatment, but it has been suggested that hyperventilation may play an important role in panic attacks (Lum, 1976; Hibbert, 1984), and breathing control effectively reduces anxiety attacks (Kraft & Hoodguin, 1984; Clark *et al*, 1985). Abdominal-diaphragmatic breathing is enhanced by relaxation, but suppressed

by conditions of emotional strain (Faulkner, 1941); in view of the effect respiratory function can have on other physiological systems, especially cardiovascular function (Grossman, 1983), some training in breathing exercises or control is therefore strongly indicated for anxiety control techniques.

Recently, the move towards psychological treatment of anxiety has been boosted by the increasing recognition of dependence problems associated with benzodiazepines (Ashton, 1984; Lader, 1983; Tyrer *et al.*, 1983) and of the fact that they do not increase the efficacy of behavioural treatments (Grey *et al.*, 1981; Sartory, 1983). This has been especially true in primary care (e.g. Teare Skinner, 1984). The use of such methods as relaxation training by different professions, often simply by handing out taped instructions, has generally increased, and such taped instructions and booklets are now widely available. However, little is known about the effectiveness of such modes of presentation.

The present study was performed to examine a number of questions:

- Would applied relaxation training (ART) be efficacious in generalised anxiety?
- Could ART be taught in a single session and be learnt through home practice?
- Would the mode of instruction differentially affect efficacy?
- Would different types of symptoms be differentially affected?
- If efficacious, could a non-specific effect be responsible for improvement?
- What factors are important in predicting improvement?

Method

Subjects

Fifty consecutive referrals to a district psychology department were randomly allocated to one of four treatment conditions or to a waiting list control group; patients had been referred either from the Department of Psychiatry or directly from general practitioners.

Patients were included in the study if their main presenting problem and referred problem was that of generalised anxiety. This was defined as:

- experiencing panic attacks that were not situationally determined
- experiencing high levels of general anxiety and tension, and complaining of an inability to relax most of the time
- physical symptoms of anxiety being present and a major source of complaint.

Many patients also complained of other problems such as situational anxiety (e.g. agoraphobia) and mild-to-moderate depression; those whose principal complaint was

of situational anxiety or who were at risk of suicide were excluded. Many patients were on psychotropic drugs, but those who thought their medication was no longer producing any improvement were included, and were asked to continue taking their medication. Details of the characteristics of the sample are presented in Table I.

Dependent variables

Self-report instruments. At pre-treatment and post-treatment assessment, patients completed the Symptom Rating Test (SRT) (Kellner & Sheffield, 1969; 1973) and the Epstein-Fenz Anxiety Scale (EFAS) (Fenz & Epstein, 1965; Fenz, 1967). The SRT consists of 30 items, each rated on a four point scale, and provides both a total score and four sub-scales: anxiety depression, somatic symptoms and inadequacy. It is designed to measure distress, and has been shown to discriminate between neurotic and normal subjects, and to change in the expected direction in neurotic patients after treatment (Sheffield & Kellner, 1970). The EFAS consists of 43 items, mostly from the Taylor Manifest Anxiety Scale, each rated on a five point scale. The sub-scales relate to autonomic anxiety, striated muscle tension, and feelings of fear and insecurity. All three sub-scales have been shown to differentiate between normal subjects and psychiatric patients 'diagnosed as anxiety neurotics' (Fenz, 1967).

Benefit gained. At post-treatment assessment, the subjective benefit of treatment was assessed on a three point scale

TABLE I
Sample characteristics

No. of subjects	50
Proportion of females	60%
Mean age: years	40.5 (s.d. 10.8)
Mean duration of symptomatology: years	5.34 (s.d. 5.92)
Marital status	
Single	10%
Married	74%
Other	16%
Unemployed	44%
Source of referral	
Psychiatrist	84%
GP	14%
Self-referral	2%
Taking hypnotics	14%
Taking minor tranquillisers	46%
Taking antidepressants	28%
Taking neuroleptics	4%
Previous psychiatric history	42%
Previous treatment	
None	32%
GP ¹	26%
Psychiatrist	42%

1. Patients who had received psychotropic medication from their general practitioner

(0 = none or minimal benefit, 1 = beneficial, 2 = very beneficial). The patient was asked what effect the treatment had had on his anxiety problem; a rating of 2 was only given if the patient responded spontaneously that the treatment had been very beneficial.

Further treatment. A number of patients needed further treatment, and it was considered unethical to withhold this from patients so that follow-up data could be collected. However, such data from patients not requiring further treatment would be impossible to interpret, due to the sample being highly biased. A measure of further treatment required was used instead, the scale being 0 = further treatment not required; 1 = further instructions on relaxation methods needed only; 2 = further treatment besides relaxation required. Once patients were discharged, they were advised to contact the department if they needed to; classification was made at post-treatment, or if they contacted the department during a minimum 6 months post-discharge period. Since some patients required other treatments for multiple problems, e.g. exposure for situational anxiety, this measure of improvement is conservative.

Subjective compliance. Patients were instructed to practise their exercises at least once a day. If they felt these were helpful, or if they were troubled either by a panic attack or by high levels of tension, then they could practise more frequently. Patients were asked to keep a record, on a provided record sheet, of every time they practised the full exercise procedure, and to estimate their anxiety levels before and after on a scale of 0–100. They were told that the purpose of the record keeping was to see if their anxiety levels decreased after the exercises. Compliance of 100% would be achieved if the exercise routine was practised once a day, and practice in excess of this would be regarded as a behavioural index of perceived usefulness of the exercises.

Procedure

Patients were randomly allocated to one of four treatment groups or to a waiting list control; those in each of the treatment groups were consecutively allocated to either an expectancy or counter-expectancy condition (see below). Patients in the waiting list control group were sent an appointment for approximately six weeks ahead, together with the SRT and EFAS and instructions to complete these (indicating how they had felt over the past week) and to return them in the envelope provided. Assessment of suitability was made both from the referral letter and at interview. Patients in the four ART treatment groups were interviewed, and recruited if suitable for study. They then completed the SRT and EFAS; a further appointment was given for one week later.

Treatment groups

The four ART treatment groups were each instructed by a different method: (a) handout (written instructions); (b) tape (taped instructions); (c) participant demonstration

(verbal instruction and practice); and (d) combined methods (written and taped instructions to take home, with verbal instruction and practice during the session).

All patients received a rationale for applied relaxation training, which emphasised the ability to control physical symptoms through the use of correct breathing and tension release exercises, and the use of imagery to distract attention from anxiety-associated cognitions.

The ART method has the following components: (a) self-monitoring of anxiety levels; (b) correct breathing—monitoring of breathing and the use of exercises to encourage diaphragmatic breathing; (c) progressive muscle relaxation, consisting of tension release exercises; (d) positive mental imagery.

In each of the treatment groups the content of the material was the same but the mode of presentation differed. After the basic rationale, the Handout group received written instructions on the ART exercise routine. Patients in the Tape group were given taped instructions only. Patients in the Participant Demonstration group were given a demonstration of the ART exercise routine in which they participated by following the instructions and model of the therapist. The Combined Treatment Group received the live instruction during the therapy session, as had the Participant Demonstration Group, and were also given written instructions and a tape as in the other two groups.

Expectancy conditions

The ten patients in each treatment group were alternately allocated to either an expectancy or counter-expectancy group. Patients in the former group were told that they should expect continued benefit every time they fully practised the exercise routine, which would increase the more they practised. This point was strongly emphasised. Patients in the counter-expectancy group were told that benefit was slow to become apparent, and that they should not expect to feel any benefits from the exercise routine before the next appointment (post-treatment assessment); they should, however, practise as instructed. Again, these points were strongly emphasised. The counter-expectancy condition was designed to reduce the non-specific effects of the expectancy of improvement from receiving treatment.

Patients were given a further appointment for approximately five weeks later, during which the post-treatment assessment took place. Four patients failed to attend for this assessment (two from the Handout group and two from the Combined Treatment group).

Statistical analysis

Results were analysed using a standard statistical package. Results are presented in terms of percentages or of means and standard deviations. Bivariate comparisons are presented in terms of analysis of variance, while outcome is described in terms of analysis of variance of paired *t*-tests. Correlation coefficients (*r*) between variables are also presented. Attempts were made to incorporate multiple regression techniques in the prediction of the various outcome measures. To do this, the ϵ^2 coefficient was calculated; this can be interpreted as the proportion of the total

variability in the dependent variable that can be accounted for by knowing the value of the independent variable (Nie *et al.*, 1980).

Results

Success of the experimental design

There were no differences across the groups in age, marital or employment status, duration of symptomatology, source of referral, or medication usage (whether minor tranquilisers, antidepressants, hypnotics, or neuroleptics). By chance, there were no males in the demonstration group, leading to a gender imbalance, but since there was no gender difference on any of the outcome measures this did not seem to merit further consideration. As would be expected, the average time of treatment sessions was less for the Handout and Tape groups than for the Demonstration and Combined groups.

There was no difference on initial psychometric scores between the expectancy conditions, demonstrating that random allocation had been successful. At outcome, the counter-expectancy group showed a trend towards high SRT total scores, ($\epsilon^2 = 9.9\%$, $P < 0.07$) and SRT depression subscale scores ($\epsilon^2 = 10.7\%$, $P < 0.06$). A clear difference was evident on the SRT anxiety sub-scale ($\epsilon^2 = 13.9\%$, $P < 0.03$). No difference was evident on the other scales.

Association between variables

There was a significant negative correlation between the psychologist's assessment of further treatment need and the patient's self-rating of perceived benefit ($r = -0.6$, $P < 0.001$). A complete association between the two measures would not be expected, since although a patient may perceive benefit for his generalised anxiety he may still need treatment for other problems such as situational anxiety or depression. There are also significant positive correlations between further treatment need and SRT (total score) ($r = +0.33$, $P < 0.05$), SRT (anxiety) ($r = +0.43$, $P < 0.05$) and insecurity ($r = +0.38$, $P < 0.05$) at post-test. There were no significant correlations with pre-test measures. There were significant negative correlations between perceived benefit and SRT (total score) ($r = -0.38$, $P < 0.05$), SRT (anxiety) ($r = -0.42$, $P < 0.01$), SRT (depression) ($r = -0.37$, $P < 0.05$), and SRT (inadequacy) ($r = -0.39$, $P < 0.02$). There were no significant correlations with pre-test measures: this indicates a high agreement between the psychologist and patient on improvement. The agreement between the interview ratings and the standardised self-rating tests is significant, but fails to explain a considerable amount of the variance.

Outcome of study

Perceived benefit. Approximately 70% of the treated groups reported at least some benefit. The difference among the four treatment groups was minimal ($\epsilon^2 = 2.0\%$, NS) (Table II).

Further treatment needs. The therapist considered that about 60% of subjects were in need of further treatment;

differences among the treated groups were not significant ($\epsilon^2 = 7.4\%$, NS) (Table II).

Self-reported compliance. One third of the group had complied with the instructions on 14 days or less; about 25% practised on 29 days or more. The mean number of days practice was 23.1 (s.d. 15.7). There were no significant differences between treatment groups. Compliance was also calculated as the percentage of times practised, where once per day would indicate 100% compliance; 24% of patients had a compliance of 76% or more, and there was a mean compliance of 68% s.d. 42.0, range 0–200%. There were no significant differences between groups ($\epsilon^2 = 4.9\%$, NS). Compliance was significantly correlated to perceived benefit ($r = +0.68$, $P < 0.001$) and significantly negatively correlated to further treatment need ($r = -0.40$, $P < 0.02$).

Psychometric scores. No differences were found between the pre-treatment psychometric scores for the different treatment groups, confirming that random allocation had not by chance produced any pre-treatment psychometric differences. Results for all patients are given in Table III. Significant differences were found between pre-treatment and post-treatment on SRT (total score), SRT (somatic), and the three EFAS subscales and approached significance on SRT (anxiety). No differences were found in the Waiting List group.

Some psychometric differences among the treatment groups are evident. The Handout group showed a reduction only in autonomic anxiety ($t = 2.61$, $P < 0.05$). The only clear difference evident in the Tape group was a reduction in insecurity ($t = 2.29$, $P < 0.05$), although non-significant trends were noted in SRT (total) ($t = 2.00$), SRT (anxiety) ($t = 2.06$), SRT (somatic) ($t = 2.15$), and autonomic anxiety ($t = 2.05$). The Participant Demonstration group showed a significant reduction in autonomic anxiety ($t = 2.37$, $P < 0.05$), as did the Combined group ($t = 3.27$, $P < 0.02$); the latter group also showed a significant reduction in insecurity ($t = 2.08$, $P < 0.05$).

Expectancy. There were no significant differences between the expectancy conditions on any of the four major treatment variables, so that it was unnecessary to consider expectancy as a covariate in the rest of the analyses.

TABLE II
Results of treatment

Perceived benefit	
No benefit	30.6%
Some benefit	50.0%
Great benefit	19.4%
Further treatment	
None	38.9%
Relaxation therapy	16.7%
Other behaviour therapy	44.4%

TABLE III
Effect of treatment

Scale	All treatment groups (n=36)			P <	Waiting list (n=10) ¹		
	Pre-treatment score	Post-treatment score	t		Pre-treatment score	Post-treatment score	t
SRT							
Total	60.56	55.33	2.32	0.03	65.00	63.20	0.59
Anxiety	18.50	16.86	2.01	0.06	19.20	18.50	0.65
Depression	15.11	14.17	1.75	NS	17.80	16.40	1.83
Somatic	13.22	11.75	2.60	0.02	13.60	13.40	0.22
Inadequacy	13.89	13.03	1.36	NS	15.10	15.10	0.00
Epstein-Fenz							
Autonomic anxiety	38.39	33.81	4.65	0.001	36.50	36.80	0.20
Muscle tension	34.47	31.92	2.55	0.02	33.70	33.10	0.03
Insecurity	50.14	45.67	3.51	0.002	49.50	50.30	0.42

1. P not significant for any scale

Conclusion

In general, treatment groups improved significantly more than the Waiting List control group, but there were few differences among the different types of treatment. Random allocation to experimental and control groups proved successful. The initial demographic and clinical variables were unrelated to outcome, but initial psychometric scores, unsurprisingly, were highly correlated with final levels. Manipulation of the expectancy condition was influential in terms of differences on some of the psychometric measures at outcome, but showed no relationship with perceived benefit, further treatment need, or the two measures of compliance.

Discussion

The results will initially be discussed in terms of the questions previously formulated.

(a) The results indicate that ART does produce short-term improvements in patients with generalised anxiety, compared with a waiting list control group (who showed no improvement).

(b) It does appear to be possible to teach a therapeutically effective self-help skill in a single session followed by home practice. Approximately 70% of patients reported having received some benefit from the single session and home practice, and nearly 20% reported receiving great benefit. However, approximately 60% required some form of further intervention. Although reported compliance with home practice was extremely variable, it appeared to be associated both with the patients' and the psychologist's assessment of improvement; however,

correlations do not allow an interpretation of the direction of this relationship. This subjective rate of compliance is approximately equal to the 71% found by Barr Taylor *et al* (1983) with hypertensive patients.

(c) The type of instruction given did not significantly effect outcome. There was no consistent superiority in any of these four methods of presentation.

(d) Measures that reflected the physical and somatic aspects of anxiety, e.g. the SRT anxiety and somatic scales and the EFAS, responded best to treatment. There was no significant improvement in the SRT inadequacy scale, which represents a more cognitive dimension of anxiety, and no evidence that ART had any effect on depression. Hence there is a differential treatment effect on types of symptoms, reflecting an improvement in physical and somatic symptoms, but with little generalisation to cognitive and mood-related symptoms. This is also reflected when the four treatment groups are examined separately. Surprisingly, the muscle tension scale did not show significant improvement. It would have been expected that a treatment method that included progressive muscle relaxation would have an effect on reports of striated muscle tension, but this was not demonstrated.

(e) There was evidence of a non-specific effect in treatment: two scales of the SRT showed a significant difference, and two other scales approached significance. The results indicated a better outcome for patients who had an expectancy of improvement with practice. These differences were not related to

any of the treatment groups, and can therefore be thought of as a generalised expectancy effect. However, expectancy of improvement was not related to subjective estimates of benefit, nor to the psychologist's estimate of further treatment needed. Nor was there a relationship between expectancy and compliance, so the effect of expectancy was not mediated by amount of practice. The evidence suggests that non-specific effects manipulated by the therapist do contribute to treatment outcome, but that not all improvement can be explained solely by expectancy manipulation.

(f) Attempts to identify predictors of improvement were unsuccessful. It was interesting that different types of psychotropic medication did not affect outcome.

This was a clinical trial of a method of anxiety reduction. The subjects, 42% of whom had a previous psychiatric history, included patients who had experienced symptoms for long periods (mean 5.34 years, s.d. 5.92, range 2.4 months to 18 years). Only 30% of those who received ART were not taking psychotropic medication. Although this population included many patients with a chronic anxiety problem who were not improving on other treatment regimes, one session during which instruction in ART was given produced small but significant improvements. Of these patients, 39% needed no further treatment, and a further 17% needed only further instruction in relaxation. Although 44% needed further behavioural treatment, some had also presented with other clinical problems.

In a trial of AMT with a similar clinical population of generalised anxiety patients, Jannoun *et al* (1982)

demonstrated the superiority of AMT over a no-treatment waiting list period; their treatment method can be thought of as most comparable to the combined treatment group in the present study, although their patients received further instruction in relaxation as part of the procedure. The results of the present study support those of Jannoun *et al* in demonstrating the usefulness of anxiety management-type techniques in generalised anxiety. No evidence was found in the present study to demonstrate a consistent reduction in depression.

In contrast to previous studies on relaxation (Borkovec & Sides, 1979; Hillenberg & Collins, 1982), we found no advantage in live presentation of relaxation methods during the treatment session. This may be due to the fact that only one treatment session was given; if treatment had been extended over a number of sessions it is possible such differences would have become apparent. However, since there is no advantage in a single session live participant demonstration, giving taped or written instructions may be desirable, due to a significant economy of time. If patients do not improve, then more intensive efforts can be employed. The fact that the patient's expectancy of improvement—manipulated by the therapist—can affect outcome signifies the importance of presentation of the treatment rationale, and the method employed is probably crucial.

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Disturbance of Water and Sodium in a Manic-Depressive Illness

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Specific questioning and frequent observation of a 69 year-old woman with cyclic bipolar manic-depressive illness showed that she had disturbances of thirst, appetite, bowel and bladder function and dramatic changes in body weight, in association with different phases of her mental illness. Examination of one manic phase under constant diet and inpatient control showed cardiovascular changes, sodium retention, body weight gain, with raised aldosterone secretion but steady vasopressin. There appears to be a sub-group of manic-depressive patients with evidence of disturbed hypothalamic functions as part of their mental illness, as shown particularly by changes in water and electrolyte metabolism.

Recent interest in the possible role of vasopressin in affective illness (Gold *et al.*, 1984) revives the need to examine water and electrolyte metabolism in manic-depressive patients.

The following case was selected for longitudinal study because in an earlier brief hospital admission her weight had suddenly risen in a few days by 3 Kg. during a manic attack, although this rise had been ascribed at that time to the drug therapy. The first study followed her through one manic-depressive cycle to confirm that her fluid balance varied with mental state independently of any drugs; the second examined a later manic attack in more detail under controlled conditions, to establish the nature of the disturbance in water and electrolyte metabolism.

The case

A former physiotherapist married to a doctor who was a keen clinical observer began at age 54 to have recurrent cycles of manic-depressive disorder lasting 45-70 days, as a consequence of which she had numerous hospital admissions and treatments, including ECT for the depressive phase on a number of occasions, and trimipramine, chlorpromazine, lithium carbonate and other drugs. At the times of study this illness had been running for 15-16 years.

The family history was positive: her father and his brother had both suffered recurrent depressions, and their father was said to have been a manic-depressive. The patient's younger sister suffered depressions following the menopause.

The patient herself had had episodes of "losing grip" lasting a week or two at ages 17 and 22, episodes of depression and tearfulness at ages 37, 38 and (puerperally) at 40.

On the physical side she had had appendicectomy at 26, left mastectomy for carcinoma at 54 and cholecystectomy (many small stones) the following year.

Description of the attacks (from husband and patient)

The cycles began with 5-7 days of gastric disturbance, flatulence, occasional epigastric pain, and loose stools. She became uncommunicative, began to feel depressed in mood, slowed down in thought and movement, had difficulty making up her mind, could not remember how to perform simple kitchen tasks or even to wash herself. These depressive phases slowly waxed and waned over about 3-4 weeks.

They were followed at once by hypomanic phases, each heralded by a sudden desire, continuing for two weeks, to eat chocolate or honey. She became constipated, was awake and active all night or at least until 3 a.m., became thirsty (up to 3 L. per day) and developed some urinary urgency and occasional incontinence. She was busy, talkative, sociable, unpredictable, an overspender, generous, broke things easily, was emotional, sexually alive, and felt happy. This phase waxed and waned over 3 weeks and was then followed by about three weeks of normality before the mood cycle started round again: cycle length about 70 days, noted, when in hospital, from age 62 at latest.

Study I

The patient was admitted for observation over seven weeks of a complete mood cycle. She was just two weeks into a depressive episode, with slowed movement and laboured speech, monotonous and without spontaneity, mostly about the hopelessness of her condition. She had been taking lithium carbonate ('Priadel') 1200 mg each night, and diazepam 5 mg at night and thrice daily for a long time, and it was decided to hold these constant through the whole admission rather than complicate the phenomena in the limited available time by introducing a withdrawal and wash-out period. The plasma lithium at 9 a.m. on many occasions from April 1 until mid-May was always 1.0 or 1.1 mmol/L., but on May 13 was 0.9, on May 15 0.6 mmol/L. (hypomanic phase ending, weight dropping) then returned to the 1.0 level, rising on June 3-20 to 1.5 mmol/L. at a time of great weight loss, before settling again to 1.0 mmol/L.

No attempt was made to control the patient's food intake, except that she ate only the official ward meals provided. Her fluid intake was measured as far as possible, and her urine was collected as a series of 24-hour specimens, which were analysed for creatinine (and for Na, K, Ca, Mg) by autoanalyser or absorption spectrometry. Daily urinary creatinine measurement is often a rough constant at about 20 mg/kg body-weight of a patient, and in this woman (weight about 56 kg) was very often 0.9–1.1 g per day, but at other times between 0.7 and 1.4 g due either to errors in timing consecutive urine specimens or to fluctuations in renal blood flow.

During the manic phase urine samples were taken on several occasions for aldosterone and vasopressin radioimmunoassay, very kindly carried out by Dr. J. D. H. Slater and his colleagues at the Middlesex Hospital (Jowett *et al.* 1973).

Plasma samples were taken at intervals for packed cell volume by carefully standardised haematocrit, as a measure of blood hydration, and for plasma Na and K and serum albumin by standard methods.

The patient was weighed daily before 8 a.m. in her night-dress, standardised as described by Crammer (1957, 1983).

Results

Figure 1 shows a depressive episode which began March 14 and ended about April 21, or after five weeks. The hypomanic phase began May 1, and ended about May 14 (two weeks), to be followed by a slow onset of depression from about May 22. Thus the complete cycle appeared to be March 14 to May 22, or 69 days long, which agreed with previous reports in this patient. The daily body weight was far from steady (unlike that of a normal person) and showed at least four changes greater than the maximum gain per day (250 g) expected from forced feeding, or maximum loss per day (500 g) from complete starvation without extra sweating (Crammer, 1983):

- Phase 1: April 9–17, weight rises from 53 to 57 kg, or average 500 g per day while depression lessens: fluid retention.
- Phase 2: April 18–20, weight falls 57 to 55 kg, a loss of 1.5 kg in two days: fluid loss.
- Phase 3: May 2–15 weight rises steadily 54.5 kg to 59 kg, 350 g per day: fluid retention. While hypomanic, this was accompanied by complaint of thirst, high fluid intake, and a 24-hour urine output of 5 litres.
- Phase 4: May 15–24, weight falls 59 to 56.5 kg then levels off at 57 kg for a week.
- Phase 5: May 29 to June 1, fall from 57 to 54 kg at a time of mounting depression.

Weight gain due to fluid retention is accompanied usually by sodium retention (maintaining osmolarity), and weight loss due to extracellular fluid loss is accompanied by a loss of sodium in urine. Table I shows the results of daily urine analyses in the different phases, with mean daily excretion in each phase. The values must be considered with caution, since food intake, which was not regulated, can alter these values. However the main

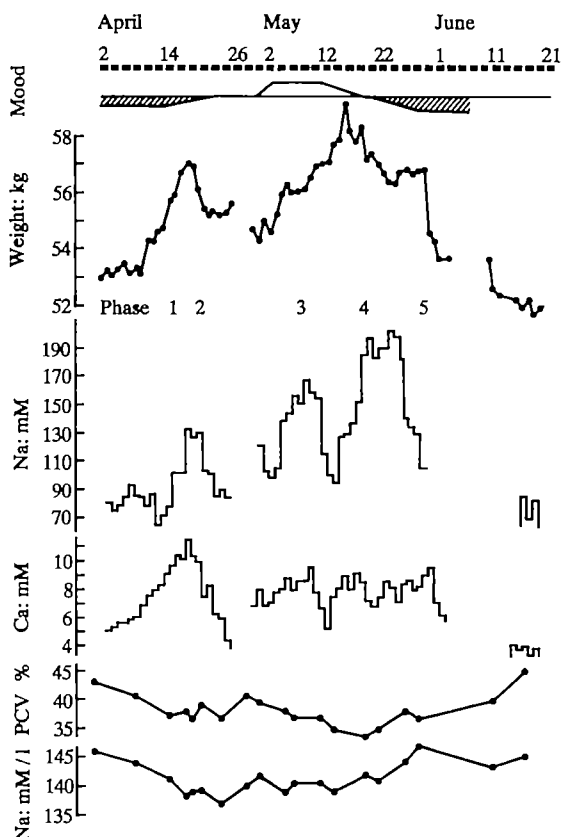


FIG 1 Changes in body weight, urinary sodium and calcium, and in plasma haematocrit (packed cell volume) and sodium between 2 April and 21 June (Study I). Below the dashed line at the top representing days is an indication of mood state, shaded areas below the horizontal line representing depressive episodes, clear enclosed area above manic phase. Note particularly that while body weight rises constantly during manic phase (phases 3–4), daily urinary sodium loss shows two stages, marked at first, but with a very striking drop during the last 5 days of the manic phase suggesting marked sodium retention at this time.

changes are consistent within the phases and the results are suggestive.

When weight fell in phases 4 and 5 the daily urine loss of sodium was of the order of 160–180 mmol, when weight rose in phase 1 the loss was only 80 mmol. If dietary sodium was about 150 mmol per day, these are the urine values to be expected with the recorded weight changes. In phase 3, however, the situation was more complex, with a big weight gain yet a daily sodium loss of nearly 140 mmol, about the same as in the weight loss of phase 2. However at this time the patient complained of thirst and certainly

TABLE I
Daily urinary cation excretion in different phases (all values in mmol per 24 hours)

		Sodium	Potassium	Calcium	Magnesium
<i>Phase 1</i>					
Depression declining	Apr 9	65	50	6.5	8.6
Weight rising	10	85	56	8.2	9.2
	11	89	55	8.5	8.5
(8 days)	12	70	48	7.7	7.3
	13	40	26	8.7	7.5
	14	109	54	11.9	11.4
	15	85	38	8.9	9.0
	16	114	61	11.0	8.6
8-day total (and daily mean)		657 (82)	388 (46)	71.4 (8.9)	70.1 (8.7)
<i>Phase 2</i>					
Weight falling	Apr. 18	176	83	12.8	10.3
(2 days)	19	106	52	7.6	6.1
2-day total (mean)		282 (141)	135 (67)	20.4 (10.2)	16.4 (8.2)
<i>Phase 3</i>					
Hypomanic	May 2	110	50	7.2	7.6
Weight rising	3	128	56	6.4	8.4
	4	186	74	10.5	9.3
(13 days)	5	125	65	7.8	9.4
	6	165	71	8.8	9.4
	7	172	89	8.2	9.7
	8	173	98	9.5	11.8
	9	140	85	8.8	10.5
	10	160	83	11.3	12.2
	11	57	27	3.9	5.7
	12	96	36	5.4	6.6
	13	145	64	7.1	7.6
	14	152	71	11.2	12.5
13 day total (mean)		1809 (139)	869 (67)	106.0 (8.1)	120.7 (9.3)
<i>Phase 4</i>					
'Normal'	May 15	99	37	6.4	5.1
Weight falling	16	173	61	9.7	9.4
	17	184	72	9.2	10.4
(9 days)	18	207	56	9.4	9.4
	19	210	68	7.8	8.7
	20	146	46	5.2	5.8
	21	222	59	8.4	9.0
	22	213	59	9.6	8.4
	23	187	59	8.4	7.2
9-day total (mean)		1641 (182)	517 (57)	74.1 (8.2)	73.4 (8.1)
<i>Phase 5</i>					
Depression increasing	May 29	162	57	12.0	7.9
weight falling	30	177	63	10.2	6.3
(3 days)	31	144	53	7.4	5.3
3-day total (mean)		483 (161)	173 (57)	29.6 (9.9)	19.5 (6.6)

See text for discussion of values

drank more: Table II shows urine output of 4.0 to 5.4L during manic phase in May, but only 1.5 to 2.5L in the following depression. She may have eaten more too because the potassium excretion rose to a daily 67 mmol on average, certainly at its highest in this phase, yet the Na:K ratio remained steady through phases 1, 2, 3 at 1.8, 2.1, 2.0 respectively (but rose to 3.2 during the sodium and weight loss of phase 4).

The complexity of the manic phase was further emphasised by the plasma measurements (bottom of Fig. 1). In spite of thirst and climbing body weight the plasma sodium was about 137 mmol/L, having fallen from 146 mmol/L in depression and climbing back to that value about May 28 as depression reappeared. The packed cell volume also fell to its lowest as manic phase ended. In other words during phase 3 the plasma was more hydrated, the osmotically active sodium lower than at any other time, yet this was the time of thirst and greater fluid intake. One explanation could be a small inappropriate secretion of antidiuretic hormone at this time, and the incomplete figures available for Table II show the urine osmolality low yet the antidiuretic hormone well maintained. During the depressive phases, when the plasma sodium was higher, and water formed a smaller fraction of whole blood, the patient experienced no thirst. It was necessary to re-examine these phenomena under better control in a later manic phase.

Study II

The purpose was to re-examine the manic phase (phase 3) under better controlled conditions, and confirm the findings

The patient was re-admitted the following January towards the end of a depressive phase and placed on a constant diet of known composition for 16 days. The first five were settling-in days: manic phase began on the sixth day (January 18). Mineral and fluid balance, with some relevant hormonal measurements, and clinical observation of mental state, body temperature, blood pressure, etc. were carried out daily, essentially as before. Since Study I her husband had noted three manic phases, each associated with restless nights, marked thirst, a degree of urinary

incontinence, and a weight gain (on bathroom scales) of about 6 kg

Diet

A dietician (Miss P. T. Carden) from the nearby general hospital (King's) very kindly prepared a diet adequate in calories and liked by the patient, in which *the same quantities of the same foods were eaten each day*, and the daily mineral intake calculated with the help of tables (McCance and Widdowson 1960). The diet allowed 50 g salt-free bread, 30 g salt-free butter, 400 g milk, 100 g orange squash, and otherwise distilled water, 40 g lamb and 30 g chicken breast, oatmeal, 50 g potato, and other vegetables and tinned fruit, jelly, Nescafe, and so on, to give a varied menu in the 24 hours. Care was taken to cut all meat from the same joint, and to use a single baking of bread, etc., to aim for uniformity of analysis. The calculated mineral intake was sodium 34 mM, potassium 56 mM, calcium 19.4 mM and magnesium 4 mM. The 24-hour urine content of these ions on the fourth and fifth pre-manic days, which may be taken as the basal starting state, was Na 36, K 42, Ca 1.13 and Mg 1.8 mM: since it is known that in man virtually all the day's sodium intake and four-fifths of the potassium are normally excreted in the 24-hour urine, these values agree well with the calculated values for the diet. It had been intended to have a higher daily sodium intake than this by supplementing with a known weight of oral sodium chloride, but this was omitted in error. However, the results are still interpretable, although the effect was probably to reduce the degree of sodium retention and of weight gain (as in another patient, Crammer 1959b).

Fluid intake was free, but always measured, and in fact was roughly constant at 1.5–1.75 litres daily, until with the onset of thirst on the seventh manic day it rose above 2.0 L. (see Fig. 2). The patient had her own bedroom, stayed in the ward under nursing supervision the whole time, and only had access to water and toilet facilities under observation. All urine was collected as a sequence of 24-hour urine specimens. She continued to receive her

TABLE II
Urmay aldosterone and vasopressin in Study I

Date	Mental State	Volume/24 hrs.	Osmolality	Aldosterone	Arginine Vasopressin
2 May	Hypomanic	4500 ml	149 mOsm.	6.6 µg	111 pg/min
3	"	4000 ml	179 mOsm.	6.3 µg	156 pg/min
14	End of manic	5440 ml	180 mOsm	6.1 µg	214 pg/min
15	"	4600 ml	108 mOsm.	2.0 µg	90 pg/min
1 June	Depression	1580 ml	311 mOsm.	5.6 µg	176 pg/min
2	"	2400 ml	298 mOsm.	8.9 µg	278 pg/min
17	"	2550 ml	490 mOsm	-	164 pg/min
18	"	1520 ml	370 mOsm	-	174 pg/min

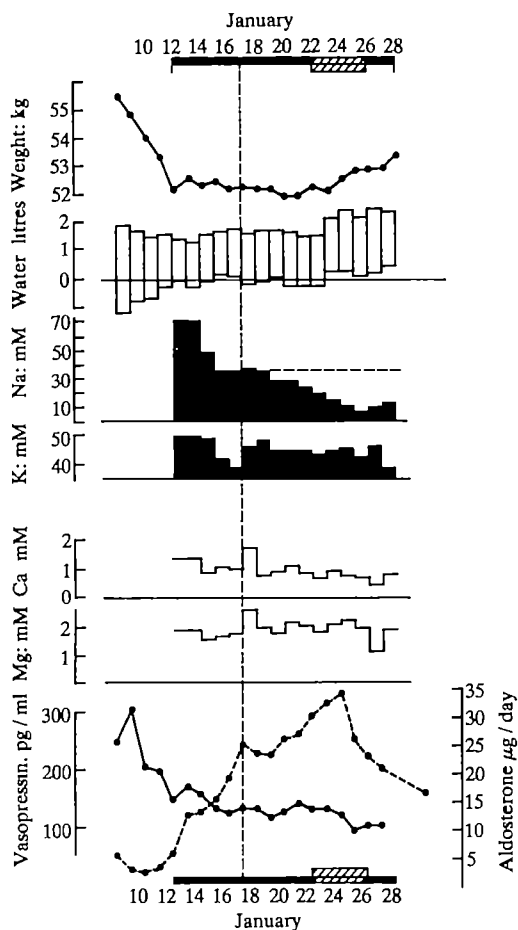


FIG. 2 End of depression, start of manic phase indicated by vertical dotted line. Constantly daily sodium intake 13–28 January, indicated by the dark horizontal line: the shaded rectangle indicates the four days when lithium doses were omitted.

Water intake shown by the top of each plain rectangle, urine output by the bottom: where this is below baseline there is net fluid loss, where above baseline net fluid retention (from January 24).

Sodium excretion is steady for four days, and retention (below horizontal dotted line) begins on January 20.

Aldosterone (dashes in lowest figure) rises anew from January 21 to January 24; vasopressin (continuous) fairly level January 12–28.

usual drugs, nitrazepam 5 mg at night, and 800 mg lithium carbonate (Priadel) every morning, except on the four days shown below.

Blood pressure

At 09.30 each day she had always been lying supine for at least 30 minutes, when I took her pulse and blood pressure,

and a blood sample. She then got up, walked about for half an hour, and then sat down, erect, for a repeat of pulse, blood pressure and venesection.

Laboratory

Na, K, Ca, Mg were measured by atomic absorption spectrometry; creatinine, and plasma concentrations of urea, urate, albumin, bilirubin, cholesterol, phosphate and chloride by autoanalyser; packed cell volume by haematocrit; plasma osmolality by freezing point determination; aldosterone, antidiuretic hormone, and plasma renin were kindly measured by Dr J. D. P. Slater and his staff at the Middlesex Hospital.

Results

(a) *Clinical*: On first four days (January 9–12), she was at first slowed, with inability to concentrate, concerned about her bowels, slightly nauseated, not thirsty, but improving to the end of this period. On January 13 she started her special diet, and continued to develop some spontaneity; on January 15 and 16 she was free of depression, more active and normal. Normal stools were passed on January 11 (126 g) and 12 (176 g) and also on January 15. A further stool was passed on the night of January 16, and two further stools on January 17, and these last three were all much softer and came with some urgency. Thereafter she was constipated, with small stools on January 20 and January 28 only.

On January 18, the manic phase began, but at first it was extremely mild. However, by January 22 she was sleeping less (drop from 8 to 5 hours), had become talkative, with some urge to speak, and not always to the point. By January 25 she was very talkative, thoughtless, restless, and joyful, and was complaining somewhat of thirst, as in previous cycles. She longed to smoke a pipe, and eat chocolate.

(b) *Cardiovascular*: The resting pulse was very steady from day to day at 47.6 per min average on the five pre-manic days, and 50 per min from January 18–29 (manic phase) (Table III). But when the effect of exercise and posture was examined there was a striking difference between pre-manic and manic. In the first (six) days the pulse was always quickened after exercise by 3–9 beats/min, but on January 18 and thereafter it was always slowed by a similar amount, an abnormal response.

Blood pressure showed a 27% increase in resting diastolic pressure with onset of manic phase (see Table III), with a much smaller rise in systolic pressure. As expected, both pressures increased on exercise and change of posture, to about the same extent in pre-manic as in manic phases (normal). The changes in pulse and diastolic pressure occurred at the very start of hypomanic phase, long before there was a noticeable increase in physical activity. They were consistent with the idea that there was a change in central vasomotor control from the start of manic phase, resulting in a change in renal blood flow and an increased sodium retention.

(c) *Sodium and water balance*: Figure 2 and Table IV show what happened. There was little change over baseline until the third manic day (January 20) when sodium began to be

TABLE III
Standard morning blood pressures before and during manic phase (all values in mmHg)

	<i>Supine</i>		<i>Erect</i>		<i>Postural gain</i>
	<i>Sys.</i>	<i>Dias.</i>	<i>Sys.</i>	<i>Dias.</i>	
<i>Premanic</i>					
Jan. 13	120	62	135	90	
14	110	55	110	60	
15	110	58	132	90	
16	108	68	120	90	
17	100	65	125	98	
Av.	109.6	61.6	124	80.5	+ 14/+ 19
<i>Manic</i>					
Jan. 18	115	90	124	100	
19	110	65	130	100	
20	110	65	130	100	
21	130	90	140	100	
22	112	85	120	95	
Av.	115.6	79	129	101	+ 13/+ 22
<i>Manic gain</i>					
	+ 6	+ 17	+ 5	+ 21	

retained, and this continued for the remaining metabolic days. Water only began to be retained on the seventh day (January 24). The weight gain over 5 days (January 24–29) was only 1.3 kg, the total sodium retention by the 29th of the order of 160 mM, which at an extracellular fluid concentration of 140 mM/L is worth a weight gain of nearly 1.2 kg. The positive fluid balance over the last five days implies retention of about 1.5 L. These values agree quite well. In the hypnomanic phase of Study I, and in the husband's reports they were three times bigger. Here they are probably small because of the rather low sodium in the diet. In an earlier case (Crammer, 1959b) varying the daily sodium intake altered the sodium and water retention.

If we look at plasma changes (Table V) the haematocrit dropped with the onset of manic phase, 42% to 39%, a smaller change than noted in Study I, but again pointing to increased hydration of the blood. Plasma sodium showed a tendency again to fall, most noticeable in the last five days, the plasma osmolality likewise, and the plasma albumin and urea also fell in the last four or five days. This was a time when clinically thirst was most in evidence and a higher plasma osmolality would have been expected.

In a short-cycle manic-depressive previously studied (Crammer, 1959b) while off all drugs, phases of sodium and water retention were observed in step with the mental states, with fall in resting pulse, rise in body weight, and in particular the same fall in PCV and plasma sodium and evidence of increased hydration of the blood as noted here. Jenner *et al.* (1967) also noticed such a change in their case.

Antidiuretic hormone (Fig. 2 and Table V) fell steadily as osmolality fell, and therefore could not be seen to be playing any part in the fluid retention and thirst of January 24 and after. Paradoxically, at the end of the depression (Phases A and B), plasma osmolality and sodium were quite high (higher than in Study I) but there was no thirst, antidiuretic hormone was not greatly raised, and in fact sodium and water excretion were up, and the body weight falling. There is no evidence here of a role for arginine vasopressin in the pathophysiology of this illness (compare Gold *et al.*, 1984). If antidiuretic hormone can come into play it may do so following a much greater degree of sodium retention.

The salt retention took priority in this patient's manic phase, and her aldosterone rose at this time, although already at a high steady level because of the rather low daily sodium intake. No increase in potassium excretion was seen, however. Aldosterone was not raised in Study I, perhaps because dietary sodium was freely available. Plasma renin activity did not show any relevant change. It was higher while the controlled low sodium diet was given, but did not rise in the manic phase, and the effect of posture and exercise remained similar in non-manic and manic conditions.

(d) *Calcium and magnesium*: Urinary magnesium increased in the manic phase, starting on the fourth day (January 21), a change from about 1.75 to 2.10 mM per day, a 20% rise. Calcium remained roughly constant, with a 10% drop only in the last four days, and plasma calcium was constant until this time and then fell slightly.

TABLE IV
Daily urine excretions during metabolic experiment (Study II)
Urine data on constant diet. All values millimol

Diet (same every day) from tables: Na 34.0, K 56, Ca 19.4, Mg 4 mM

State	Date	Volume	Creatinine	Na	K	Mg	Ca	Li
End of Depr. A	9 Jan.	3055 ml	8.6	156	58	3.76	3.79	18.6
	10 Jan	2600	8.4	166	55	3.46	3.38	18.5
	11 Jan.	2220	8.4	153	51	2.53	2.44	16.7
	12 Jan.	1800	8.3	101	43	2.16	2.23	17.1
Diet, B	13 Jan.	1000	7.3	66	41	1.61	1.26	15.3
	14 Jan	2010	12.5	76	60	2.29	1.51	17.3
	15 Jan	1630	9.1	49	49	1.66	0.90	13.0
	16 Jan	1500	8.4	36	42	1.77	1.13	13.2
	17 Jan.	1555	8.4	36	39	1.83	1.09	13.0
Mania Onset, C ₁	18 Jan.	1820	8.7	38	46	2.66	1.78	19.2
	19 Jan.	1730	9.0	36	48	2.08	0.87	13.8
	20 Jan.	1660	8.3	29	45	1.86	1.00	16.1
	21 Jan.	1790	9.0	29	45	2.24	1.20	18.6
	22 Jan.	1640	8.9	25	45	2.14	0.98	18.2
Lithium Stop, C ₂	23 Jan	1620	9.2	21	44	1.94	0.82	11.3
	24 Jan.	1890	9.1	16	45	2.21	1.04	8.9
	25 Jan	2240	9.0	12	46	2.24	0.90	6.7
	26 Jan.	2040	9.0	9	43	2.08	0.84	4.9
Lithium Resume	27 Jan.	2240	9.0	11	47	1.34	0.56	7.2
	28 Jan.	1940	8.5	12	39	2.02	0.97	10.9

Note the raised magnesium, lithium and calcium on the switch day (Jan. 18)

Thus, the suggestion from Study I of increased urinary magnesium loss in manic phase was confirmed by the metabolic balance study. The onset of manic phase on January 18 was marked by looser stools, before constipation set in, and by high excretions of magnesium and lithium, and to a lesser extent of calcium, just for 24 hours.

The significance of these changes is uncertain: it would be necessary to confirm their re-occurrence at each manic onset, or in other similar patients, and to examine changes in fecal minerals at the same time to begin to analyse what may be coincidence, an effect of physical exercise, or of changed bowel habit or altered renal function, or have some other explanation.

Discussion

Physical signs and biochemical disturbances in the course of a major psychiatric illness may be purely coincidental, perhaps due to some concomitant physical illness, or quite commonly result from a change in the patient's behaviour (Kety, 1959). In spite of many years of hope and study (Barnes & Francis, 1909; Pighini, 1907; Gjessing, 1976), no

biochemical disturbance has been found causal of a major psychosis. There is, however, another but neglected possibility, that changes in the regulation of the body temperature (Nikitopoulou & Crammer, 1976), of pulse and blood pressure, or of urinary secretion may be signs of central nervous disturbance quite as much as are the appearance of hallucinations or of pathological mood.

In the present case, long-standing undoubted bipolar affective disorder has been shown to be accompanied by pathological variations in body weight, due in part to changes in the renal excretion of sodium and in the retention of water, and accompanied by pathological thirst in the manic phase. This phase has also exhibited a change in cardiovascular regulation. These phenomena were all small, and easily overlooked. They have on occasion been reported in other patients before (Klein & Nunn, 1945; von Stockert, 1958; Crammer, 1959b; Jenner *et al*, 1967; Hullin *et al*, 1967, 1977; Allsopp *et al*, 1972), mostly in cases who were drug-free. It

TABLE V
Plasma measurements during metabolic balance (Study II)

	Day	PCV	OSM	(Na)	aVP	(Ca)	PRA recumbent	erect
Phase A	9			250				
	10				300			
	11	39	305	153	207	2.40	1.52	2.26
	12							
B	13	Diet begun						
	14	41	305	152	169	2.43	5.26	6.83
	15	42	304	145	155	2.40	5.39	7.02
	16	42	303	150	138	2.39	5.57	7.07
	17							
Manic Onset C	18			146	138	2.41	5.55	7.20
	19	39	300		136			
	20			144	120	2.36	5.58	7.30
	21							
	22	38	291	145	145	2.40	5.61	
	23	39	296	148	135	2.45	5.57	6.88
C ₂	24	(wt. starts upwards)						
	25		291	141	125	2.35	5.56	6.94
	26	39	292	145	98	2.31	5.55	6.90
	27		287	142	107	2.24	5.34	6.93
	28		277	144	107	2.23	5.31	6.90
	29		281	141		2.19	5.27	6.81

Phase A depressed, Phase B start of constant diet, Phase C onset of manic phase

PCV, packed cell volume %; OSM, osmolality in mOsm/Kg, (Na) (Ca), sodium, calcium concentrations in mM/l, aVP, arginine vasopressin in Pg/ml, PRA, plasma renin activity.

seems evident that in some, though perhaps only in a minority, functional psychoses may have physical signs and biochemical signs just like other illnesses. It is least difficult to detect them in repeating periodic illnesses (Crammer, 1959a; Jenner, 1968) but there is no reason to suppose they will be confined to such illness and they need to be sought much more often. We accept the idea that hormonal provocation tests such as the dexamethasone suppression test (Carroll, 1982) or the clonidine-growth hormone test (Checkley *et al*, 1981) may help in diagnostic differentiation, in exploring central nervous dysfunction, and in predicting success in pharmacotherapy. Should we not similarly take advantage for the same purposes of the disturbances in regulation of water and electrolytes which occur naturally in the course of psychiatric illness? They are easy to follow clinically in terms of weighing the patients - and collecting timed blood and urine specimens for simple analyses. Psychiatric diagnoses are based on the grouping of symptoms, and a label such as catatonic schizophrenia or major bipolar affective disorder carries no guarantee of pathophysiological unity. On the other hand cases which have in common a disturbance in cardiovascular

regulation, or in body weight may be found to have also a similarity in pharmacological response.

The observations here concentrated on the manic phases. In Study I on free food and water, weight gain appeared as a consequence of thirst and water retention; the blood becoming increasingly hydrated. Some pilot data suggested that vasopressin secretion, (Gold *et al*, 1984) might be playing a role, while aldosterone appeared unimportant. In Study II, which examined the manic onset rather than the manic ending, and which severely limited daily sodium intake, the reverse seemed to be true. Sodium retention came first, before there was any thirst or increase in water intake or in weight, and it seemed possible that aldosterone was involved rather than vasopressin. Hullin's group (Allsopp *et al*, 1972; Hullin *et al*, 1977) have been pioneers in emphasising the importance of aldosterone in these cases. One might speculatively develop a hypothesis that the manic phase may begin with a vascular disturbance, particularly renal and leading to inappropriate aldosterone secretion and sodium retention. At a certain point, the sodium input stimulates thirst and vasopressin secretion, and the resulting dilution of sodium results in a cut-off of thirst and of manic

symptoms and return to the starting point. Thus the manic phase would have different mechanisms predominating at different times in its course.

However this may be, modern renal physiology emphasises the angiotension-renin-aldosterone system in the genesis of thirst, and a cardiac natriuretic hormone, and nervous control of renal function (Ganong and Barbieri, 1982; Seldin and Gebisch, 1985) as factors also to be taken into account. Elucidation of the mechanisms at work in the fluid disturbances of psychiatric cases will require the application of this knowledge and much better and fuller hormonal measurements than those reported here. The purpose of this paper is to draw attention

to physical signs—cardiovascular, changes in body water, changes in magnesium excretion—which appear to be part of a psychotic illness. Explanation comes later.

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Panic Attacks: New Approaches to an Old Problem

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Anxiety attacks were included in the first descriptions of the syndrome of anxiety neurosis. Recently it has been suggested that such attacks (now usually called panic attacks) characterize a distinct form of anxiety disorder—panic disorder. It has also been proposed that panic attacks result from a biochemical disorder and require pharmacological treatment. Some of the evidence for these ideas is presented, and two other explanations for panic attacks are reviewed: that they are caused by hyperventilation, and that they result from a cognitive disorder. It is concluded that although it is not possible on the present evidence to choose between the three theories, there is strong indirect support for the cognitive theory and good reason to investigate cognitive factors more thoroughly. Future work on biochemical causes of, and pharmacological treatments for panic attacks should take account of such factors.

The syndrome of anxiety neurosis was first described by Freud (1985) in a paper published in a neurological journal under the title "The justification for detaching from neurasthenia a particular syndrome: the anxiety-neurosis." Freud described all the features that we recognise today, and drew attention to one feature in particular: anxiety attacks. Although this was the first description of anxiety neurosis, similar attacks had been noted by da Costa (1871) in his description of 'soldiers' heart'. However, da Costa ascribed the symptoms to a cardiac rather than a nervous disorder—a view shared by many patients today.

A description of panic attacks

Freud's description of the symptoms of an anxiety (or, as it is now usually known, panic) attack is closely similar to that incorporated in the current American diagnostic system, DSM-III (American Psychiatric Association, 1980), under the rubric for Panic Disorder. An attack consists of a rapid onset both of intense symptoms of autonomic arousal and of mental symptoms. Physiological recordings reflect the disturbance, showing increased skin conductance, muscle blood flow, and heart rate (Lader & Mathews, 1970). The mental symptoms were well described by Freud (1895), who drew attention to the interplay of the two kinds of disturbance:

"An anxiety attack may consist of a feeling of anxiety alone, without any associated ideas, or accompanied by the interpretation that is nearest to hand such as ideas of the extinction of life, or a stroke, or the threat of madness . . . or the feeling

of anxiety may have linked to it a disturbance of one or more of the bodily functions—such as respiration, heart action, vasomotor innervation or glandular activity. From this combination the patient picks out in particular now one, now another, factor. He complains of 'spasms of the heart', 'difficulty in breathing', 'outbreaks of sweating' . . . and such like; and in his description, the feeling of anxiety often recedes into the background."

Panic attacks are of two kinds. The first kind are provoked by a stimulus that the patient recognises; in other words, they are a particularly intense form of phobic anxiety. The second kind appears to arise spontaneously; to quote Freud (1895) again, they "erupt into consciousness without being called forth by any train of thought." Nowadays it is generally assumed that the two kinds are equivalent, and they are studied together. However, they could arise in quite different ways, and might respond to different treatments. Indeed, there is a strong clinical impression (though the evidence is still incomplete) that panic attacks which occur in phobic situations and are accompanied by avoidance respond to exposure treatment, while those occurring spontaneously do not but respond instead to imipramine. This account is mainly concerned with the second kind—the apparently spontaneous attacks of panic.

Do panic attacks define a separate disorder?

Freud described panic attacks as a general feature of anxiety neuroses. For many years most psychiatrists shared this view, but it is now commonly suggested

that anxiety neuroses with panic attacks are a separate entity, and this idea is now incorporated in DSM-III. The alternative view—that panic attacks are merely an index of the severity of an anxiety disorder—is still held by many psychiatrists, and it is appropriate to examine briefly the strength of the evidence for the opinion that there is a separate panic disorder.

The idea that panic attacks indicate a distinct form of anxiety disorder was originally proposed by Klein (1964). He based this claim mainly on the observation that panic attacks respond to treatment with imipramine and that the background of generalized anxiety does not. Many of Klein's patients appear to have been agoraphobic, showing avoidance as well as panic attacks; they thus belonged to the first of the two types noted above. Because Klein regards avoidance behaviour as secondary to panic attacks, he has considered it appropriate to combine in his studies patients who avoid (agoraphobics) and those who do not (generalized anxiety neuroses). While this is a tenable position, it is important to be aware of it, because the treatment in the major clinical trial by Klein and his colleagues included both exposure, directed at avoidance, and imipramine, directed at panic (e.g. Zitrin *et al*, 1983). Since there is evidence that exposure alone can reduce the panic attacks of agoraphobic patients (e.g. Hibbert & Chan, 1986) it is necessary, in order to support Klein's position, to show that imipramine alleviates panic attacks in generalised anxiety neurosis. To date this has not been proved beyond doubt, since the evidence published so far is based only on a small group of patients (Garakani *et al*, 1984). However, further evidence on this issue should be available soon from trials now in progress.

Do patients with panic attacks differ from those with generalised anxiety disorder in ways other than their response to treatment? Several authors have reported differences in symptoms. However, these differences are directly related to the symptoms of the panic attacks themselves and are therefore not helpful in answering the question. They include more somatic symptoms (Hibbert, 1985; Hoehn-Saric, 1982; Anderson *et al*, 1984) and more preoccupations with ideas about illness and dying (Hibbert, 1985) among panic attack patients.

Are there any differences not directly due to the panic attacks? The evidence on this point is confounded by the inclusion of agoraphobic patients in the panic attack groups, so that the observed differences may be due, at least in part, to differences between agoraphobia and general anxiety neurosis rather than between panic disorder and general anxiety neurosis. Thus Raskin *et al* (1982) found that

panic attack patients reported more disturbances in their childhood environment, but not difference in loss or separation. They also found that the panic disorder group reported more previous depressive disorders. Crowe *et al* (1983) studied the relatives of probands with panic disorder and found an excess of panic disorder among their first degree relatives, but no increase of generalised anxiety disorder. These family differences suggest the possibility of genetic differences between panic disorder and generalized anxiety disorder, but these differences might only be in the degree of genetic loading, with a greater genetic influence in severe anxiety neuroses than mild ones. It can be concluded that there is as yet no strong evidence that panic attacks mark out a different disorder, rather than a more severe degree of the same one. However, whatever the truth of this issue, the causes of panic attacks still need to be explained.

Causes of panic attacks

The normal responses of fear and anxiety are the final result of a complicated series of physiological and psychological mechanisms. Panic attacks must result from a disorder of some of these mechanisms, and this disorder might be the same in every case, or differ between cases.

A discussion of the causes of panic attacks requires a broad understanding of the mechanisms of anxiety: there are physiological and psychological components, and each has several elements. The physiological mechanisms include control centres in the brain and effector systems in the periphery. The central mechanisms include the locus ceruleus, a nucleus involved in the control of sympathetic activity and in the regulation of arousal. The peripheral physiological mechanisms include autonomic arousal, increased tension in skeletal muscles, enhanced respiration, and the release of adrenaline and noradrenaline.

Peripheral noradrenergic mechanisms

Many of the most obvious symptoms of a panic attack reflect overactivity of the sympathetic nervous system. This overactivity might arise in two ways: from heightened sensitivity of end organs, or from increased activity in sympathetic nerves. The first possibility was suggested by Wearn and Sturgis (1919) who gave intramuscular injections of adrenaline to patients with 'irritable heart' (a condition now generally thought to be identical with severe anxiety disorders). They observed that the same amount of adrenaline produced more symptoms in the anxious patients than in normal controls. This finding might

indicate that anxious patients are unusually sensitive to the effects of adrenaline. However, the differences could be due to variations in absorption from the injection site, and the crucial experiment requires an intravenous infusion of adrenaline. This was done by Fraser & Wilson (1918) and by Hume (1918), and when Wood (1941) combined their results he found that anxiety neurosis patients and controls responded equally.

Since that time, it has been shown that these peripheral sympathetic effects depend on β -adrenergic mechanisms. Nesse *et al* (1984) examined the effect in anxious patients and controls of the specific β -adrenergic agonist, isoproterenol. They found no evidence of increased β -adrenergic receptor responses in panic anxiety patients; indeed, there was an indication that the response was slightly decreased, a finding that could reflect down-regulation of receptors in response to sustained high levels of noradrenaline. Thus although some of the symptoms of panic disorder are undoubtedly related to increased sympathetic activity, this does not seem to be due to hypersensitivity of post-synaptic β -adrenergic receptors.

Central autonomic regulatory mechanisms and panic attacks

The central regulatory mechanisms for anxiety are complicated, but for the purpose of this review they can be simplified by focusing attention on a single important structure: the locus ceruleus, which is situated in the floor of the fourth ventricle. In animals, ascending noradrenergic pathways can be traced from the locus ceruleus to the hippocampus and forebrain, where they probably modulate arousal levels. Descending pathways from the locus ceruleus affect peripheral sympathetic activity.

Three kinds of evidence point to the importance of the locus ceruleus in the regulation of anxiety in animals (Redmond & Huang, 1982). Firstly, stimulation of cells in the locus ceruleus leads to behaviour like that produced by anxiety-provoking stimuli. Secondly, lesions in the locus ceruleus abolish the behavioural response to anxiety-provoking stimuli. Thirdly, the drugs piperoxan and yohimbine, which increase activity in cells of the locus, produce anxiety-like behaviour in animals. Not all the evidence points in the same direction; for example, the drug buspirone increases activity in the locus but reduces anxiety (Sanghera *et al*, 1983).

If a control mechanism of this kind exists in man, panic attacks could arise from excessive stimulation of neurons on the locus ceruleus or from inadequate

inhibitory mechanisms. At present most interest is centred on possible failures of the inhibitory mechanisms. The noradrenergic neurons in the locus ceruleus have receptor sites for several inhibitory neurotransmitters, notably α_2 adrenoceptors, and receptors for gamma-aminobutyric acid (GABA) and encephalins (see Redmond & Huang, 1982). In this review only the α_2 adrenergic autoreceptors will be considered (see Grant *et al*, 1980, for the possible role of GABA-benzodiazepine receptors).

If these poorly-functioning autoreceptors are a cause of panic attacks, yohimbine (a drug which penetrates the blood/brain barrier and inhibits these receptors) should reduce their activity more readily than in generally anxious or normal subjects. Removing an inhibitory mechanism on these cells will increase autonomic activity and arousal. It should also result in a greater increase in plasma 3-methoxy-4-hydroxy-phenylethylene glycol (MHPG), which seems to reflect central noradrenergic turnover.

Charney *et al* (1984) gave yohimbine orally to drug-free anxious patients and to healthy controls. The drug produced greater increases in tremors, palpitations and systolic blood pressure in the patients than in the controls. Anxiety also increased more, and patients reporting the most panic attacks before the test showed the greatest rise in plasma MHPG in response to yohimbine. These findings are consistent with the view that panic attacks develop when there is an abnormal balance between excitation and inhibition at the locus ceruleus (and perhaps other areas of the brain), possibly due to abnormal α_2 receptor function. However, the findings could also be explained if the direct effects of yohimbine are restricted to increases in the physiological symptoms of tremor, pulse rate and blood pressure, while the feelings of panic result from the patient's psychological response to these physical changes. Furthermore, yohimbine is a less selective α_2 adrenoceptor antagonist than certain recently discovered compounds; to be certain that noradrenergic changes are of primary importance this work needs to be repeated with these more specific agents.

Clonidine is another drug that acts on α_2 autoreceptors. Because clonidine is an α_2 agonist it stimulates the autoreceptors, thereby reducing activity in the noradrenergic neurons of the locus. If α_2 receptors are underfunctioning in the patients with panic attacks, clonidine would not be expected to be a powerful way of controlling the attacks. Ko *et al* (1983) gave clonidine to panic attack patients and found only a weak anxiolytic effect, although there were definite effects in lowering blood pressure and producing sedation. These findings are compatible

with the α_2 hypothesis of panic attacks, but certainly cannot be taken as strong evidence for it.

Imipramine and panic attacks

The revival of interest in panic attacks can be traced to the observation by Klein (1964) that imipramine seemed to have a specific effect on this aspect of anxiety disorders. A subsequent clinical trial (Zitrin *et al.*, 1983) appears to confirm this observation, although the size and generality of the effect is difficult to assess due to the high drop-out rate from both the imipramine-treated group (29%) and the control group. Also, patients had phobias with panic attacks rather than generalized anxiety with panic attacks.

If imipramine does suppress panic attacks, how might this come about? Two clinical observations are relevant. Firstly, many anxious patients complain of unpleasant side-effects when they first take imipramine even in small doses (Zitrin *et al.*, 1978). Although the point has not been tested formally, it is reported that these side-effects are more severe than those experienced by most depressed patients; thus Zitrin (1983) states that some patients are exquisitely sensitive to the drug, developing insomnia, irritability, unusual energy and apprehension with doses as low as 10 mg/day. Secondly, it appears that the anti-panic effect is slow to develop, taking up to four weeks before panics cease (Zitrin, 1983). This contrasts with the early onset of side-effects.

These clinical observations can be compared with pharmacological findings about imipramine and related antidepressants. The immediate effect of these drugs is to block the re-uptake of noradrenaline, thereby increasing the amount of noradrenaline acting post-synaptically. This action could account for the side-effects noted early in treatment. The subsequent changes are quite complex. Long-term administration of tricyclic antidepressants reduces post-synaptic β -adrenergic receptor function and increases post-synaptic α_2 adrenergic function (Charney *et al.*, 1981). Also, chronic administration of some tricyclic antidepressants decreases α_2 adrenergic autoreceptor function (Charney *et al.*, 1981). The net effect of these changes on noradrenergic function is extremely difficult to predict. One way around this difficulty is to assess overall transmission through the synapse. Cowen *et al.* (1985) have studied transmission through adrenergic synapses using the release of melatonin as an index of this (the release of melatonin in the pineal depends on noradrenergic transmission). These experiments show that in normal subjects after desmethylinipramine has been taken there is an initial increase of adrenergic transmission which lasts for at least a week; after three

weeks of treatment it returns to the pre-treatment level. This return to normal despite continuing treatment suggests some kind of compensatory mechanism that overcomes the effect of the initial rise in noradrenergic transmission. If, as seems likely, imipramine has effects similar to those of desmethylinipramine this compensatory mechanism might have an effect that stabilizes adrenergic synapses. If that were the case, it might also account for the anti-panic effect.

Cowen *et al.* (1985) studied normal subjects, and comparable data are not available from patients with panic attacks. However, Charney & Heninger (1985) have reported decreased MHPG concentrations in the plasma of such patients after 8–12 weeks of imipramine. This finding suggests that central noradrenergic function has been reduced by the drug. The mechanism is not understood, but Charney & Heninger suggest that it is not mainly due to changes in α_2 receptors, since responses to yohimbine (which acts on these receptors) are unchanged.

As yet these ideas are speculative, because we do not know how this compensation is brought about. However, this is an obvious area for research, and the answer might increase our understanding not only of the actions of imipramine in patients with panic attacks but also the abnormality in nervous regulation that is present in these patients.

Hyperventilation as a cause of panic attacks

An increase of respiratory activity is part of the normal 'fight or flight' response. Patients with anxiety neurosis increase their breathing as well as their level of sympathetic arousal and, since they engage in neither fight nor flight, they may breathe off more CO_2 than they produce (i.e. hyperventilate). Hyperventilation produces symptoms resembling those of panic anxiety, as can be seen when the symptoms of the two conditions are listed in parallel (Table). It is possible that panic attacks occur when the symptoms of hyperventilation occur alone or when they are added to the other manifestations of an anxiety response which would otherwise produce less intense symptoms.

The most direct way of deciding whether panic attacks are caused by hyperventilation is to estimate concentrations of carbon dioxide in arterial blood (paCO_2) during a panic attack. These concentrations can be estimated indirectly in a laboratory by measuring end-tidal CO_2 or directly by intra-arterial sampling. However, panic attacks seldom occur in the controlled conditions of the laboratory.

Until recently it was not possible to make measurements of paCO_2 during panic attacks occurring

TABLE I
Comparison of symptoms of panic attacks and hyperventilation

<i>Diagnostic criteria for panic attacks¹ (at least four required)</i>	<i>Symptoms of hyperventilation²</i>
Dizziness, light-headedness or unsteady feelings	Dizziness
Depersonalization or derealization	Unreality
Faintness	Faintness
Paraesthesiae	Paraesthesia
Trembling and shaking	Tremor and coarse twitching
Shortness of breath or smothering sensations	Shortness of breath
Palpitations or tachycardia	Palpitations
Chest pain or discomfort	Atypical chest pain
Apprehension	Apprehension
Choking	
Sweating	
Nausea and abdominal distress	
Hot flushes or chills	
Fear of dying	
Fear of going crazy or of doing something uncontrolled	

1. From DSM-III-R (American Psychiatric Association, 1985)

2. From Lewis (1954)

outside the laboratory, but a method has now been described which can estimate paCO_2 indirectly in ambulant patients. Hibbert (1986) has combined a miniaturized method for measuring transcutaneous CO_2 , developed at University College Hospital, with the Medilog recorder, which was originally developed for 24-hour monitoring of ECG. The principle is this: the rate at which CO_2 leaks through the skin depends on paCO_2 , skin metabolism and skin blood flow. The last two can be held constant in the skin under a pH electrode by warming that skin to a constant temperature. Under these circumstances measurements of pH reflect transcutaneous CO_2 leakage, and have been shown to be highly correlated with paCO_2 (Clutton-Brock & Rithalia, 1984). Using this method, Hibbert has shown that some patients hyperventilate during panic attacks. Not all do this, however, and it is not yet certain what proportion of panic attack patients do. On the present evidence it seems that although hyperventilation may contribute to panic attacks in some patients, it is not likely to be the principal cause in the majority.

Psychological causes of panic attacks

The psychological mechanisms involved in anxiety are no less complicated than the physiological ones. However, for the present discussion it will be enough

to consider one important psychological component: the cognitive changes.

Paradoxically, the psychological theory of panic attacks begins with the results of pharmacological experiments. It has been explained that panic attacks can be provoked by voluntary overbreathing and that yohimbine, an alpha-adrenergic blocker, has a similar effect. It is also well-known that infusions of sodium lactate provoke panics, and do so more readily in anxious patients than in normal subjects (Pitts & McClure, 1967). The mild stimulant caffeine has a similar effect (Boulenger *et al.*, 1984). These observations could be explained if the various agents have the common pharmacological property of stimulating activity in the locus ceruleus (or some other structure controlling anxiety). However, there is an equally plausible alternative explanation, which is worth considering because not all panic attack patients who receive these provoking agents respond by developing a panic: psychological factors may account for the variability in response.

This psychological explanation starts with observations on individual differences in response to one of the provoking agents—hyperventilation. Clark & Hemsley (1982) asked volunteers to overbreathe and recorded three variables: the symptoms experienced, the pCO_2 level reached, and the neuroticism score of each patient. Physical symptoms, especially

palpitations, were quite highly correlated with the lowering of $p\text{CO}_2$ achieved during hyperventilation, while psychological symptoms were related more to measures of neuroticism. Clark & Hemsley suggested that there might be a two-stage process in which overbreathing leads to physical symptoms, which in turn give rise to worries that increase the level of anxiety further. They pointed out that a response of this kind could readily give rise to a vicious circle leading to a panic attack.

Neuroticism is a broad concept; can the psychological component be identified more precisely? It was noted earlier that Freud (1895) stressed the anxious patient's preoccupation with fears of dying, a stroke, or loss of sanity. Beck *et al* (1974) drew attention to similar symptoms, and added fears of heart disease and fainting. Hibbert (1985a) assessed all these fears in a standardized way and confirmed the earlier observations. Of course, the fears might be a consequence of the panic attacks rather than a cause. This problem can be addressed most readily by attempting to treat panic attacks by changing the cognitions with a psychological treatment.

Clark *et al* (1985) have described a treatment in which cognitions are changed by combining voluntary hyperventilation, intended to provoke symptoms, with cognitive restructuring in which it is explained how panic attacks can result from the misinterpretations of bodily sensations resulting from overbreathing and other innocuous causes. Patients then practise controlling these attacks both in subsequent occasions of treatment and during panic attacks arising occurring in everyday life. Two clinical trials have shown that this treatment leads to a substantial improvement in panic attacks in patients who did not improve during a preliminary waiting period (Clark *et al*, 1985; Salkovskis *et al*, 1985). A stricter test of a randomized comparative trial will have to be carried out before these findings can be evaluated fully. However, the existing evidence does seem to give quite strong, although necessarily indirect, support to the cognitive theory of panic attacks.

Conclusion

The various mechanisms for the cause of panic attacks which have been reviewed are not mutually exclusive: they are concerned with different stages of a complicated psychophysiological process which could go wrong in more than one way. Just as we now recognise more than one cause of hypertension or diabetes, so we may need to recognise more than one cause of panic attacks.

There is no support for the theory that peripheral β -adrenergic mechanisms are hypersensitive in patients with panic attacks. Although there are interesting pointers to a possible disorder of the central mechanisms controlling sympathetic activity, there is as yet insufficient firm evidence to support this hypothesis strongly. Nevertheless, this is clearly one of the more promising lines for future research. There is no convincing evidence that the majority of attacks are principally caused by hyperventilation, though overbreathing may play a part in some attacks—it is still not known how many.

The psychological hypothesis that cognitions are abnormal in people who experience panic attacks and that these cognitions amplify the anxiety response is quite strongly supported by evidence, albeit mostly indirect evidence. The idea receives further support from the effects of a cognitive treatment, though the specificity of the effect has not yet been established beyond doubt.

We are still some way from solving the problem of panic attacks which Freud drew to our attention. Nevertheless, much progress has been made in recent years towards a better understanding of the causes, and it seems likely that the eventual solution will be of a particularly interesting kind, integrating pharmacological, physiological and psychological approaches to the problem.

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Schizophrenia therapy: the importance of balance

Too often, neuroleptic therapy swings the mood of the schizophrenic patient too far. Withdrawn patients' moods can be raised so high that they become agitated; whilst agitated patients may sink so low that they become withdrawn.

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Gamma Glutamyl Transpeptidase and Mean Corpuscular Volume: Their Usefulness in the Assessment of In-Patient Alcoholics

R. W. LATCHAM

Gamma glutamyl transpeptidase and mean corpuscular volume are shown to be of little value in the assessment of patients admitted to psychiatric beds for problem drinking.

The measurement of serum gamma glutamyl transpeptidase (GGT) and mean cell volume (MCV) as markers of alcohol consumption has become widespread in surveys of alcohol abuse (e.g. Chick *et al.*, 1981), and as a matter of routine in patients admitted for alcoholic diagnoses. MCV, raised by the direct toxic effect of alcohol on erythropoiesis, reflects long-term drinking habits. GGT, by contrast, is an enzyme, predominantly microsomal in location, which is raised in approximate proportion to recent alcohol consumption. After cessation of drinking the plasma enzyme level returns to within normal range in 3-6 weeks in an exponential fashion (Lamy *et al.*, 1975; Waldstein & Skude, 1979). Morgan (1980) has reviewed the use of these and other markers for detecting and monitoring alcohol abuse. Like others (Chick *et al.*, 1981; Madden & Krasner, 1984) she concludes that although markers in combination are of some use in the routine screening and monitoring of alcoholics, "no simple reliable marker for alcohol abuse exists currently". The poor sensitivity and specificity of both GGT and MCV are discussed by Chick *et al.*, who describe in particular the effects of other illness and drugs on both GGT and MCV, of age and weight on GGT, and of smoking on MCV.

In the present study the correlations of these two markers with a number of variables associated with severity of alcohol dependence or abuse were determined in a large group of patients admitted to psychiatric beds for alcohol dependence. In addition, proportions of the groups identified by the tests separately and in combination were determined, and subjects with and without one or both tests in the 'abnormal' range were compared. It was then possible to describe the sensitivity of the tests and their usefulness as an adjunct to clinical assessment of problem drinkers.

Method

The subjects were first admissions to a psychiatric hospital in 1981-1982, and diagnosed by their admitting psychiatrist

as having the alcohol dependence syndrome, ICD9 category 303 (World Health Organisation, 1979). Three treatment centres (Inverness, Montrose and Canterbury) were selected for the purposes of this and other studies reported elsewhere (Latcham *et al.*, 1984; Latcham & Kreitman, 1985; Latcham, 1985). Samples were consecutive except for those whose length of stay in hospital was so short that contact was not possible.

A total of 190 men and 47 women were interviewed using a standardised instrument in which extremely high inter-rater reliability, already demonstrated by Chick (1980), was obtained by the author and three trained interviewers. Eleven males and two females were excluded at this stage because of serious physical illness not attributed to alcohol, or because they were taking anticonvulsants (including chlormethiazole and barbiturates). For the remaining 179 men and 45 women various items and scores were derived from their interview concerning

- (a) age, weight and smoking habits
- (b) consumption of alcohol during the last week before admission, and during a typical week from the past three months (if the last week was atypical) using the one week retrospective diary method
- (c) the Edinburgh Dependence Schedule (Chick, 1980) from which two measures of alcohol dependence were derived: one categorical (dependent or not) and one measuring severity of dependence on a continuous basis (the number of symptoms of dependence present in the past three months added to form the Edinburgh Alcohol Dependence score)
- (d) a history of health problems reported by patients to have occurred in the past two years. Problems were required to be ascribed by the respondent to alcohol consumption. A health problem score was derived by adding the number of problems which included neuropathy, alcoholic hepatitis and cirrhosis, fits, impotence and chronic diarrhoea.

MCV was measured using Coulter-S counters, and GGT according to the method of Szasz (Szasz & Persija, 1974). The number of days that had elapsed between cessation of drinking and the sampling of blood was recorded. The presence of hepatomegaly was determined by abdominal palpation in a subsample of 66 men and 16 women.

TABLE I
Means and standard deviations of variables by sex

Variable	Mean (<i>n</i> = 179)		Women (<i>n</i> = 45)	
	Mean	s.d.	Mean	s.d.
Alcohol consumption in week before admission ¹ : units of alcohol ²	183	131	111	80
Alcohol consumption in typical week: units of alcohol	185	120	114	74
Alcohol dependence score	27.5	11.5	25.7	10.0
Health problem score	2.6	1.5	2.5	1.5
GGT: iu/l	128	188	146.6	280
MCV: fl	95.7	5.9	97.8	7.9
Age: years	40.4	11.5	41.4	10.9
Days elapsed between last drink and blood sampling	4.2	9.1	3.2	4.8

1. For this variable *n* = 153 (males), 42 (females) (not all patients drank in the last week).

2. 1 unit = one half pint of ordinary beer or equivalent.

Analysis

Analyses of two kinds were performed. Firstly, the subjects were divided into two groups, according to whether their GGT was above or below 50 international units/litre (iu/l). The two groups were compared for mean alcohol consumption, mean number of alcohol-related health problems, mean alcohol dependence score, proportion with and without dependence, and (in the subsample) hepatomegaly. This analysis was repeated six times using varying criteria for GGT and MCV to divide the sample. Each sex was analysed separately. A number of patients (36 men and 7 women) were excluded from analyses concerning GGT because they were taking regular paracetamol, opiates or amphetamine, or because they had last drunk alcohol more than 14 days before their blood was sampled.

Secondly, after log transformation of the data, correlations between alcohol consumption, severity of alcohol dependence, health problem scores, MCV and GGT were calculated; 28 males and 6 females who took opiates, amphetamines or regular paracetamol were excluded, leaving 151 men and 38 women for this analysis. These correlations were recalculated controlling for the effects of age, weight, smoking and number of days between last drink and blood sampling.

Results

The means and standard deviations of measured variables are shown in Table I for the complete sample. In addition, 41 of the 179 men and 7 of the 45 women were found not to be alcohol dependent. In the subsamples whose abdomens

were examined, 26 of 66 males and 4 of 16 females had palpable hepatomegaly. 81% of the men and 92% of the women smoked.

The sensitivities of GGT and MCV (at each cut-off value) are shown in Table II, both separately and in combination. Less than three-quarters of both men and women were identified as having either GGT > 50 iu/l or MCV > 96 fl. Less than a third of either sex had results for both these tests in the 'abnormal' range.

Analysis showed that in neither sex were there significant differences between groups however they were categorised, using two-tailed *t*-tests for continuous variables (severity of dependence, alcohol consumption both in a typical week and week before admission and health problem score), and chi-squared or Fisher's exact test for categorical variables (hepatomegaly and presence or absence of dependence). There were two minor exceptions: males with an MCV above 96 fl were significantly more likely to have hepatomegaly (Fisher's exact test $P < 0.01$), and males with an MCV above 96 fl drank significantly more the week before admission ($t = 4.99$, d.f. 1, 151, $P < 0.05$).

The correlation matrices are shown in Table III. When age, smoking, weight and days elapsed between last drink and blood sampling are controlled, only the negative correlation in women between health problem score and the consumption of alcohol in the week before admission becomes significant at the one in twenty level; otherwise there are no effects either for the men or the women. GGT and MCV are not significantly correlated with any variable for women, and only with each other for males. For both sexes the alcohol dependence score is positively correlated with alcohol consumption in a typical week, and with the health

TABLE II
Percentages of sample by sex identified at various levels of blood results

Blood level criteria	Males			Females		
	n	Sample size	%	n	Sample size	%
GGT > 50 iu/l	83	143	58.0	12	38	31.6
MCV > 96 fl	82	179	45.8	24	45	53.3
MCV > 98 fl	58	179	32.4	21	45	46.7
GGT > 50 iu/l or MCV > 96 fl	102	143	71.3	24	38	63.2
GGT > 50 iu/l or MCV > 98 fl	94	143	65.7	21	38	55.3
GGT > 50 iu/l and MCV > 96 fl	47	143	32.9	8	38	21.1
GGT > 50 iu/l and MCV > 98 fl	39	143	27.3	8	38	21.1

TABLE III
Correlations of GGT and MCV with other variables¹

	Consumption ² in last week	Consumption in typical week	Alcohol dependence score	Health problems score	GGT	MCV
Consumption in last week		0.29***	0.12	0.20**	-0.08	0
Consumption in typical week	0.14		0.49***	0.38***	0	0.07
Alcohol dependence score	-0.10	0.58***		0.50***	0	0.02
Health problems score	-0.30	0.20	0.41*		0.04	-0.04
GGT	-0.23	0.19	0.20	0.26		0.44***
MCV	0.03	0.24	0.13	0	0.24	

1. Correlations of log-transformed data are shown for men above the diagonal, and for women below.
2. $n = 151$ (males) and 39 (females), except for correlations concerning consumption in last week, for which $n = 145$ (males) and 36 (females).

*** $p < 0.001$; ** $p < 0.02$; * $p < 0.05$.

problems score. No item in the health problem score was included in the alcohol dependence score, which in turn did not include a measure of consumption.

Discussion

With reference to Table I, the absence of correlations between MCV or GGT and clinical measures of severity of alcohol-related problems might be considered to be the result of the narrow range of variables which might be expected in a group of problem

drinkers so severe that they required in-patient treatment. However, this is not the case; the range of all variables was considerable.

It might also be suggested that the self-reports of problem drinkers are unreliable in their accounts, both of quantity consumed and extent of symptoms. The significant correlations obtained for both sexes between a measure of consumption, the alcohol dependence score, and the health problems score militates against such a suggestion. Unexpectedly,

GGT and MCV do not reflect severity of dependence, consumption of alcohol, or the extent of alcohol-related health problems. Also, patients of either sex, divided into groups according to the results of their blood tests in various ways, did not differ on most clinical variables measured.

Conclusion

In this study, the results of GGT and MCV do not identify more than about two thirds of patients even when the tests are taken in conjunction. Neither test identifies problem drinkers who are more severely

dependent, or who drink more. Neither test correlates with severity of dependence, with reported alcohol consumption, or with the extent of alcohol-related health problems. Although, as many have suggested (Rosalki, 1977; Morgan *et al*, 1981; Chick, 1983), serial values of GGT and MCV may be useful in monitoring the progress of problem drinkers, these tests are unlikely to add to the initial clinical assessment of such patients.

Acknowledgements

I would like to thank Mr John Duffy and Dr Stephen Platt for their help in the preparation of this paper.

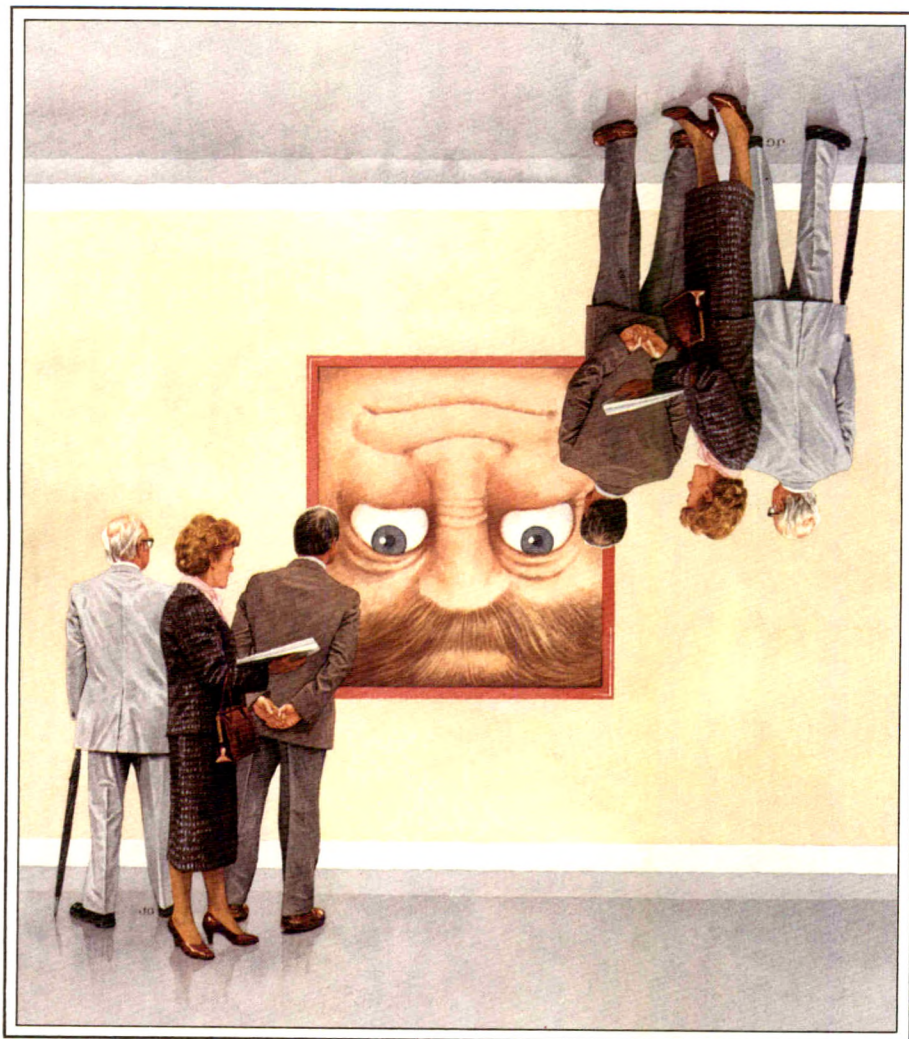
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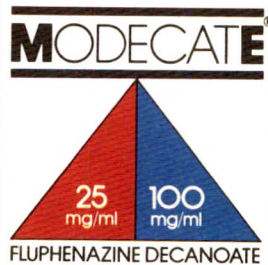
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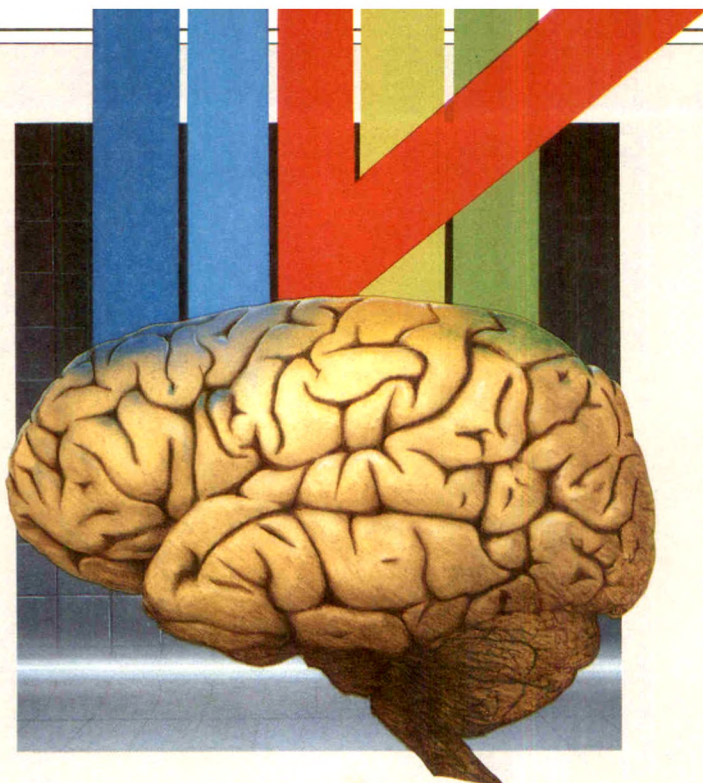


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1) Johnson DAW Br J Psychiat 1975; 126: 457-461.

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A Survey of Practising Psychiatrists' Views on the Treatment of Schizophrenia

S. ANDREWS, K. VAUGHAN, R. HARVEY and G. ANDREWS

Practising psychiatrists' views about the treatment of schizophrenia were investigated as part of a Quality Assurance Project. A questionnaire which asked for treatment recommendations for each of four case descriptions of patients with schizophrenia was mailed to a one-in-six random sample of Australian psychiatrists; 90% responded. Psychiatrists almost uniformly advocated the use of antipsychotic drugs and usually recommended concurrent supportive psychotherapy or family/social intervention procedures. The recommendations varied systematically, according to the initial history obtained and to the initial response to treatment.

The aim of the Quality Assurance Project is to write a set of outlines for the treatment of the common psychiatric disorders, using three sources of information: the research literature on outcome of treatment, the views of a sample of practising psychiatrists, and the views of persons nominated as experts in the treatment of the disorder under study (Quality Assurance Project*, 1982a & b, 1983). This report summarises the results of the survey of practising psychiatrists' recommendations concerning the treatment of schizophrenia. The treatments recommended for different types of patients with schizophrenia are compared, and these recommendations are evaluated in relation to the treatment guidelines formulated by the panel of experts (Quality Assurance Project, 1984), which concluded that

"The three sources of information agree that antipsychotic drugs are the principal treatment during the active phase of the disorder. As schizophrenia is so often a serious and disabling chronic illness, the treatment recommendations stress the need for continuity of care during the post-acute and residual phases in order to prevent relapse and minimise residual deficits. The outlines particularly emphasise the consistent but conservative use of antipsychotic drugs, and the implementation of a specific type of family management programme in addition to the customary social interventions".

Method

The general issues involved in surveying medical practitioners' preferred methods of treatment have been

*Quality Assurance in Aspects of Psychiatric Practice is a project under the aegis of the Royal Australian and New Zealand College of Psychiatrists, funded by the Australian Department of Health. Director: Gavin Andrews.

discussed elsewhere (Quality Assurance Project, 1982b; Hall *et al.*, 1982). The essential conclusions were that the wide geographical distribution of the sample demanded a questionnaire format, and that the questionnaire should specify diagnosis, in order to reduce the confounding of diagnostic issues with treatment recommendations.

The questionnaire

Four 350-word case vignettes were constructed. *Case 1* was a withdrawn and suspicious 19-year-old youth suffering from his first episode of schizophrenia, paranoid type (DSM-III). He had shown prodromal signs of social withdrawal and role deterioration since early adolescence. He lived with his highly critical father and anxious overprotective mother. *Case 2*: a female aged 19 suffering from her first episode, characterised by sexual delusions and auditory hallucinations. Unlike the first patient, premorbid development was normal, with no evidence of a prodrome, so that the DSM-III diagnosis was schizophreniform disorder. Her father had died when she was 12, but her mother appeared warm, concerned and co-operative.

Case 3 was a 53-year-old married woman with three teenage daughters, who had an 18-year history of recurrent episodes of schizophrenia, undifferentiated type (DSM-III), reflected in chronic thought disorder, labile affect, and poor concentration between episodes and periodic exacerbations of acute psychotic symptoms. She had had eight previous hospitalisations and despite continual phenothiazine medication, her condition caused considerable tension and disruption for her family. The same diagnosis had been applied to *Case 4* who was a 28-year-old invalid pensioner with a six-year history of chronic fixed grandiose delusions. Completely lacking in social support and chronically impaired in his functioning, he had a history of recurring hospitalisation and of contact with local psychiatric, social and police agencies. A recent decrease in medication had resulted in signs of tardive dyskinesia, which had disappeared when the dose was raised again.

The first two cases represented first presentations of illness, which were distinguished from each other by the presence or absence of prodromal symptoms and the differing nature of their delusions, while the third and fourth cases were chronic patients who differed principally in their degree of residual impairment and in the presence of social support.

For each case, respondents were asked to specify the components of their treatment plan at three points in time: at initial presentation, one month later, presuming a positive response to initial treatment (no 'improved' description was provided for Case 4), or alternatively, three months later, presuming little or no response to the initial treatment. Treatment components were to be selected from a glossary of 132 different treatment components, grouped under the following major headings:

TREATMENT LOCATION

Pharmacotherapy

Antidepressants
Major tranquillisers
Antiparkinsonian agents
Anxiolytics
Sedative/hypnotics
Vitamins and hormones
Electroconvulsive therapy

Psychotherapies

Individual therapies
Group therapies
Family therapies
Use of adjunctive therapists

Referrals

Other professionals
Specialized day programmes
Residential changes

Each category was further specified so that there were, for example, brief descriptions of ten types of psychotherapy and nine types of family therapy to allow psychiatrists to specify the details of their treatment. The layout of the questionnaire allowed respondents to indicate the relative time course of the introduction of the various components of each treatment plan. As well as specifying their treatment plans, psychiatrists were asked about the number of patients of each type they saw, whether involuntary treatment or referral was indicated and the anticipated length and frequency of treatment.

The sample

Essentially the same sample of practising psychiatrists has been surveyed for each phase of the Quality Assurance Project. In 1981, this sample was drawn from a consolidated list of the 1154 psychiatrists practising in Australia. By 1983 (the time of the schizophrenia questionnaire), 23 psychiatrists from the original sample had died, retired,

moved overseas, or withdrawn. These were replaced with predetermined substitutes, resulting again in a total sample of 193, from whom 173 codable questionnaires were received.

Results

Sample characteristics

Of the respondents, 78% were male. Their median age was 46, and half were between 39- and 55-years-old. The median year of qualification (MB.BS) was 1962; 71% had obtained their psychiatric qualifications in Australia, 22% in Britain, 3% in New Zealand and 5% elsewhere; 85% of the sample were members of the Royal Australian and New Zealand College of Psychiatrists and 36% had double qualifications (e.g. British DPM and Australian membership). Of the sample 58% identified themselves as being in private fee-for-service practice, the remainder being in salaried university or public sector employment. The majority (70%) of those in public practice worked in institutionally-based inpatient or outpatient facilities. The median percentage of time that the sample as a whole spent in clinical contact with patients was 80%, indicating that they were, as intended, a group of practising psychiatrists.

Method of analysis

Merely listing the frequency of various treatments recommended for each case is an exercise of little interest. Instead we have, in addition, compared treatments across and within cases across time. Where possible, we have commented on factors which may have influenced these differing recommendations. The method of analysis used was to test mutually orthogonal linear contrasts. For across-case comparisons, the contrasts tested compared the two acute patients (Cases 1 and 2) with the two chronic patients (Cases 3 and 4); then the two acute cases were compared with each other to see if treatment strategies differed according to premorbid history; and finally, the two chronic cases were contrasted to investigate differences in response to the chronicity of residual impairment. The within-case contrasts investigated differences between recommendations made initially and those made one month later, when the patient had improved (this comparison could not be made for Case 4); and between initial recommendations and those made three months later, when the patient had failed to improve with the initial treatment recommended.

For the purpose of performing statistical tests, the contrast coefficients were applied to the logarithms of the cell frequencies. This procedure derives from the log linear model of categorical data (Fienberg, 1978; Goodman, 1964) and is equivalent to testing whether the odds ratio corresponding to a particular contrast could have arisen by chance. Because the resulting analysis involved conducting multiple tests, the Bonferroni inequality (Miller, 1966) was used to adjust the critical value for each test, to ensure that the Type I error rate for the set of decisions was not inflated by the number of tests conducted (Bird, 1975).

TABLE I

The percentage of the sample who said, for each case, that (1) "involuntary treatment was indicated" and that (2) "they would refer" to other practitioners and that (3) they would treat in hospital initially, if improved after one month or if unimproved after three months treatment

	Case 1 %	Case 2 %	Case 3 %	Case 4 %	Average %
(1) Involuntary treatment indicated	17	5	56	42	30
(2) Referral indicated	27	14	52	49	36
(3) Initial	60	47	92	25	66
If "improved"	19	4	5	—	9
If "unimproved"	48	52	27	41	54

Questionnaire results

Involuntary treatment, referral and hospitalisation

The proportions of the sample who indicated that they did not see involuntary treatment as warranted in a particular case, or who reported that they would refer the patient rather than treat themselves, are presented in Table I.

Analysis of these data revealed that psychiatrists were more likely to recommend involuntary treatment for the chronic than the acute cases ($z = 10.68$; $P < 0.001$). Of the two acute cases, involuntary treatment was more frequently seen as indicated for Case 1, whose premorbid development was poor, than for Case 2 ($z = 6.89$; $P < 0.001$). Within the chronic cases, psychiatrists were more likely to recommend involuntary treatment for Case 3, whose episodes were recurrent and disturbing to the family, than for Case 4, who was chronically delusional and disturbing to society ($z = 8.89$; $P < 0.001$).

Referral elsewhere was more likely for the chronic than the acute cases ($z = 8.27$; $P < 0.001$). Psychiatrists were significantly more likely to treat the good premorbid acute case than the acute patient with a poor premorbid history ($z = 3.02$; $P < 0.01$). The frequency with which in patient hospital care was recommended after initial interview, after one month presuming improvement, or after three months presuming no improvement, are given in Table I. The major unexpected finding was that, of the two chronic cases, Case 4, without family support and with a poor prognosis, was less often hospitalised ($z = 3.7$; $P < 0.01$).

Frequency of consultations

Psychiatrists were asked how many consultations they expected to have with the patient alone, the family alone, and conjointly in the first three months of treatment. The median values obtained for each case were compared but the differences were evaluated in terms of the overlap between confidence intervals around these medians

(Snedecor & Cochran, 1937). Psychiatrists expected to have more consultations with acute cases and their families than with chronic cases, more consultations with Case 2 than Case 1, and with Case 3 than Case 4. The overall difference in the frequency of consultations for the two chronic cases was purely a function of family and conjoint consultations, presumably because Case 4 was described as having lost contact with his family. The difference between consultation frequency for the two acute cases was more complex, with psychiatrists indicating that they would have more patient consultations with Case 2 than Case 1, but more family and conjoint consultations with Case 1 than Case 2. These differences cannot be explained in terms of the availability of the family, since both case vignettes indicated that the family was involved in the referral. Rather, the frequency of family and conjoint consultations appears to be a function of the implied degree of conflict between patient and family, since they were more frequently recommended for Cases 1 and 3, where tension was implied, than for Case 2, where the mother was described as being co-operative and concerned.

Treatment plans

In order to facilitate comparisons of the treatment plans recommended for the four cases, recommendations were grouped to allow the testing of a number of mutually independent contrasts. Firstly, with regard to pharmacotherapy, the contrasts compared the proportion of psychiatrists recommending a drug alone with the proportions recommending no drug, and those recommending a drug in conjunction with some form of psychotherapy. Then, amongst those recommending the use of drugs, comparisons were made of the proportions recommending low potency, high potency, and depot antipsychotic preparations.

Questions were then asked concerning the use of non-drug therapies. In retrospect, we did not think that the treatment glossary had allowed sufficient room, particu-

larly within the family therapy codes, for psychiatrists to demonstrate their preference for the newer family management techniques (e.g. Falloon *et al.*, 1982) recommended by the expert committee in schizophrenia (Quality Assurance Project, 1984). For the purpose of the treatment comparisons, therefore, recommendations for family therapy/counselling were combined with those for social interventions such as day programmes to give a joint estimate of family/social management. The contrasts evaluating non-drug therapies compared the proportions who recommended no non-drug therapy at all with those who did; the proportions recommending both psychotherapy and family/social techniques with those who recommended either procedure singly; and the proportions recommending psychotherapy with those recommending family/social interventions.

The percentage of the sample who recommended no drug, a drug alone, or a drug plus some form of psychotherapy are summarised in Table II. By far the most popular treatment was a combination of drug and other therapy. Initially, practitioners were more likely to recommend a drug alone than no drug, but the reverse was true when the patient had improved ($z=6.09$; $P<0.001$), and this was so for all cases. There was even an overall reduction in drug use when the patient failed to improve relative to initial presentation ($z=3.28$; $P<0.01$). The only significant difference between cases was that Case 4 was more likely than Case 3 to have a drug alone initially, whilst failure to improve resulted in a reversal in these proportions ($z=3.10$; $P<0.05$).

The percentage of psychiatrists whose drug therapy consisted of a low potency, high potency, or depot antipsychotic drug are summarised in Table III, for each case and each stage of treatment. Comparisons of the drugs recommended initially with those recommended following

improvements in symptomatology revealed that, overall, depot medications were recommended more frequently than either high or low potency short-acting antipsychotic drugs ($z=5.25$ $P<0.001$); this was more evident for Case 3 than for Cases 1 or 2. Recommendations for the two acute cases differed, in that high potency antipsychotics were more commonly recommended than low potency phenothiazines for Case 1 but not Case 2 ($z=3.64$; $P<0.01$), and that depot medications were relatively more commonly recommended for Case 1 than Case 2 ($z=3.56$; $P<0.01$).

The usage of depot medication was greater overall when the patient had failed to improve than initially ($z=5.24$; $P<0.001$), and this increase was proportionately greater for the acute than the chronic cases ($z=3.94$; $P<0.01$), although overall depot medication was more frequently recommended for the chronic than acute cases ($z=12.92$; $P<0.001$). Within the short-acting antipsychotic drugs, usage of high potency preparations was somewhat more common than of low potency ones for acute presentations of illness, while the reverse was true (although not to a level of statistical significance) for chronic patients ($z=2.85$). The only significant difference between the types of drugs recommended for the two acute cases was that depot medications were more common for Case 1 than Case 2 ($z=3.11$; $P<0.05$). Recommendations for the two chronic cases differed in that Case 3 was more likely to have low potency than high potency drugs recommended, while the reverse held for Case 4 ($z=3.84$; $P<0.01$), and that a failure to improve resulted in an increased frequency of depot medication for Case 3, but a decreased use of depot medication for Case 4 ($z=3.21$; $P<0.05$).

Non-drug therapies

The proportions of the sample who recommended only

TABLE II

The percentage of the sample recommending no drug, a drug alone, or a drug plus other concurrent therapy for each case and each stage of treatment: as initial treatment, if improved after one month of that treatment, or, if unimproved after three months of the initial treatment

	Case 1 %	Case 2 %	Case 3 %	Case 4 %	Average %
<i>No drug</i>					
Initial	1	4	1	1	2
Improved	13	29	6	—	16
Unimproved	9	6	9	7	8
<i>Drug alone</i>					
Initial	13	10	11	25	15
Improved	1	2	3	—	2
Unimproved	6	12	28	14	15
<i>Drug plus other therapy</i>					
Initial	86	86	88	74	84
Improved	85	69	91	—	82
Unimproved	85	82	63	78	77

TABLE III

The percentage of the sample recommending low potency, high potency and depot anti-psychotic drugs for each case and at each stage of treatment: as initial treatment, if improved after one month of that treatment, or, if unimproved after three months of the initial treatment

	Case 1 %	Case 2 %	Case 3 %	Case 4 %	Average %
<i>Low potency</i>					
Initial	32	49	27	6	29
Improved	25	46	11	—	27
Unimproved	15	21	16	6	15
<i>High potency</i>					
Initial	61	49	15	11	34
Improved	60	51	5	—	39
Unimproved	52	62	3	24	35
<i>Depot</i>					
Initial	7	2	58	83	38
Improved	15	3	84	—	34
Unimproved	33	17	80	70	50

TABLE IV

The percentage of the sample who recommended various categories of non-drug therapy for each case and each stage of treatment: after the initial interview, if improved after one month of that treatment, or, if unimproved after three months of the initial treatment

	Case 1 %	Case 2 %	Case 3 %	Case 4 %	Average %
<i>No drug</i>					
Initial	13	10	11	22	14
Improved	1	1	3	—	2
Unimproved	6	12	28	14	15
<i>Psychotherapy</i>					
Initial	16	35	12	22	21
Improved	9	81	4	—	31
Unimproved	8	39	19	16	21
<i>Family/social</i>					
Initial	18	5	43	38	26
Improved	40	1	65	—	35
Unimproved	48	14	26	48	34
<i>Both</i>					
Initial	53	49	34	18	39
Improved	49	17	28	—	31
Unimproved	38	36	27	22	31

drug therapy, psychotherapy, family/social treatment, and both are summarised in Table IV. Non-drug therapies were popular ($z=6.77$; $P<0.001$), particularly the combination of psychotherapy and family/social interventions rather than either treatment in isolation ($z=6.83$; $P<0.001$), and especially after improvement had begun

($z=3.45$; $P<0.05$). The differences across cases centred mainly on the relative use of psychotherapy versus family/social treatment: family/social therapy was more frequently recommended for Case 3 than either Case 1 ($z=3.34$; $P<0.05$) or Case 2 ($z=8.77$; $P<0.001$); and for Case 1 than Case 2 ($z=7.19$; $P<0.001$).

When the patient had failed to improve, the chronic patients were less likely than acute cases to have both drug and non-drug treatments recommended concurrently ($z=5.13$; $P<0.001$) or to have psychotherapy alone recommended ($z=4.61$; $P<0.01$). Further, the bias towards psychotherapy for acute patients and family/social treatment for chronic patients, which was evident at initial presentation, was less marked when patients had failed to improve ($z=3.13$; $P<0.05$). There were no significant differences between the non-drug recommendations for the two chronic cases, and the only difference between the acute cases reflected the fact that psychotherapy was recommended more frequently than family/social therapy for Case 2 but not Case 1 ($z=7.34$; $P<0.001$).

Discussion

Schizophrenia is a serious mental illness from which only about 25% of patients recover completely (Quality Assurance Project, 1984). The symptoms during the active phase are usually unpleasant and frightening for both patients and their families, and the residual impairments of functioning, even in those whose psychotic symptoms remit, are often so severe that patients are unemployable and require continual care. It is presumably psychiatrists' recognition of these difficulties that prompted 36% to refer patients elsewhere for treatment, and for chronic patients, this figure was 50%. A significant proportion saw the need for involuntary hospitalisation for the two chronic cases, even though neither posed a danger to themselves or others—an attitude contrary to the thrust of current mental health legislation.

A summary of the recommended treatment plans at each stage, averaged across all cases (for the "improved" recommendations, averages represent only the first three cases), is presented in Table V. These figures indicate considerable consensus concerning the need for antipsychotic medication to control positive symptoms: only 2% of psychiatrists failed to include such drugs as an initial treatment component. There is also a recognition of the fact that some patients with schizophrenia may be able to do without medication when positive symptoms remit (overall 16% of the sample recommended no drugs when the patient had improved), particularly if the episode has had a sudden onset and a quick resolution, as was described for Case 2. Some sensitivity to the possibility that a failure to improve may indicate a resistance to drug treatment is indicated by the increased percentage recommending no drug in the "unimproved" (8%) than the initial (2%) treatment stage.

Recommendations for particular types of drugs appear to be tailored to the individual patient. Besides the obvious preference for depot medications for the chronic patients, psychiatrists also demonstrated differential usage of low versus high potency antipsychotic drugs. These differences reflect the drug's sedative properties, because the less sedating high potency preparations were initially more common for Case 1 who was socially withdrawn than for Case 2 who was not. Psychiatrists showed a preference for thioridazine over chlorpromazine at *all* stages of treatment for Case 2 but for Case 1 they recommended chlorpromazine more frequently than

TABLE V
The percentage of the sample recommending various categories of treatment at each stage of treatment averaged over cases

	Initial %	Improved %	Unimproved %	Average %
<i>Drug therapy</i>				
No drug	2	16	8	9
Drug alone	15	2	15	10
Drug plus	84	82	77	81
<i>Type of drug</i>				
Low potency	29	27	15	24
High potency	34	39	35	36
Depot	38	34	50	41
<i>Type of non-drug therapy</i>				
None	14	2	15	10
Psychotherapy	21	31	21	24
Family/social	26	35	34	32
Both	39	31	31	34

thioridazine, even though skin photosensitivity is a problem in Australia.

Comparison of the dosages recommended for the most commonly recommended preparations, expressed in chlorpromazine equivalents and averaged over all cases (Table VI) revealed, as might be expected, lower dosage recommendations when the patient had improved. Thus, practising psychiatrists appear to agree with the expert committee in schizophrenia (Quality Assurance Project, 1984) that "the guiding principle in drug therapy is to get the dosage as low as possible". Also in accord with the recommendations of the expert committee is the fact that only 15% of the sample recommended that antiparkinsonian medications be used prophylactically, i.e. as an initial treatment.

Whilst antipsychotic drugs were seen as a necessary part of treatment, only 10% recommended that a drug be used without concurrent non-drug therapy, and non-drug treatments were seen as particularly important when the patient had shown improvement within a month (only 2% then recommended a drug alone), indicating recognition of the need for treatment to continue past the point when active psychotic symptoms are in remission.

Despite the lack of empirical evidence for the effectiveness of psychotherapy for patients with schizophrenia (Quality Assurance Project, 1984), more than half of the sample (58%) did recommend psychotherapy, either alone or in conjunction with family/social interventions. However, many of these recommendations were for Case 2 who, even initially, and especially when improved, showed insight into her condition; indeed, 98% recommended psychotherapy once she had improved. There is clearly a strong belief in the importance of individual psychotherapy amongst Australian psychiatrists. Moreover, for the other cases, the category of psychotherapy selected from the glossary was nearly always "supportive psychotherapy", which was defined as the use of "empathy, guidance, support and reasoning... to decrease anxiety in order that the patient may deal more effectively with real-life situations". Given this definition, it is just possible that psychiatrists intended some of their psychotherapy sessions to include counselling concerning the nature of schizophrenia, the goals of

treatment, the importance of drug therapy, and the need for the patient and family to establish independent social networks, which is emphasised by the family management approach of Falloon *et al* (1982). In this sense, there may be some overlap and/or common intention in recommendations for supportive psychotherapy and family counselling.

The treatment recommendations indicated that psychiatrists are aware of the evidence implicating the "expressed emotion" of the families of patients with schizophrenia in their relapse (e.g. Brown *et al*, 1972). Family/social interventions were the non-drug therapy of choice for both Cases 1 and 3, where the vignettes described family members as being variously critical, hostile, and overprotective—factors defining high expressed emotion. Moreover, recommendations for family/social treatment for Cases 1 and 3 increased when the patients had improved relative to initial presentation, indicating a recognition of the need to continue family intervention procedures, even when psychotic symptoms have improved, if relapse is to be prevented.

Conclusion

This survey revealed considerable consensus among practising psychiatrists in Australia as to the appropriate management of schizophrenia. Antipsychotic drugs, in combination with either supportive psychotherapy or family/social intervention procedures, were recommended by almost all psychiatrists who were prepared to treat the types of patients described in the questionnaire. There was less consensus concerning the appropriate treatment situation for patients suffering active symptoms, perhaps reflecting differences in the availability of outpatient or day patient services. Psychiatrists generally agreed that hospitalisation should be brief when improvements in symptomatology are evident.

Psychiatrists exhibited some reluctance to treat patients suffering from chronic schizophrenia. This is understandable in terms of the poor prognosis evident for such individuals. But the costs of schizophrenia to the community will remain high unless attention is devoted to increasing psychiatrists' confidence in their ability to manage such patients.

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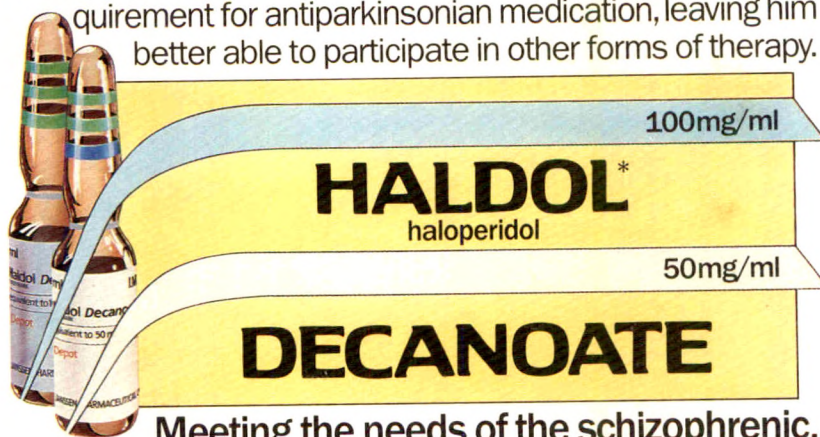


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Post-Traumatic Stress Disorder Following Combat Exposure: Clinical Features and Psychopharmacological Treatment

A. BLEICH, B. SIEGEL, R. GARB and B. LERER

Post-traumatic stress disorder may follow combat stress or civilian psychological trauma. In 25 retrospectively studied patients, symptoms were severe in terms of number of DSM-III items fulfilled, chronicity, and severity of psychosocial disability. Antidepressants had good or moderate results in 67% of cases treated, but major tranquilisers were much less effective; response to drug treatment was not clearly related to somatisation symptoms, significant depression, or panic attacks. Pharmacotherapy appeared to have had a positive impact on psychotherapy in 70% of cases.

Post-traumatic stress disorder (PTSD)—a syndrome manifested months or years after the event by people who have been exposed to significant psychological trauma or stress—has been increasingly recognised as a public health problem of major proportions (Keane & Fairbank, 1983). It occurs most frequently in soldiers who have suffered acute combat reactions and thereafter develop chronic symptoms, which may result in severe social and occupational incapacitation (Figley & Leventman, 1980). PTSD may also occur among civilians who have been victims of natural catastrophes, criminal assaults, rape, industrial or traffic accidents, and terrorist attacks (Horowitz *et al*, 1980).

DSM-III (American Psychiatric Association, 1980) proposed an empirical clinical definition of the PTSD syndrome which is considerably influenced by the observations of Kardiner (1941). The characteristic symptoms involve re-experiencing the traumatic event, numbing of responsiveness to or reduced involvement with the external world, and a variety of autonomic, dysphoric, or cognitive symptoms. Beyond the core features of the syndrome, related to re-experiencing the original traumatic event, the clinical picture encompasses a number of symptoms of depression and anxiety. These include diminished interest in work and activity, sleep disturbance, guilt, difficulty in concentrating, hyper-alertness, and exaggerated startle responses. It is therefore highly plausible that psychopharmacological agents which are effective in alleviating symptoms of depression or anxiety might be clinically efficacious.

The empirical use of psychopharmacological agents by many clinicians treating PTSD patients has been only sporadically reported in the literature, and no controlled trials have been published. Hogben &

Cornfield (1981) reported five cases successfully treated with phenelzine (45–75 mg/day): traumatic dreams, flashbacks, startle reactions and violent outbursts were all improved, while the drug appeared to enhance psychotherapy by promoting abreaction. Walker (1982) also observed positive results with phenelzine in three PTSD patients, traumatic dreams and flashbacks being particularly improved, while Levenson *et al* (1982) reported therapeutic effects of phenelzine (75 mg/day) in a single case: spontaneous, intense preoccupation with the traumatic experience was attenuated. Shen & Park (1983) reported a single patient who responded to phenelzine (105 mg/day) but complained of sexual inhibition, which continued when he was switched to tranylcypromine. Burstein (1984) administered imipramine (50–350 mg/day) to ten patients with PTSD of recent onset, and observed significant improvement in forced recollections, sleep and dream disturbance, and flashbacks; the relatively quick onset of action suggested a specific mechanism of the drug on sleep and dreaming. A similar positive effect of imipramine on post-traumatic night terrors was reported by Marshall (1975).

The present report summarises the clinical features and response to a number of drugs, particularly antidepressants, of a group of 25 PTSD patients referred to the PTSD Unit of the Israel Defence Forces Psychiatry Division.

Method

Clinical notes of patients referred to the PTSD Unit were retrospectively reviewed. The diagnosis of PTSD, as made by the treating clinician, was re-evaluated on the basis of DSM-III criteria, and 25 patients fulfilling these criteria

were identified. Details of the clinical picture, drug treatments administered, and response to each of the treatment agents were summarised. The summaries were reviewed by three clinicians; two of these have been involved in treating a number of the patients, while the other had no direct familiarity with the clinical material. A consensus regarding response to treatment was established in each case. Retrospective judgments as to the interaction between drug treatments and concurrent psychotherapy were obtained by contacting the therapist involved, who was asked to assess, using his notes, whether drug treatment had had a significant effect on the therapy process and to define the nature of this impact.

Results

Population

The mean age of the 25 patients in the sample was 32 years (range 20–42); all were males, all were married, and all were on reserve duty at the time of the traumatic event. The average length of time intervening between the traumatic event and referral for treatment was 8 months (range 1–20). Seven patients had been treated in the field by psychiatric units for acute combat stress reaction, while 18 were new referrals to the Mental Health Division. In 17 cases the traumatic stressor was an event or series of events that occurred on the battlefield: sustained artillery barrage (nine cases), open combat (five cases), or wounds or fatalities sustained by others in the same unit (three cases). In four cases the stressor was an encounter with bodies of soldiers killed in action; in two cases it was acts of sabotage; and in two cases it was traffic accidents in the operational zone. At the time of referral none of the subjects were able to continue soldiering, and reserve duty had been temporarily deferred; all had significant impairment of occupational adjustment and general psychosocial functioning.

Clinical symptom picture

The clinical symptom picture at the time of referral of each patient was well documented in the notes. Table I summarises the frequency of each of the DSM-III items in the patient population, as well as additional clinical features not encompassed in the PTSD diagnostic criteria. Intrusion symptoms such as sleep disturbance, recollections of the event, traumatic dreams, and hyper-alertness or startle response were equally represented among the most frequent clinical features, along with avoidance symptoms such as decreased interest in significant activity, avoidance of activities that evoke recollections of the event, and detachment from others. The severity of the clinical syndrome in the group is indicated by the fact that 17 of the 25 patients fulfilled ten or more of the thirteen DSM-III criteria; seven patients fulfilled 7–9 of the criteria, and only one less than seven criteria. (The minimum number of criteria required for the DSM-III diagnosis is five.)

Of those clinical features not encompassed in the DSM-III criteria, somatic complaints were most commonly reported; these were usually multiple and ill-defined, although in some cases dominant symptoms were

TABLE I
Clinical symptom picture in 25 PTSD patients

<i>DSM-III items</i>	<i>No. of Patients</i>
Re-experiencing of trauma	
Recollections of the event	23
Dreams of the event	22
Re-experiencing the event	5
Numbed responsiveness	
Decreased interest	24
Detachment or estrangement	22
Constricted affect	10
Symptoms not present before trauma	
Sleep disturbance	24
Avoidance	23
Hyper-alertness/startle response	20
Memory impairment/concentration difficulty	19
Re-intensification by exposure	18
Guilt-feelings	12
<i>Non-DSM-III items</i>	
Somatic symptoms	16
Significant depression	6
Panic attacks	3
Alcoholism (suspected)	2

identified—most commonly headache (seven cases), impotence (five cases) and chest pain (four cases). In two cases with marked somatisation, the symptoms were sufficiently pronounced and numerous to satisfy DSM-III criteria for somatisation disorder. Significant depression was documented in six cases; all fulfilled DSM-III criteria for dysthymic disorder, but not for major depression. Three patients reported panic attacks which fulfilled DSM-III criteria for panic disorder. In two cases, alcoholism was suspected but could not be confirmed; there was no evidence for any other form of substance abuse in any of the patients.

Response to pharmacotherapy

Table II summarises the outcome of all the medication trials undertaken in the patient group which exceeded two weeks in length. In 17 cases there was only one trial per patient, in 4 cases two, and in 4 cases three. Except for one patient who had amitriptyline together with methotrimeprazine combined treatments were not used. Treatment outcomes were rated as good if there was substantial and sustained symptomatic improvement and enhanced psychosocial functioning, and as moderate if these parameters of improvement were incomplete or if effects were transient in spite of continued treatment.

There were a total of 28 antidepressant trials. Fourteen patients received amitriptyline; dosages averaged 139 mg/day, and the average length of treatment was 6.5 months.

TABLE II
Trials of psychopharmacological agents in 25 PTSD patients

Drug	Treatment response			Total
	Good	Moderate	Poor	
Antidepressants				
Amitriptyline	4	9 ¹	1	14
Doxepin	1	3	3	7
Maprotilene	—	1	1	2
Clomipramine	—	1	1	2
Phenelzine	—	—	2	2
Trazadone	—	—	1	1
Total	5	14	9	28
Major tranquillisers				
Methotrimeprazine	—	—	3	3
Thioridazine	1	1	—	2
Sulpiride	—	—	2	2
Fluphenazine	—	1	—	1
Total	1	2	5	8
Minor tranquillisers				
Chlordiazepoxide	1	—	—	1
Total (all drugs)	7	15	15	37

1. Combined in one patient with methotrimeprazine (50–75 mg/day).

Four patients had a good clinical response to amitriptyline, characterised by a complete alleviation of symptoms and significant improvement in psychosocial functioning, while another eight had moderately good responses, only one patient showed a poor response to amitriptyline. Doxepin was used in seven cases (mean dose 100 mg/day), with an average length of treatment of seven weeks; one patient manifested a good response and three a moderate one; while three others responded poorly to this treatment. Good responses were not obtained with any of the other anti-

depressants tried, although one subject had a moderate response to maprotilene (150 mg/day) and one a moderate response to clomipramine (150 mg/day). Neither of the two cases in whom phenelzine (30–45 mg/day) was tried showed any therapeutic benefit, although in one of these cases treatment was only for two weeks.

In those patients who responded well or moderately to antidepressants, the most prominent beneficial effects were on sleep, traumatic dreams, hyper-alertness, and startle response, memory, and concentration difficulties. One patient with enuresis reported disappearance of the symptom, along with overall general improvement, following doxepin treatment. Only two of the five patients with impotence had a therapeutic response, which was moderate in both cases. There was no clear relationship between presence or absence of somatisation, significant depression, or panic attacks and response to antidepressant treatment; neither did the severity of the syndrome, as indicated by chronicity and number of DSM-III items present at intake, appear to have any clear relationship to outcome.

Major tranquillisers were tried in eight patients. Dosages tended to be low (methotrimeprazine, 25–75 mg/day; thioridazine, 50–100 mg/day; sulpiride, 100–400 mg/day; fluphenazine, 5 mg/day). One patient had a good response to thioridazine (100 mg/day for two months), while one responded moderately well to thioridazine (50 mg/day for one month) and fluphenazine (5 mg/day for one month) at different times; neither patient was psychotic. The symptom picture in both these subjects was more activated, with prominent insomnia, nightmares, explosiveness, and hyper-vigilant symptoms. Major tranquillisers yielded a poor response in five of the eight cases. One patient was treated with chlordiazepoxide (40 mg/day for one month) with good results; his symptom picture resembled that of the two patients who responded moderately well to major tranquillisers.

Interaction of drug treatment and psychotherapy

Twenty of the patients were receiving psychotherapy at the time that drug treatments were instituted; in four cases, the prescribing doctor was himself the psychotherapist. In ten cases, the psychotherapy was defined as 'supportive', and in

TABLE III
Interaction of psychopharmacological treatment with psychotherapy

Type of interaction	Dynamic psychotherapy (n = 10)	Supportive psychotherapy (n = 10)
Positive interaction		
Increased motivation	8	6
Working-through enhanced	6	1
Negative interaction	0	1
No interaction	2	3

the rest there was a major emphasis on dynamic processes, uncovering, or abreaction.

Table III summarises the retrospective assessment by the psychotherapist of the perceived effect of concurrent drug treatment on psychotherapy response: there was felt to have been a positive interaction with psychotherapy in 14 of the 20 cases, with enhanced ability to 'work through' in 7. In all, motivation for treatment was believed to have been increased.

Discussion

In terms of severity of symptoms, chronicity, and psychosocial disability, the patients in this sample may be categorised as suffering from disabling post-traumatic syndromes. Their central clinical features closely resemble those described by Horowitz *et al* (1980), as well as those found in an epidemiological study of Israel combat veterans (Solomon, 1986). Recollections of the traumatic event were a major feature of the clinical picture, and were manifest both in the waking state and during sleep. However, relatively few patients reported suddenly acting or feeling as if the traumatic event was recurring (DSM-III, item B3), and there was a relatively low incidence of survivor guilt (DSM-III, item D2), although the unstructured interview techniques used may have reduced the emphasis on both these items, which in many cases require active elicitation. Symptoms indicative of psychic numbing and diminished responsiveness were as prominent as activation-type symptoms, with the exception of constricted affect (DSM-III, item C3). This item is less amenable to uniform clinical definition, which may explain its relatively lower frequency.

There was a major difference between the present subjects and Vietnam veterans with PTSD previously reported (Sierles *et al*, 1983; Yager *et al*, 1984): the virtual absence of alcoholism, drug abuse, and anti-social personality features. Twenty-one of the 25 veterans studied by Sierles *et al* (1983) met definitive diagnostic criteria for substance abuse, as opposed to 2 out of 25 subjects with suspected but unconfirmed alcoholism in the present sample. Antisocial personality was not diagnosed in any of our sample, and explosive, aggressive, or impulsive behaviour, while noted in some of the cases, were not major clinical problems. The combination of such behaviour traits with substance abuse has led to an almost stereotyped perception of the Vietnam veteran with PTSD, from whom our subjects differed markedly (Yager *et al*, 1984). Possible explanations for these differences are still purely speculative. It is, however, conceivable that the social environment of combat veterans with PTSD may strongly influence the symptomatic

expression of the syndrome beyond these core features common to patients from different cultures. Both alcoholism and drug abuse are considerably less prevalent in Israeli than in other societies, and this factor may be important. A further consideration is that public attitudes in Israel tend to be strongly supportive of the veteran, particularly one disabled in combat, and this may diminish any tendency towards alienation and anger.

Sierles *et al* (1983) emphasised the importance of considering other DSM-III diagnoses in patients with PTSD, and saw this approach as having important implications for treatment. However, other than substance abuse and anti-social personality, the incidence of additional diagnoses such as somatisation disorder, endogenous depression, and organic mental syndrome was relatively low in their sample. Dysthymic disorder was diagnosed in 24%, panic disorder in 12%, and somatisation disorder 8% of our patients; there did not appear to be a clear relationship between presence or absence of additional DSM-III diagnoses and response to drug treatment, though the small sample size and retrospective nature of the study preclude any definitive conclusions in this regard. This issue has important treatment implications, but its resolution will have to await prospective studies in larger groups of subjects.

Among the psychotropic drugs we administered, antidepressants yielded the best results. Amitriptyline was the most frequently used antidepressant and the most effective, but on the whole, responses to antidepressants tended to be partial or transient. Major tranquillisers had good or moderate results in only two of the eight patients to whom they were administered: their major effect appeared to be one of sedation, and in the absence of psychotic symptoms they cannot be regarded as indicated in PTSD patients. The single patient treated with a minor tranquilliser, chlordiazepoxide, reported a highly favourable response.

Previous studies with psychotropic agents in PTSD have also been open ones, and have focussed almost exclusively on tricyclic antidepressants and monoamine oxidase inhibitors. While antidepressants improved intrusion-type symptoms in our patients, the most prominent overall effect was in alleviation of insomnia and general sedation: this was also the clinical impression in the few patients who responded to major or minor tranquillisers. The possible specificity of tricyclic antidepressants and monoamine oxidase inhibitors for PTSD has important implications not only for treatment but for our understanding of the possible neurobiological underpinnings of the disorder (Van der Kolk *et al*, 1985).

The positive interaction observed between drug treatment and psychotherapy is also of considerable practical importance, since in a disorder such as PTSD, where psychological antecedents are compelling, there may be a reluctance on the part of psychotherapists to interpose drug treatment into the therapy process. Previous reports on the impact of drug treatment on psychotherapy in PTSD have been essentially positive, although the enhancement of abreactive processes noted by Hogben & Cornfield (1981) has not been emphasised by other authors (Levenson *et al*, 1982). The positive impact in the present sample appears to have been primarily mediated by improvements in sleep and overall

sedation, leading to a more positive and motivated approach to psychotherapy on the part of the patients, but in addition drug treatment was felt to have actively enhanced the psychotherapy process in terms of amenability to uncovering and working-through in a minority of subjects. Although impressionistic in nature, these findings suggest that drug treatment should be actively considered in planning treatment strategy.

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Childhood Encopresis extended into Adult Life

The late prognosis of childhood encopresis is unclear: our literature reviews have failed to reveal any studies of it. Halpern (1977), discussing the effectiveness of treatment, states that this is difficult to evaluate "since encopresis is a self-limiting symptom". Bellman (1966) followed up 186 children with encopresis seen at two Stockholm clinics; their age range at follow-up was 6-19 years. She found a steady decline in encopresis from the age of six in boys and eight in girls. It disappeared by the age of 16, although there were two relapses—one at 17 and one at 19.

Encopresis can occur in psychotic adults and in those who are severely mentally handicapped (Bellman, 1966). The only reported case of an adult encopretic who did not fall into either of these groups was that of a 36 year-old 'infantile neurotic', who had been encopretic until the age of 13-14 and relapsed at 31, after he had "broken with his dominating mother and sister owing to the bad relations between them and his wife" (Gurewitsch, 1932).

As part of a community-based treatment programme for childhood encopresis, an approach was made through the local media to see if there were adult sufferers prepared to come forward. The replies came mostly from people who had become incontinent in old age and from the relatives of chronically incontinent, mentally handicapped adults. There were three possible exceptions to this, only one of whom finally accepted an offer to be seen.

Case Report

Mrs A, aged 40, still suffered from encopresis which had started when she was 10, after the sudden deaths of both her parents from cardiovascular disease. She was subsequently brought up by older siblings, most of whom died of cardiovascular disease at an early age. Her clear recollection of her symptoms starts at the age of 12; episodically from then, two or three times per year (mainly at moments of great anxiety) she would experience an urgent call to stool, and would pass a small amount of faeces into her clothes. She disliked school intensely, but has no recollection of being embarrassed by her soiling there. She married at the age of 18, without her husband being aware of this symptom, and it was not intrusive in the early years of their marriage. Two children were born, three and five years later. Subsequently, the symptom became more obtrusive, and led to increasing limitation in her social activity.

Relatively little treatment had been sought or offered. In an exacerbation eight years ago she was seen in a gastroenterology clinic, but it seems unlikely that she made her true history clear. Ordinary investigations did not suggest an organic cause for her disorder.

She was prompted to reply to our news article by her most recent exacerbation, which originated at a time when her husband experienced symptoms suggestive of serious cardiac disease and her son had just left home. At interview, she was apprehensive and unforthcoming; she gave the dreadful details of her family history with little expressed emotion, although somatic signs of anxiety and arousal were very evident. She regarded herself as sensitive and said she cried readily, though there were no somatic symptoms of depression. She experienced a mood of sadness and apprehension, which she thought might be either the cause of or an effect of her encopresis.

Her husband saw the symptoms as becoming more and more intrusive in social and domestic life and, although he was more placid since his cardiac disorder, he still felt irritated and controlled by his wife's unusual symptom.

Treatment was undertaken using a combination of behavioural and psychotherapeutic approaches; the former did not suit her personality structure, and five monthly meetings were arranged which were more psychotherapeutic. The focus was on grief work, and during the sessions she was able to weep over her childhood losses. Following these sessions, her husband reported that she had been radically transformed and was smarter, brighter, and happier. Her occasional encopresis persisted, but her mental state remained excellent over a period of a year.

Comment

A diagnosis of social phobia, as described by Marks (1969), could explain much of this history, the symptom of encopresis being serviceable in controlling the amount of exposure to social situations. However, encopresis does not seem to have been reported as a presenting symptom in social phobia. In addition, the urge to defecate, and occasional encopresis, can occur when the patient is at home alone and not overtly anxious. The symptoms of encopresis and social phobia thus appear to have arisen independently, but with the encopresis being used as an added justification for her social withdrawal. The stool passed is normal in consistency, and she is capable of retaining most of the faecal matter which is in the rectum: Kanner (1953) used the term 'partial encopresis' to describe this type of soiling pattern.

Parental death has been noted as a trigger to the onset of encopresis (Lehman, 1944), and regression to faecal incontinence following the sudden deaths of both parents could have been the mechanism which produced encopresis in this patient. Encopretic children have been described as inhibited, slow to warm up, and disliking school (McTaggart & Scott, 1959)—features which could be applied to our patient. Bawkin & Bawkin (1960) have described how encopretic children may remain clean for several months and then have a soiling episode.

Throughout her lifetime with this symptom the patient made little effort to obtain any advice or treatment, and her own management consisted of

avoiding situations where she could be humiliated by it. Holman *et al* (1982), in a follow-up study of children with encopresis who had been treated in a psychiatric in-patient unit, comment that the families rarely sought further help for the problem.

We wonder in how many other people encopresis has extended into adult life. Certainly, our experience of dealing with adults with late onset bowel incontinence who responded to us leads us to suppose that people will tolerate their symptoms for a long time rather than risk the humiliation of disclosure, although the media gave some help, editorial censorship precluded a wider exposure of our interest in helping these sufferers.

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Changes in Mood, Appetite and Psychomotor Retardation in Depressed Patients Given ECT

Clinical experience suggests that treatment with electroconvulsive therapy (ECT) produces an early improvement in psychomotor retardation. Warnings have been given of the increased risk of suicide when ECT-treated patients recover motor activity before their depressed mood is alleviated (Slater & Roth, 1977).

In a recent study (Hibbert *et al*, 1984) the

Montgomery-Åsberg depression scale (MADS) was used to investigate changes in severity of a variety of depressive symptoms over time. It was found that whereas most of the items on the MADS co-varied closely, 'biological symptoms' such as reduced appetite and reduced sleep did not show a clear relationship to other depressive symptoms. This suggested that the MADS might be an appropriate instrument

with which to investigate the clinical impressions quoted above concerning the rates of recovery of different depressive symptoms during ECT.

Method and Results

Ten patients who fulfilled criteria for major depressive disorder (Spitzer *et al.*, 1978) were recruited for the study. Five were taking antidepressant drugs, but were judged by their consultant psychiatrist to be non-responders. All subjects received right-sided unilateral ECT from an Ectron Mark IV duopulse machine, with methohexitone, atropine and succinylcholine being given before ECT.

The subjects were rated using a modified version of the MADS, which has been fully described by Hibbert *et al.* (1984). The scale for psychomotor retardation (Åsberg *et al.*, 1973) was included. Interviews were carried out daily, before the start of ECT and throughout the course of treatment; no reference was made to the previous days' rating. For each item of the MADS the mean number of ECTs required to produce a 50% reduction in pretreatment rating score was calculated; differences between the means were assessed by the Mann-Whitney test.

Items on the MADS measuring reduced appetite and psychomotor retardation improved most quickly, at rates significantly greater than those for reported and apparent sadness and inability to feel ($P < 0.01$) (Fig 1). Inability to feel improved at a rate similar to that of reported and apparent sadness (Fig 1), confirming previous observations that the three symptoms of depressed mood co-vary over time (Hibbert *et al.*, 1984). There was no relationship on any item between initial MADS score and rate of improvement.

Discussion

Our findings suggest that motor retardation and reduced appetite are the earliest depressive features to improve during a course of ECT. However, because of the limitations of our study, the results must be regarded as preliminary. Firstly, the number of patients rated was small, and some were on psychotropic medication, which could have interacted with ECT. Secondly, the study was conducted openly and rater bias cannot be excluded. However, we did not predict the rapid improvement in appetite which followed ECT, although it is consistent with an earlier report by Russell (1960).

Our observations could be explained if reduced appetite and psychomotor retardation were more sensitive to change than other items in the MADS. However, data reported by Montgomery & Åsberg (1979) do not support this possibility. It is recognised that retardation is a relatively infrequent symptom (Montgomery & Åsberg, 1979), which is difficult to rate reliably (Hibbert *et al.*, 1984). In our study, however, seven of the ten patients had obvious psychomotor retardation which responded well to ECT.

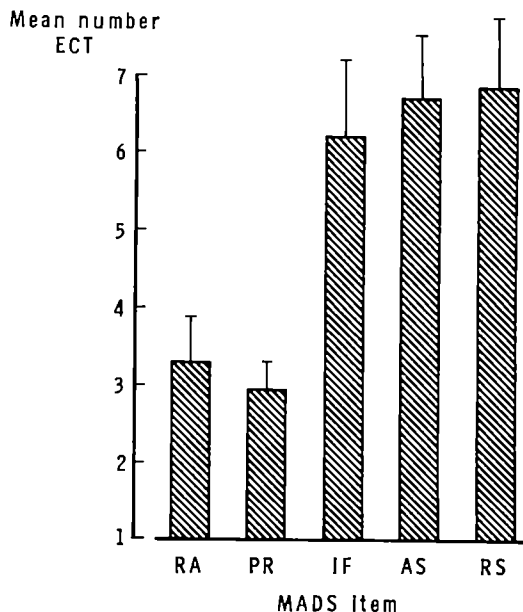


FIG 1 Mean number of ECTs required to produce a 50% reduction in MADS score (RA = reduced appetite, PR = psychomotor retardation, IF = inability to feel, AS = apparent sadness, RS = reported sadness). The error bars on top of each column show the SEM.

We cannot exclude the possibility that the changes we have observed in the remission of different symptoms are part of the general pattern of recovery from depressive illness and might also be seen, for example, following treatment with antidepressant drugs. However, it is recognised that certain cases of depression, particularly those with retardation and failure to eat and drink, are sometimes resistant to antidepressant medication but respond quickly to ECT (Kendall, 1981; Paul *et al.*, 1981). In animals repeated electroconvulsive shock, but not tricyclic antidepressants, produces a rapid increase in dopamine-mediated behaviours (Cowen *et al.*, 1980). In man, there is also evidence for increased dopamine-mediated responses following ECT (Costain *et al.*, 1982). Since dopamine pathways may play an important role in locomotion and feeding (Ungerstedt, 1979), it is possible that the early effectiveness of ECT in treatment of retardation and poor appetite is mediated by change in brain dopamine mechanisms.

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Clopenthiol Decanoate in the Management of Aggressive Mentally Handicapped Patients

The management of aggressive and disruptive behaviour in mentally handicapped patients is a severe problem, and it is widely accepted that drug therapy is only one component in the range of measures required for a comprehensive regime, although the use of depot phenothiazines is widespread (Craft & Schiff, 1980). The evidence for their therapeutic effectiveness in the mentally handicapped is usually empirical, and sometimes based on anecdotal evidence. Clopenthiol decanoate is widely used in the management of aggressive and disruptive behaviour in such patients, probably as a result of experience of the use of this drug in schizophrenic patients.

Carney & Rutherford (1981) reported a marked improvement in both aggressive and non-aggressive behaviour disorders in 20 chronic schizophrenic patients treated for 11 months with clopenthiol decanoate, while Visiwanathan & Rajhkowa (1982) stated that it was of undoubted value in the management of chronic in-patients, especially those with 'positive affective' symptoms such as hostility, aggression, suspiciousness, and irritability.

The only published report on the use of clopenthiol decanoate in mentally retarded patients (Yar-Khan, 1981) described a calming and behaviour-modifying effect in 15 subjects with

TABLE I
Behaviour and total number of recorded episodes of aggressive behaviour during the six months prior to starting and the six months on clopenthixol decanoate

Patient	Age	Sex	Six months before treatment	Behaviour and number (n) of recorded explosive incidents Six months after treatment	Final global rating
1	35	F	Shouts at staff, slams doors, antagonistic, jealous and impatient (n = 40)	Outbursts less severe, less frequent, participates in ward activities more (n = 1)	B ¹
2	33	F	Absconds, hits staff when apprehended, hits other patients indiscriminately (n = 31)	Less unpulskive but still absconds, able to concentrate in occupational therapy (n = 24)	S ¹
3	31	F	Runs to other wards, eats cigarette ends, agitated and aggressive to staff (n = 52)	Much calmer, long periods between aggressive outbursts (n = 11)	B
4	23	F	Emotionally unstable, attention seeking, aggressive if not cuddled, swears, spits, hits out at staff, resents male staff (n = 19)	Long periods of quiet and calm behaviour, only occasional outbursts (n = 11)	B
5	16	M	Spitful to staff and patients, unprovoked attacks (n = 47)	Less aggressive, shorter duration of aggressive episodes (n = 22)	B
6	20	M	Unprovoked attacks on patients and staff, bangs furniture, rips clothes, rolls down curtains, restless (n = 27)	Fewer aggressive episodes, patient more amiable between outbursts of aggression (n = 27)	B
7	21	M	Unprovoked attacks on patients and staff, destructive to furniture, clothing and windows (n = 18)	Outbursts less frequent, happier in-between outbursts (n = 9)	B
8	27	F	Bites fellow patients, excitable, overturns tables, and other furniture (n = 9)	No change in behaviour, more excited and disturbed (n = 15)	S
9	19	M	Restless, shouts, excitable, attacks and bites others, unprovoked outbursts of rage (n = 17)	Larger intervals between outbursts, enjoys other activities much more (n = 20)	B
10	32	M	Aggressive when depressed, agitated, wandering, incontinent, mood swings (n = 19)	Stable moods, no longer attacks other people, improved concentration and is more tolerant (n = 6)	B

¹B = better than before; S = same as before

aggressive, disruptive, agitated, and violent behaviour disorders. We report here preliminary observations of the clinical use of clopenthixol decanoate in the management of aggressive and disruptive mentally handicapped patients.

Method

Ten patients treated for behaviour disorders at the Stoke Park Group of Hospitals were included. This was the first group who received the drug there, because other types of medication had been unsuccessful in controlling their behaviour.

The Charge Nurse or Sister, in consultation with the ward staff, was asked to describe in detail any changes in patients' behaviour during the time of treatment with clopenthixol decanoate. A checklist questionnaire on the side-effects of the drug was also completed, and any side-effects were described in detail in the case notes. After six months, a final rating was made by the senior nurse as to whether the patient was better than before treatment, unchanged, or worse. The authors examined all the clinical notes made by the doctors and nursing staff during the period of treatment and for six months before treatment. Biographical information and details of medication both before and during treatment with clopenthixol were noted, along with the doses and side-effects of clopenthixol recorded in the case notes. The recorded incidents of explosive, aggressive outbursts were counted for the six month periods both before and after the start of treatment.

Results

Sample

The ten patients were all long-stay, ambulant, mentally handicapped in-patients, with an IQ range of 20-60, age range of 16-35, and mean age of 25.7. The sexes were equally represented. Three patients had epilepsy, and one was deaf. The behaviour disorders are shown in Table I. All patients had previously received drug treatments specifically aimed at controlling their aggressive behaviour. All had already been tried on oral phenothiazines, two patients had been given carbamazepine, and three had been treated with haloperidol, which was abandoned because of side-effects; lithium carbonate and fluspirilene had also been tried on two patients. However, none had been given depot phenothiazines. All other regular psychotropic medications were gradually withdrawn during the period of study.

Dosage and side-effects

The two psychiatrists used different starting doses, one used the manufacturer's recommended starting dose of 100 mg i.m., while the other began with 50 mg i.m. All three patients who received the recommended adult starting dose of 100 mg developed extrapyramidal side-effects (tremor, rigidity, and dystonia) in the first two weeks after starting treatment, which led to an initial increase in the number of episodes of aggressive behaviour. When these side-effects were treated with anti-parkinsonian drugs, the patients'

behaviour settled. None of the patients who received the smaller test dose showed any extrapyramidal side-effects. The maintenance doses were gradually built up, and at the end of the study the maximum weekly maintenance dose being given and tolerated was 300 mg.

The range of other side-effects observed included dry mouth (five patients) and a significant gain in weight (four patients, all females).

Therapeutic effectiveness

Eight patients were rated as being better than before, i.e. much calmer and more receptive to other therapeutic interventions such as behaviour modification and participation in ward activities. Six had a significant reduction in the number of recorded incidents of explosive aggressive outbursts during the treatment period (Table I). Two patients, both females, were rated the same as before treatment: one was an epileptic who became more unsettled during treatment, and another was a lonely and withdrawn girl who only became periodically aggressive when asked to join in activities of the ward. Both became so excitable and hyperactive during treatment that clopenthixol had to be stopped. It was generally felt that the patients who responded well to clopenthixol were those whose episodes of aggression were seen against a background of a chronic state of high arousal.

Discussion

Our observations indicate that clopenthixol decanoate may be useful in reducing aggressive and disruptive behaviour in mentally handicapped adults. This confirms the clinical impression of Yar-Khan (1981), who appealed for further study, and the need for a properly conducted controlled trial is clearly established. However, future studies will need to have an operational definition of what constitutes 'aggressive behaviour' and to standardise the methods of selection and assessment of patients. The nurses' assessments in this study were open to bias and not standardised, but it was interesting to see a dramatic reduction in the number of recorded episodes of aggressive behaviour during treatment with clopenthixol decanoate.

Any therapeutic response observed in a drug trial may be due to 'specific' or 'non-specific' effects of the treatment (Shepherd, 1975), so careful control of all the factors which can contribute to the observed response is essential. This calls for a careful record of any concurrent medication administered, and of any changes in the environment or life events experienced by each subject, because all these factors can contribute to the observed outcome. For instance, most patients who responded well to clopenthixol decanoate in these preliminary observations had their ward environments renovated and upgraded during

the period of treatment. This experience alone could have non-specifically contributed to improved behaviour. A prospective controlled trial, involving subjects selected according to specified criteria, would be the most appropriate method of study.

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Paranoid Reaction and Underlying Thyrotoxicosis

Woodbury (1918) described the manifestations of Graves' disease as fatigue, irritability, intolerance to heat, fine tremor, restlessness, insomnia, excitability, labile emotional disposition, nervousness, weight loss, palpitations, doubts, and fears. Similarly, Sachar (1975) included nervousness (manifested as apprehension, restlessness, and inability to concentrate), marked emotional lability, and hyperkinesia as the most common presenting symptoms. That fatigue, irritability, emotional instability, and excitability are all common features of the hyperthyroid state, as are episodes of severe and often disabling anxiety, is therefore, accepted. On the other hand, no clear picture exists concerning any fixed constellation

of psychotic symptoms or progression of psychiatric disease. The psychiatric presentations vary from a delirious state to periods of hyperexcitability simulating mania that alternate with periods of exhaustion and depression. Greer & Parsons (1968) reported that some patients do, however, develop a clear schizophrenia-like picture, closely associated with the course of hyperthyroidism, with prominent paranoid symptoms and no pre-morbid history of psychosis.

We present a case of paranoid reaction in association with hyperthyroidism, which responded to neuroleptic treatment and the restoration of the euthyroid state.

Case History

A 43 year-old woman had been unwell for five days before admission: she had become increasingly restless, irritable, and unable to concentrate or sit still, according to her 19 year-old daughter (an unmarried mother living with her parents). The patient kept telling the daughter that there were problems with her marriage, and also became very critical about the way in which her daughter dealt with the baby. The patient became so agitated that she was taken to her general practitioner; there, she became convinced that her husband was hiding from her in the surgery and that she could hear his voice in the distance, talking about her. She was restless and agitated, but co-operative. She seemed apprehensive, and expressed paranoid ideas about her husband. She complained of feeling tired and unable to sleep; her sensorium was clear, but she lacked insight into her condition.

There was no evidence of social or psychiatric problems prior to this episode, but the patient described herself as a very "worrying type" of person, who tended to 'bottle up' her feelings. The patient's mother had a thyroidectomy for thyrotoxicosis, but there was no family history of psychiatric illness. On physical examination, she had a smooth enlarged goitre with a bruit. She had slight tremor of the hands, exophthalmos with lid-lag, and a resting pulse of 105/min. Her laboratory results indicated elevated thyroid indices, with T_3 uptake of 39.2% (normal range 25–35%), thyroxine 289 nmol/l (normal range 55–145 nmol/l), free thyroxine index 107 (normal range 18–46). Consultation with a specialist in endocrinology confirmed the diagnosis of hyperthyroidism. Treatment with carbimazole (15 mg t.d.s.), propranolol (20 mg t.d.s.) and chlorpromazine (50 mg t.d.s.) was started. The paranoid symptoms resolved dramatically after two months, she was euthyroid within four months, and on review after nine months she still remained symptom-free on reduced dosages of carbimazole (10 mg), propranolol (20 mg b.d.), and chlorpromazine (50 mg once/day).

Discussion

This case serves as a reminder of psychiatric presentations of thyrotoxicosis, and highlights its responsiveness to phenothiazine treatment. However, the mechanism by which hyperthyroidism causes psychiatric disorder is still not clear. Various views have been put forward on pathogenesis, and claims have been made that the behavioural, neurotic, and psychotic manifestations of hyperthyroidism are related more to the biochemical abnormalities associated with the disease than they are to the patients' previous personality. Ettigi & Brown (1978) stated that the psychiatric disorder is probably not caused by the cellular hypermetabolic state alone, since hyperthyroidism is also associated with an increased sensitivity of central nervous system neurotransmitter receptors to catecholamines, depressed monoamine oxidase activity levels, and increased norepinephrine turnover rates. The acute paranoid reaction in our case appears to have been associated with hyperthyroidism, but the patient's anxiety-prone personality may also have been a contributory factor. We assume that a combination of metabolic, biochemical, pre-morbid personality, and emotional factors were implicated in the causation of this psychiatric disorder. Our observation confirms the suggestion by Burch & Messerey (1978) that if a hyperthyroid patient developed psychotic symptoms, this can often be controlled with chlorpromazine.

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Multiple Pathology in a Mentally Handicapped Individual

A doctor is trained, perhaps over-trained, to try to limit his diagnosis to a single disease process or entity. This may lead on occasion to a somewhat blinkered approach to the management of patients.

The following case illustrates multiple and concomitant pathology in a single individual.

Case Report

Miss JF now 40, was admitted in 1951, aged seven to Leytonstone House, which was then a typical, traditional, custodial institution for the mentally handicapped. She was a first child. Her mother was 29 at the time of her birth, and had suffered from hyperemesis gravidarum during pregnancy; she was admitted to hospital soon after going into labour, which lasted four days. JF developed petit mal attacks at the age of three months, when she had already developed whooping cough. Her developmental milestones during the first nine months were normal, although she was described by both parents as difficult, moody, and fretful. She babbled at ten months, but her speech never developed beyond a few words. By 18 months she was dry and clean, but during her moody periods she would smear her faeces—a habit which continued till she was seven.

The parents noticed that from the age of two she was fond of popular tunes and was often heard humming and babbling them; they regularly played music to quieten her down. Both parents vividly recollected that their daughter never braced herself on being taken up into their arms, during her second and third years she was emotionally unresponsive to their attentions, including cuddling, and became upset by the slightest digression from her daily routine. They also noticed that although she occasionally bit her hands, she never seemed to feel pain, even when she injured herself, to the extent sometimes of producing cuts which bled, this habit still recurs during periods of frustration. On admission, she was diagnosed an 'imbecile'; psychological testing placed her IQ at 30 (Terman Merrill and Merrill Palmer scale). She was at that time restless and easily distracted. Her self-mutilating tendencies continued, as did her aggression to others. At the age of eight she developed grand mal epilepsy, and these seizures still occur at an average rate of one per month.

At the age of 30 she developed a depressive illness marked by tearfulness, social withdrawal, loss of appetite, and

disturbed sleep. She responded well to amitriptyline (75 mg nocte), but soon after her recovery (which took six weeks) became overactive—dancing, humming, and frequently biting other people. Amitriptyline was stopped and chlorpromazine (100 mgs t.d.s.) was given for three months, but there was no significant change. During the fourth month, however, her hypomania subsided.

Two years later she again relapsed into a state of excitement and aggressive behaviour and was again put on chlorpromazine. She recovered from this episode after about five months. Six years later she developed a severe depression which responded to dothiepin (75 mg nocte). Again, her recovery was followed by an episode of elated mood with disruptive and aggressive behaviour, which this time responded to haloperidol (10 mgs t.d.s.).

After a diagnosis of manic-depressive psychosis, she was started on lithium carbonate (800 mg nocte). Since this regime was commenced, she has had over three years free of affective disorder, and has become much more manageable. In February 1984, an EEG showed "marked abnormal record, slow sharp waves over the temporal region with right sided emphasis".

Comments

Boullin *et al* (1970) stated that infantile autism could be due to a genetic defect causing disturbance in tryptophan metabolism, while Post (1980) has pointed out that manic-depressive illness may be due to a single dominant gene with incomplete penetration, leading to a disturbance in the metabolism of tryptophan. When infantile autism and manic depressive illness occur together, it is reasonable to assume that the same gene may be responsible for both, since the same metabolic error may be shared. In this particular case, epilepsy may indicate an organic factor which could be a component of infantile autism. The possible relevance of abnormal tryptophan metabolism to the above syndromes should be considered in future research strategies.

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Books Reconsidered

Sanity, Madness and the Family: R. D. Laing and A. Esterson

In this series, Andrew Smith (1982) reconsidered R. D. Laing's *The Divided Self* (1960). In his sensitive, wideranging review of Laing's first book, he added to the growing weight of opinion in some psychiatric circles that it is a major though idiosyncratic contribution to our understanding of schizophrenic psychopathology. It is after all on the Royal College of Psychiatrist's current psychotherapy reading list. *Sanity, Madness and the Family*, first published in 1964, and the work which accompanied it, remains outside mainstream psychiatry yet I will argue has had an impact exceeding that of *The Divided Self* on today's practice.

The first Tavistock publication was subtitled, *Volume 1, Families of Schizophrenics*, with the implication that further volumes would deal with families of other psychiatric patients or normals. The preface to the second Penguin edition (1970) explains that on reflection the use of a control group would not help the authors answer *their* question, namely, "are the experience and behaviour that psychiatrists take as symptoms and signs of schizophrenia more socially intelligible than has come to be supposed?" (p.12). The term "intelligible" replaces "comprehensible" as in *The Divided Self*—and is crucial. It is a term borrowed from Sartre meaning the making sense of that which is between "what is going on (process) and who is doing what (praxis)" (p.22). We will come back to whether or not this question was answered or indeed whether it was a useful question to ask in the first place. Pausing to examine the preface itself, reveals that it is its stridency and vituperation which incensed reviewers and perhaps discouraged them from reading further. The late Eliot Slater (1971) reviewed the 2nd edition for this *Journal* (the first was not submitted) and stated that "to formulate this question involves a hypothesis regarding schizophrenia" despite the authors disclaiming any intention to propose such a hypothesis, indeed the word schizophrenia is isolated in parentheses hoping perhaps it would wither and die. Slater's answer to the authors' question was an emphatic "no" and he went on to describe the whole enterprise as "irrelevant" and "disingenuous". Disingenuous it may have looked, being bereft of reliable measures, controls and unbiased sampling, but irrelevant it certainly

was not. In a review of *The Self and Others* (Laing, 1969) Professor J. K. Wing (1971), though describing Laing's observations on families of schizophrenics as "subjective and partisan" goes on to confirm his importance to psychiatry if only as a curious social phenomenon.

R. D. Laing and Aaron Esterson, a contemporary from Glasgow University, set about elucidating the context of behaviours and experiences in order to make schizophrenia intelligible, comprehensible, meaningful and understandable. Following on from earlier work, it challenges the notion that the quintessence of schizophrenia is its un-understandability (Jaspers, 1963). This meaningfulness is seen as residing in the family "nexus" with the implication that "the schizophrenia" somehow exists only within the shared experience of a family "system", a concept derived from cybernetics and sociology and brought to bear upon families by the influential Bateson group (Bateson *et al.*, 1956). Laing and Esterson did this by exhaustive interviews, often totalling 50 hours, with 11 female schizophrenic patients both when well and psychotic, alone and with family members in all combinations. So rich in detail were their observations that one family case history, the Danzigs, was expanded into a book (Esterson, 1970). This appreciation for the subtle nuances of communication, verbal and non-verbal, and the realization that the interaction between a patient and his or her family may be infinitely more revealing than individual interviews was ahead of its time. Such concern for detail and recording belies the charge that Laing and co-workers were less than serious students of interpersonal relationships. The question of whether this effort answered their hypothesis is largely in the eye of the beholder. The data presented is of necessity highly selective. For example, does the repeated invalidation of patient Maya Abbott's feelings/beliefs/desires by her parents such as "you don't really think that," explain her ideas that her thoughts are being taken away or controlled? Does the fact that a mother listens to her daughter's telephone calls render intelligible her delusion that people listen to her telephone calls? Is Clare Church's impoverished affect explicable in the light of her parents' inconsistent and contradictory attitudes to

Clare's affection for others? For most psychiatrists, the provision of context is insufficient to explain the form that a patient's experiences take, which is recognisable as pathological. Indeed the authors acknowledge that to understand fully Lucie Blairs' "phenomenology" they have to invoke "internalization and re-projection" (p.74). The meaning which Laing and Esterson ascribe to "content" is ingenious and in itself a valuable contribution since many of the phenomena they examine are undoubtedly schizophrenic, frequently Schneiderian symptoms. If we allow a qualified "yes" to their question it still leaves many of ours, perhaps more important, untouched. Contemporary researchers were left to discover whether families of schizophrenics are more disturbed than families of other psychiatric patients (see Hirsch & Leff, 1975, Rund, 1986). Does this reflect a shared genetic constitution (see Wender, 1977) or the non-specific effects on parents of having a disturbed child (see Wynne *et al*, 1976)?

The anti-intuitive finding that schizophrenics discharged from hospital fared worse when they returned to their parents (Brown *et al*, 1962) led to a substantial body of influential work from the Institute of Psychiatry reviewed recently by Hooley (1985). The techniques used for inferring family interaction from interviews with one or other parent were crude in comparison to Laing and Esterson's. The highly sensitive dissection of intra-familial communication cannot easily be rated reliably so cannot be taken into account when measuring expressed emotion (EE). This work has been extended by psychologist, Michael Goldstein and his team who have used EE and other measures called communication deviance and affective style to examine interaction directly and in more detail. If it were possible to rate Laing and Esterson's recordings and transcripts they would almost certainly be found to be heavy with all these parameters. It seems that the authors were grappling with the same abnormalities that have provided others with a venerable area of research. Their mistake was to claim more for their studies than the methodology allowed. For instance, their statement that all families of schizophrenics are seriously disturbed is clearly an overstatement which can be explained by the fact that their subjects were chronic and frequently relapsing and so were likely to come from high EE families. By sticking to the issue of relapse, workers at the Institute of Psychiatry have avoided such accusations. The Goldstein group have been more ambitious and have tried to address aetiological issues in a family study follow-up of disturbed adolescents (Goldstein, 1985). Twenty years after Laing, and with a host of collaborators, the same questions remain substantially unanswered.

One area in which *Sanity, Madness and the Family* has been particularly influential is family therapy in that the book linked schizophrenia with family pathology in a way that was vivid and convincing, especially to those practitioners without medical training. This was probably unintentional as the authors object to the notion of "family pathology" as strongly as they do to neuropathology arguing that this "biological analogy" has no place in a "social phenomenological" perspective. This major theoretical difference does little to disguise the similarity between the processes they describe and those described by family therapists such as enmeshment, disqualification, collusion and so on. Family therapy is an anathema to Laing and his colleagues as is physical treatment. David Cooper (1967), quipped that "curing" is something one does to bacon and not people called schizophrenic. For Laing and Cooper, the only "treatment" was to get the patient out of the home and in to an environment free from coercion. This was based presumably on the assumption that patterns of interaction cannot be changed, a view certainly not shared by family therapists (e.g. Minuchin, 1974; Falloon *et al*, 1985). Simply reducing face to face contact may be protective (Leff *et al*, 1982). The experimental ward, Villa 21, at Shenley Hospital in which Cooper managed schizophrenic patients may have served this purpose among others. Patients could come voluntarily and refuse treatment if they wished. They had a close one-to-one relationship with a staff member from a multi-disciplinary team. The ward was run democratically with patients holding communal responsibility. The doctor relinquished his white coat. This "daring" set-up does not sound the least revolutionary now. Indeed the District Services Centre at the Maudsley Hospital and many other places like it, provides continuing care for chronic psychiatric patients along very similar lines. The efficacy of this sort of programme was assessed in comparison to the standard treatment of the time and published in a little known paper in the *British Medical Journal* (Esterson *et al*, 1965). The authors could be said to be advocates of asylum in its broadest sense (as in the Philadelphia Association) and not community psychiatry or, for that matter, anti-psychiatry which seeks to destroy all such institutions and which Laing decries (McGinley & David, 1985).

The most trenchant accusation made of Laing and *Sanity, Madness and the Family*, is the crime of parent blaming. It is difficult to find an explicit example of this in any of Laing's writings. Nevertheless, whether it was his intention or not, his tone and posture at times leads the reader inevitably to this conclusion. There can be no victim without a

victimizer. The review from *New Society* quoted on the cover of the Penguin edition talks unabashed of the "ugly sight of children being brainwashed by parents". Families too have inferred an attack as shown by many heart-felt essays collected by the National Schizophrenia Fellowship (1974). It is not enough, *not* to blame them; families must be supported and actively encouraged (Berkowitz *et al*, 1981). In the terminology of family therapy, their attitudes and actions must be "positively connoted" if any change is sought and a therapeutic alliance is to be maintained.

In conclusion, *Sanity, Madness and the Family* may be judged more favourably in hindsight than it was when first published. In an effort to give method to extreme forms of madness, it has helped to deflect the psychiatrist's microscope towards examining social interaction in a naturalistic way rather than in

the sterile confines of the laboratory. It reminded us that "Those psychiatrists who are not prepared to get to know what goes on outside their clinics and hospitals simply do not know what goes on" (p.13). It has encouraged family approaches even in psychotic disorders and the growth of family therapy, unwittingly perhaps. Unfortunately, it probably alienated some families in the process. By virtue of its appeal to public and hence patient opinion, and influence on non-medical and some medical staff, it has contributed to an atmosphere whereby less formal, more eclectic care for severely disturbed psychiatric patients could flourish.

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Expressed Emotion and Relapse in Schizophrenia

With reference to the correspondence under this heading (*Journal*, June 1986, 148, 741-742) neither Dr Leff nor Dr Vaughn was involved in any stage of the assessment and editing of the paper between receipt of the manuscript and publication.

Editor

Deficits of Chronic Schizophrenia in Relation to Long-term Hospitalisation

Sir: Mathai & Gobinath's study (*Journal*, May 1986, 148, 509-516) casts further doubt on the role of 'institutionalisation' in determining the course of chronic schizophrenia. We have argued (Abrahamson & Brenner, 1978; Abrahamson, 1983) that the positive correlation between length of stay and negative handicaps characteristic of previous studies was a reflection of the inadequacy of a cross-sectional approach. A cohort of patients with negative handicaps which persisted largely unchanged through hospital courses of 30 years or more was mainly responsible for the positive correlations we obtained retrospectively, which were strikingly similar in pattern to those obtained by Wing & Brown (1975).

It seems possible that Mathai & Gobinath have avoided this particular pit-fall because few such patients now exist in their long-stay populations or, more probably, because they were screened out by the diagnostic and exclusion criteria. It is notable that none of the patients were aged over 55 years and that only 8.8% of the NIMHANS and 18% of the MH long-stay populations were included. Whilst avoiding one source of bias others may have been introduced by the exclusion process, or submerged in the cross-sectional analyses. We found it revealing to relate the scores on disability scales to length of stay year-by-year rather than to longer time spans. In addition, despite their limitations, case-note studies provided an invaluable longitudinal dimension, the importance of which is underlined by prospective studies of long-term courses (Bleuler, 1978; Harding & Strauss, 1985). The numbers in Mathai & Gobinath's groups are sufficiently small for detailed

case-note studies to be feasible for all the patients included and samples of those excluded.

The main impression from the Goodmayes study, which has been reinforced by continuing experience with some of the patients concerned, is of the individual consistency of courses. A longitudinal framework is therefore essential to do justice to social influences, which appear to operate within narrow, perhaps biologically determined, constraints over a very long time course. A much more complex explanation than either a disease process or a social model alone provides is likely to be required for the re-evaluation of 'institutionalisation'.

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The Northwick Park Study of First Episodes of Schizophrenia

Sir: Hidden away in the four papers on first episodes of schizophrenia from Northwick Park (*Journal*, February 1986, 148, 115-143) is information of interest to psychogeriatricians. First, the inclusion of patients "aged between 15 and 70 years, with a first psychotic illness, not unequivocally affective", would suggest that British psychiatrists accept that schizophrenia can develop for the first time in patients over the age of 40 or 45. Second, Table I (page 116) shows that three patients were excluded as being outside the age range. Were any of these over the age of 70? Third, Table I (page 129) shows that 37 of 253 (14.6%) patients were aged 40 or over on admission. The Table shows the expected excess of males over females for all ages, but does this relationship hold for the older age groups? Fourth, Table I

(page 129) also shows that there was one patient aged 60–65 who was trial eligible, but not a trial entrant. Table I, (pages 122–123) shows that randomisation was applied to patients only between ages 15 and 59. Why? Does this mean the researchers were reluctant to diagnose schizophrenia in a patient aged 60 or more?

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Drs Johnstone, Macmillan and Crow Reply

Sir: We limited our age range to 15–70 years largely for practical reasons. Our collaborators like ourselves generally only see patients within that range and furthermore a study which involved a 2-year follow up while the patients continued on drug regimes with stated minimum doses would have been associated with additional difficulties in the very young and the elderly. The three patients excluded from the trial on the grounds of age consisted of a 14 year old male, a 71 year old female, and a 73 year old female. The 37 patients aged 40 or over on admission consisted of 22 females and 15 males. The trial eligible patient aged between 60 and 65 who was not a trial entrant was a 65 year old lady who did not wish to participate in the trial. She would have been very welcome to do so but her refusal meant that there was no-one over the age of 60 to be included in the randomisation process.

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Alcohol Related Problems in Ethnic Minorities

Sir: We read with interest Dr King's paper on at-risk drinking among general practice attenders (*Journal*, May 1986, 148, 533–540). We were interested to find that there were significantly more Irish and Scots among the at-risk drinkers but were surprised to note that no Asians seemed to fall into the at-risk group. This was in spite of the fact that 28% of the screened population had a country of birth outside the United Kingdom. We have recently looked at the differences in morbidity patterns between Asians and non-Asians with a diagnosis of alcoholic liver disease at the Royal Free Hospital (Banerjee *et al*, 1986). Our results showed that of the 852 biopsies performed showing alcoholic liver disease between January 1978 and November 1984, 58 (6.8%) were from Asian patients. This is a higher percentage than one

would expect when corrected for the percentage of Asians in the population.

Previous studies have suggested that there are differences in alcohol sensitivity between different ethnic groups (Chan, 1986; Ewing *et al*, 1974). In addition, it has also been shown that drinking patterns vary between different ethnic groups (Caetano, 1984). We suggest, therefore, that Asians are a high-risk group for alcohol related problems (Balaharan *et al*, 1984) and the apparent low incidence of alcohol related problems in surveys may be due to socio-cultural taboos which result in under-referral to the patient's local general practitioner.

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Hypomania Following Cognitive Therapy

Sir: We enjoyed reading the letter from Drs Hughes & McKane (*Journal*, March 1986, 148, 344). They suggest that the patient we describe is typical of a bipolar affective disorder developing in middle life. However, continued assessments of this patient for two years after involvement in the research study have shown no further episodes of affective disturbance. This might be expected in a patient with an initial diagnosis of dysthymia, which in DSM-III terms is a low grade depressive disturbance that is rarely associated with bipolar affective disorder. We were also excited by their hypothesis that the filling in of numerous questionnaires may induce mania. An examination of the data from our study reveals 120 patient-years of questionnaire administration but no other case of mania has been found. Reluctantly, therefore, we had to abandon this hypothesis. We are left, therefore, with an isolated case of mania manifested towards the end of a programme of cognitive therapy. Although no case report proves a hypothesis, it is reasonable to conclude that the

temporal relationship between treatment and the onset of manic symptoms was not entirely coincidental and therefore worthy of report.

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Radioreceptor Assay of Serum Neuroleptic Concentrations in Psychiatric Patients

Sir: We read with interest the report by Kraska *et al*, (*Journal*, February 1986, **148**, 187-193), concerning the usefulness of radioreceptor assays for the measurement of plasma neuroleptic concentrations. We have been assessing the usefulness of a similar assay based on a lyophilised calf caudate preparation and ³H-labelled spiperone ligand, which is available from Wellcome Diagnostics as a 200 assay kit (Lader, 1980). We have found this assay simple to use and reproducible, requiring only 0.2 ml of plasma for each duplicate analysis. The assay has been found to be linear between 15 and 1000 neuroleptic units per litre (1 NU/l equivalent to 1 nmol/l haloperidol).

In contrast to Kraska *et al*, who investigated patients on long-term therapy, we are investigating the application of this assay to the management of acute schizophrenia and have so far studied nine patients. All our patients were previously untreated, fitted the RDC criteria for schizophrenia (Spitzer *et al*, 1975), and were treated with haloperidol in doses of between 1.5 and 60 mg per day according to clinical judgement. No other neuroleptic or psychotropic medication was prescribed. We found a significant linear relationship between daily dose of haloperidol and plasma dopamine receptor binding activity ($n=11$, $r=0.76$) similar to that reported by Kraska *et al*. In three patients who were intensively investigated over a 4-6 week period there was a marked clinical improvement, as assessed on the CPRS rating scale (Åsberg *et al*, 1978). We found a direct relationship between dopamine receptor binding activity, dose and clinical improvement. However, due to the small number of patients, statistical significance could not be reached. This improvement was obtained on doses of between 9 and 20 mg/day haloperidol, which achieved plasma neuroleptic concentrations of 14-48 NU/l.

Extrapyramidal side-effects, as assessed using the Simpson Rating Scale (Simpson & Angus, 1970), were completely unrelated either to dose or to plasma neuroleptic concentrations. This poor relationship between plasma neuroleptic activity and extra-pyramidal side-effects was confirmed in six additional patients. These findings underline the conclusion reached by Kraska *et al*, that for chronic schizophrenics there was no simple relationship between plasma neuroleptic concentrations and side-effects. It is interesting that side-effects seem to be so poorly related to

total plasma neuroleptic dopamine blocking "activity" as measured in a radioreceptor assay. This may be because the assay measures only the total plasma concentration of "active" drug *in vitro* rather than reflecting dopamine blocking activity in brain *in vivo*. Another major problem with the use of this technique is that dopamine receptor binding activity may differ from one neuroleptic to another by several orders of magnitude despite equivalent clinical effects. The results are therefore meaningless if the patient is on more than one neuroleptic drug at the same time, a situation which pertains frequently in clinical practice.

Although a number of early reports indicated that radioreceptor assays showed promise, more recent work has been equivocal (Dahl, 1986). It is likely that such assays offer very little advantage over alternative techniques, e.g., gas and liquid chromatography which are capable of measuring parent drugs as well as metabolites which may have activities on different neurotransmitter systems. Much more work is required before dopamine blocking radioreceptor assays can offer any useful information in the management of schizophrenic patients.

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Neuroleptic Malignant Syndrome

Sir: In their recent review of the neuroleptic malignant syndrome (NMS) Drs Abbott & Loizon (*Journal*, January 1986, **148**, 47-51) recommended sodium dantrolene and bromocriptine as the best treatment options for this syndrome. We wish to suggest the possible use of electroconvulsive treatment (ECT) in NMS in addition to these treatment modalities.

Case Report: We recently treated a patient who presented a NMS which improved with ECT. This 23 year old male schizophrenic patient developed NMS on the fourth day

of his treatment with intramuscular chlorpromazine (200 mg/day). The clinical features and laboratory results were typical for NMS, including extreme muscle rigidity, severe dysautonomic disturbances, and laboratory findings of rhabdomyolysis. In spite of the discontinuation of chlorpromazine the patient's condition deteriorated over the following week. On the seventh day of NMS a bilateral modified ECT was administered which was followed by a dramatic improvement in hyperpyrexia, diaphoresis and level of consciousness. The patient received a course of five ECT treatments within 2 days. When ECT was stopped fever and stupor recurred. Four further sessions of ECT were administered resulting in a complete recovery.

A survey of the literature revealed four case reports of successful treatment of NMS by ECT, (Powers *et al*, 1976; Jesse & Andersen, 1983; Lazarus, 1986). In another patient (Regestein *et al*, 1977) ECT during NMS was followed by permanent brain damage. This patient, however, had suffered from cardiac arrhythmia before ECT, and developed ventricular fibrillation followed by brain anoxia during the ECT. The disastrous outcome did not result, therefore, from a direct action of ECT on the brain. Besides specific contra-indications for ECT, this procedure seems an effective treatment for NMS. The ability of ECT to increase post-synaptic receptors' sensitivity for dopamine (Lerer & Balmaker, 1982) might explain its beneficial effect in NMS. This explanatory model is in line with the aetiological role attributed to an acute reduction in CNS dopamine activity in NMS.

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Sir: We were interested to read the review on the neuroleptic malignant syndrome by Abbott and Loizou (*Journal*, January 1986, **148**, 47-51). Whilst the quotation from Baastup and colleagues (1976)

accurately reflects their finding that none of the 425 patients in their review treated with both lithium carbonate and haloperidol developed the neuroleptic malignant syndrome, we were intrigued to note that the authors had omitted to mention the original report by Cohen & Cohen (1974) which had prompted Baastup *et al*, to conduct their review. Cohen & Cohen described four acutely agitated manic patients receiving the lithium and haloperidol combination who developed a toxic syndrome which they attributed to a specific haloperidol/lithium interaction.

However, Jefferson & Greist (1980) reviewed the original case reports and noted that the clinical descriptions resembled the neuroleptic malignant syndrome. This similarity has also been noted in other publications (e.g. Frankel & Spring, 1982). Since Cohen & Cohen's original paper, a number of other case reports have appeared in the literature, and other neuroleptics have been implicated in presumed toxic interactions with lithium. Having closely examined 26 similar reports of patients treated with haloperidol and lithium (Hone *et al*, 1985) we concluded that at least seven of the 26 met the criteria for the diagnosis of neuroleptic malignant syndrome, including the four patients originally reported by Cohen & Cohen.

In summary, we can find little or no support for the lithium/haloperidol interaction hypothesis. We consider that there is a need for a detailed epidemiological enquiry to identify the incidence of neurotoxic events resulting from the concomitant use of lithium and neuroleptics, together with other toxic sequelae related to neuroleptic usage. In conducting such a survey, we would hope to provide more information on these phenomena - be they attributable to lithium toxicity, drug interactions, or the neuroleptic malignant syndrome.

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Anticholinergic Anti-Parkinson Medication for Neuroleptic-Induced Extrapyramidal Side Effects

Sir: We have recently received a piece of promotional literature entitled *Disipal in the Neuroleptic Syndrome*. The content of this document conveys a general impression that orphenadrine is a safe drug to use for the treatment of drug-induced extrapyramidal symptoms both in acute situations and for long term maintenance. We find the document biased and against all current scientific opinion on the subject and would like to counter some of the arguments presented in it.

Extrapyramidal side-effects frequently accompany neuroleptic treatment. In fact, anticholinergic agents used to reduce these symptoms are the commonest form of concurrent medication with neuroleptic therapy. There is, however, much debate about the adverse effects of combined therapy with these agents. It has been claimed that their use can result in exacerbation of psychosis, a delay in symptom improvement in patients with acute schizophrenia, and a predisposition to the development and/or deterioration of tardive dyskinesia. Many studies have reported that co-administration of anticholinergic drugs resulted in lower blood levels of neuroleptics. In others, although no differences were observed, combined therapy did have clinical implications (Bamrah *et al*, 1986). Clinically stable patients on neuroleptics do not normally show accompanying extrapyramidal symptoms except when dosages are increased or if extraneous factors lead to unpredictable increases in blood levels. As clinicians, therefore, we do not see the need for long-term medication especially if this is going to be associated with other interactions.

The document alleges that orphenadrine withdrawal may precipitate "a pronounced depressive state" in patients on combined haloperidol-orphenadrine therapy (Altamura *et al*, 1983). Whereas the relationship of depression with haloperidol is controversial, it is not unreasonable to assume that stopping a "mood elevating" drug could cause a rebound precipitation of dysphoric mood. One does not have to invoke "pharmacological interaction between orphenadrine and haloperidol leading to increased bioavailability of the latter" to explain this phenomenon. It seems to us irresponsible to elevate their "mood lightening" properties to

a position of clinical relevance since these are the very properties which may give them their drug-dependence potential.

These drugs have a powerful anticholinergic action which can summate with the anticholinergic action of neuroleptics to produce severe constipation or ileus, urinary hesitancy or retention, and intolerable dry mouth and blurring of vision (Lader, 1980). Besides, anticholinergics are themselves known to produce toxic psychoses (Shader & Greenblatt, 1971). Therefore, their use should be tempered with caution. Finally, if prescribed with abandon they would only provide the depressed patient with another means of self extermination.

We accept that the anticholinergic anti-Parkinsonian agents are valuable in clinical settings where it is necessary to relieve acute extrapyramidal symptoms following neuroleptic therapy, but we do not feel that the majority of patients require this treatment for more than three to four months. A literature review suggests that only a third of patients on maintenance treatment have any real need for this additional medication to control extrapyramidal symptoms, with the majority of surveys suggesting a substantially smaller proportion (Johnson, 1985).

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Lithium Induced Hypothyroidism Presenting with Carpal Tunnel Syndrome

Sir: We describe a patient presenting with a recognised but unusual symptom of hypothyroidism which to our knowledge has not previously been reported in association with lithium.

Case Report: The patient was a 43 year old Kenyan Asian woman with a long-standing history of manic-depressive psychosis who had been treated with lithium for 10 years

At the out-patient clinic she gave a three month history of paraesthesiae in the hands, the right worse than the left, which woke her at night and were relieved by hanging her arms over the edge of the bed. There were no other symptoms of thyroid disorder. On physical examination pinprick and light touch were reduced over the median nerve distribution. There was a slight delay in ankle jerk relaxation and a smooth thyroid was easily palpable. Laboratory tests of thyroid function (which had been normal eighteen months previously) revealed total T4-12 nmol/L (NR 50-150), free T4-1 pmol/L (NR 10-28), TSH < 50 mU/l (NR 0-7 mU/l). Thyroid antibody screen was negative. Thyroxine replacement was instituted (100 ug daily) and after two to three months of treatment the symptoms of carpal tunnel syndrome resolved completely. Lithium was continued throughout.

Carpal tunnel syndrome may result from lithium therapy because of fluid retention caused by the drug. In this woman's case, however, the primary

cause must have been hypothyroidism because the symptoms resolved with thyroxine while lithium was continued. Several points are raised by this case. First, lithium induced hypothyroidism may present in unusual ways without typical symptoms such as lethargy, weight gain, voice change and cold intolerance. Secondly, hypothyroidism may present late—in this instance after 10 years therapy during which thyroid function had been normal on several occasions. Thirdly, the need for regular monitoring of thyroid function in patients taking lithium is clear (although a symptomatic presentation may still occur).

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A HUNDRED YEARS AGO

Asylum Reports

"If such is the case, and we are bound to believe the statement, we do not hesitate to say that Holloway's is a great Charity wasted, for Mr Holloway's original intention or plan was to provide for private patients just above the pauper class—*none* over 21s. a week. *Now* the class he intended to benefit is expressly excluded. Endless structural alterations are or have been required, and a great charity is nullified. 'But yet

the pity of it, Iago!' What unspeakable good *might* have been done with that money on the lines of the first intention! We are almost tempted to say that the taste of the pill though silvered is scarcely disguised, and that there are not a few 'dead flies which cause the ointment of the worthy donor to send forth a stinking savour'. We do not refer to the imperfect drainage only."

Reference

Journal of Mental Science (1887) 33, 319.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.

Book Reviews

The Oxford Companion to Medicine. Volume I. A-M. Volume II. N-Z. Edited by JOHN WALTON, PAUL B. BEESON and RONALD BODLEY SCOTT. Oxford: Oxford University Press. 1986. Pp 1534. £55.00.

That the egregious *Oxford Companion* series should be extended to medicine now seems inevitable and right, once it has been done, but this is in fact a completely new production, hard on the heels of that for literature. It is, by any standard, a formidable work—over 6000 entries, 1500 pages, 150 contributors, a £55 price, and two volumes: the Anglo-American 'special relationship' is seen in its full coverage of US institutions and practices and in the inclusion of American trade-names for drugs. Its purpose is described as to be "a comprehensive reference book, concerned with the theory, practice, and profession of medicine", and amongst the 150 major entries are psychiatry, covered by Michael Gelder, and psychology by the late H. Gwynne Jones; both are models of condensed information, elegantly expressed. Impeccable editing has ensured a remarkably uniform quality of excellence throughout the work of many disparate hands.

Since this is meant to be readable by the intelligent layman, detail on any topic is inevitably limited, but with few exceptions, practically any significant subject or person that one could think of will be found listed. Opinions will obviously differ about the amount of space appropriate for any one of them—hypnosis, for instance, might have deserved a much larger entry—perhaps at the expense of some parochial detail on institutions—and Sir Aubrey Lewis an item of his own. One of the most delightful sections is by A. M. Cooke on 'Doctors in Other Walks of Life (Medical Truants)' dealing with medical men as scientists, men of letters, churchmen, bibliophiles, linguists, missionaries, lawyers, philosophers, politicians, practitioners of the arts, and even criminals—including Dr Crippen, of course. However, Dr Mudd—who treated John Wilkes Booth after his assassination of Abraham Lincoln—seems to be missing. Some minor mistakes are inevitable in a work of this size, particularly in its first edition, and the entry on the Board of Control, for instance, records it as being still in existence, while the Mental Health Act Commission doesn't rate a separate item. This is clearly going to be an essential work of reference, and it is to be hoped that most libraries will be

able to afford it, even if few individuals can. It is a remarkable achievement.

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After Hospital: A Study of Long-Term Psychiatric Patients in York. By KATHLEEN JONES. York: Department of Social Policy and Social Work, University of York. 1985. Pp 77. £3.50.

This booklet reports a survey of 150 patients discharged from mental hospitals in York over a two-year period. 50 were long-stay patients, predominantly schizophrenic, and 100 were elderly confused patients. They had been discharged to a variety of placements. The editor and her colleagues attempted to follow-up these patients, interviewing the patient or those involved in their care. They sought to answer four questions; what services the patients were receiving, how the necessary collaboration between agencies was working out in practice, to what extent discharge plans had in fact been implemented and whether the quality of patient's lives was adequate in their new situations. This information was obtained through freeflowing interviews given structure by checklists. It is apparent from the design of the study, which did not involve before-and-after comparisons or control groups, that no rigorous answers could be given to their questions. Nevertheless there is obvious value in the 'helicopter' view which the researchers intended to provide—the report certainly gives a very clear idea of the sorts of problems that arise from discharge and from a commitment to a policy of increasingly locating care in the community. The picture is augmented by the provision of case vignettes.

The success of discharging these patients was variable. Considerable care appears to have gone into planning the placement of the long-term patients, and some of these did very well. Less planning attended the discharge of the elderly confused and their success was likewise meagre. Individually, patients often showed both gains and losses as a result of their move. Collaboration between agencies about aftercare provision was found to be far from perfect.

In their summary, the authors emphasise that community care does not mean that a majority of

patients return to their homes. In any case, discharging patients to their relatives was sometimes neither humane nor cost effective. They point out that collaboration, often imperfect and often prescribed as a panacea for healing the defects of community care, is not cost free, and recommend the provision of permanent staff for Joint Care Planning Teams, and the establishment of a post of case-manager. More day hospital places and special provisions for the disruptive elderly patient are needed.

This report, while not over-enthusiastic, suggests that community care remains a feasible and humane possibility, but that if it is to be realised even in the benignant community of York, bolstered by its tradition of service and philanthropy, additional resources and deliberate management are required.

This book is of interest to all involved in managing chronic psychiatric patients in the community in the planning of services.

PAUL BEBBINGTON, *Honorary Senior Lecturer, MRC Social Psychiatry Unit, Institute of Psychiatry, London*

Mental Health Planning for One Billion People.

Edited by TSUNG-YI LIN and LEON EISENBERG.
London: University of British Columbia Press.
1985. Pp 232. £17.50.

There is currently great curiosity about all things Chinese. How do one quarter of the Earth's population live, in a country about the size of the U.S.A., separated from us by great distance, strange language, a culture and history which barely touched ours until the 19th century, and a political system which has rarely allowed free travel and reporting? There can be no doubt at all that under Chairman Mao and his successors medical care and the public health have taken an immense leap forward since 1949, but the details of what has happened, how precisely things stand now, and what further developments are practicable remain virtually unknowable, at least to Westerners.

There are so many difficulties. Some Marxist writers naively believe that alcoholism and mental disorders are due to oppressive capitalism and simply do not exist anymore in a socialist country. People fear loss of face, or criticism, or lack of patriotism if they admit the occurrence of suicide, for example. Diagnostic categories such as neurasthenia or depression may be used differently, not defined and without clear consensus. China does not publish any reliable national statistics of the incidence of mental illnesses or psychiatric behaviours, or of the distribution of hospitals or professional workers, and one

recalls there are about 20 provinces of ethnic and geographical diversity and some autonomy in health matters.

There is no unified standard health service. In the present book Professor Liu Shie-Jie writes (p. 89) "Two parallel health care systems exist and function side by side... For example, the Capital Steel and Iron company has its own health department, which administers its own hospital and health station... and in addition has clinics in various small factories... under the supervision of the Ministry of Metallurgical Industry, not the Ministry of Health." Under the latter's umbrella there are independent special services for tuberculosis, drug control, maternity etc., and also provision of hospitals and/or clinics by provinces, cities, counties, districts, communes and brigades, administered locally with local finance. It cannot with such diversity be easy to collect information to give a clear factual picture of the working of mental health services in one city, let alone one province. There are beginning to be a few very careful small scale studies such as that reported in this book by Xiang Mengze on a survey of neurosis among general hospital patients in one county hospital in Sichuan (population 100 million).

This book consists largely of papers presented at two 1983 Beijing conferences on Psychosocial Aspects of Primary Mental Health Care, and a third of it deals with children. Its title is misleading. Planning a mental health service involves knowing what illnesses are frequent, what customers want, and what health workers, training, buildings, money are available, or could be, to meet the challenges. This book is not about such matters. Inevitably it contains much more about what might be than what is. But it does introduce us in an elementary way to 36 years of history and to mental health work in one or two places. Where there is so little information this is a beginning to be welcomed.

JOHN CRAMMER, *Reader Emeritus in Biological Psychiatry, Institute of Psychiatry, London*

The Anatomy of Madness. Essays in the History of Psychiatry. Vol I. People and Ideas. Pp 316. Vol II. Institutions and Society. Pp 283. Edited by W. F. BYNUM, ROY PORTER and MICHAEL SHEPHERD. London: Tavistock Publications. 1985. £19.95 each.

These two volumes of essays on the history of psychiatry are for me the best literary lucky-dip to come my way for many a moon. As in all lucky-dips, however, the prizes vary in value; but there is not one

here which is not well worth having. It says a lot, incidentally, for the catholicity and fair-mindedness of the editors (and of the organisers of the seminars during which most of the essays were read), that they allowed, not only such a diversity of views to be expressed, but also allowed one contributor to do battle with another.

As an excellent example of the former I would cite Anthony Clare's, 'Freud's Cases: the Clinical Basis of Psychoanalysis', a courageous exposé of the lack of scientific evidence to support any claim for therapeutic benefit in psychoanalysis, a conclusion which must have raised the hackles of his psychoanalytically orientated co-contributors.

And as an example of the latter, I would commend 'Bedlam: Fact or Fantasy?', by the redoubtable Patricia Alderidge, Archivist to the Bethlem Royal Hospital, in which she fights like a tigress in defence of her hospital. She attacks those historians, and in particular Andrew Scull, for perpetuating the myths of brutality, corruption and exploitation which have tarnished Bethlem's good name for centuries.

But if Andrew Scull is floored in this particular encounter with Patricia Alderidge, he lives to fight another day. Indeed, his contribution, 'A Victorian Alienist: John Conolly', is one of the major prizes on offer. His, as far as I am aware, is the only study in any depth of this folk-hero of British psychiatry, and it is saddening, in a way, to learn that, great as he undoubtedly was, Conolly like any other man, had feet of clay.

For idolaters in psychiatry, it is equally disconcerting, to read in Anne Digby's, 'Moral Treatment at The Retreat, 1796-1846', that patient care at The Retreat wasn't always all it has been cracked up to be. She ends her essay on an enigmatic note, "This poses the fascinating but unresolvable question of whether the distinctively religious character of The Retreat's moral treatment did not rely for its efficacy, at least in part, on an unseen kernel of spiritual healing".

It would be invidious to attempt to place the other essays in any sort of rank order, but one of the top prizes must go to Ian Dowbiggin's, 'Degeneration and Hereditarianism in French Mental Medicine 1840-1890: Psychiatric Theory as Ideological Adaptation'. This is the period in French psychiatry dominated by Doctors F. J. Moreau and B. A. Morel, and a period too, when the reputation of psychiatry and psychiatrists had hit rock-bottom. Dowbiggin's well argued thesis is that "It is undeniable that this scientific milieu after the mid-century made the theory of morbid heredity all the more acceptable as a way of improving psychiatry's desperately poor image and neutralising public enmity,

while it helped to heal the divisions within the professional community". Nevertheless, it should be noted that, despite the ethical dubiety of its origins, there can be no doubt that the theory of morbid heredity profoundly influenced the thinking of contemporary British psychiatrists, and in particular, that of Henry Maudsley.

Martin Stone's, 'Shellshock and the Psychologists', is also prize worthy and earns a bonus for reminding us of the finest examples of muscular jingoism ever pronounced. This pearl is attributed to Sir Robert Armstrong-Jones, quondam president of the Medico-Psychological Association and a bitter opponent of what he termed, "Freudism". At a meeting of the Society in 1920 he declared that psychoanalysis was, "probably applicable to people on the Austrian and German frontiers, but not to virile, sport-loving, open-air people like the British". Beat that, you cad!

HENRY R. ROLLIN, *Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey*

An Introduction to the Borderline Conditions. By WILLIAM GOLDSTEIN. Northvale, New Jersey: Jason Aronson. 1985. Pp 241. \$25.00.

The concept of "borderline" psychic functioning has been employed in psychoanalysis for half a century. Now after increasing usage recently in general psychiatry, the term has obtained formal recognition through the inclusion in DSM-III of the category "borderline personality disorder". Unfortunately, although the psychoanalytic and descriptive uses of the term apply to similar entities, there are troublesome difficulties in emphasis. Nosologically the term has been used to describe a cluster of symptoms and traits which define a diagnostic category, *the borderline personality*. Psychoanalytically the term refers to a hypothetical psychic configuration and related pattern of mental functioning, and is used diagnostically only as a supraordinate concept to differentiate *the borderline conditions* from neurosis and psychosis. This situation is a recipe for misunderstanding and conflict, dynamics which are all too prevalent in work with this difficult group of patients.

Any book which provides "an in-depth yet simplified understanding of the different uses of the term today" would be welcome indeed. Unfortunately Dr. Goldstein does not succeed wholly in his stated aim. He offers breadth at the expense of depth and simplicity.

A psychiatrist and psychoanalyst Dr Goldstein approaches the subject from the standpoint of

ego-psychology, a useful framework both for describing the characteristic structural configuration which underlies the typical symptom cluster, and for establishing a *psychodynamic* diagnosis of borderline personality organisation. He details the history and current psychoanalytic application of the concept, and outlines both the discriminant studies which defined the "borderline personality disorder" and its clinical features. There is a useful chapter describing the influential work of Kernberg, though this overview is repetitive and lacks depth. For some reason a comparable overview of Kohut's work is assigned to the section on differential diagnosis, where an exploration of the relationship between borderline and narcissistic disorders serves to confuse rather than simplify this complex field.

The section on treatment strategies includes some illustrative case studies and a useful brief overview of psychotherapeutic approaches; but it concludes with an unbalanced review of the modifications to psychoanalytic technique which have been applied in the treatment of borderline patients. This chapter captures the dilemma of this book, the conflict between Dr Goldstein's attempts to simplify the subject for the general reader and his extensive coverage of the analytic literature on the psychopathology and treatment of the borderline conditions. There are some valuable nuggets to be found here for everyone, but there is much waste too. A book for the library perhaps.

MIKE HOBBS, *Consultant Psychotherapist,
Warneford Hospital, Oxford*

International Handbook of Behavior Modification and Therapy. Student edition. Edited by ALAN S. BELLACK, MICHEL HERSEN and ALAN E. KAZDIN. New York: Plenum. 1985. Pp 471. \$24.95.

This book is an abbreviated, but not an up-dated reprint of the 1982 *International Handbook of Behavior Modification and Therapy*. It comprises thirteen complete reprints of the more than twenty chapters in the original edition: One chapter now being devoted to the history of behaviour therapy, three to methodology, five to adult and four to childhood disorders.

In 1982, the widely acclaimed original edition was an impressive comprehension of the development of the whole field of behaviour-modification and -therapy. But, it did not have a special focus on clinically relevant research, and thus included extensive reviews of literature which was more historically important than clinically meaningful.

This 1985 reprint is certainly not the "representative curriculum for an introductory graduate seminar on behaviour therapy", that the editors would like it to be. The graduate student of behaviour therapy—regardless whether from a medical, psychological or nursing background—can best be motivated for active learning by vivid, treatment-relevant materials (of course, with a historical perspective). And here in particular are the shortcomings of both editions.

For the student as well as for the clinician, since 1982 several better suited books have appeared, additionally to the well-known annuals *Progress in Behavior Modification* and *Annual Review of Behavior Therapy*. They all cover the same or very similar topics to be found in this reprint. Among these, the *Handbook of Clinical Behavior Therapy with Adults*, with main emphasis on "what experienced clinicians actually do with their patients", also appeared in 1985, and it is also edited by Hersen and Bellack (two of the three editors of the 1985 reprint).

Covering the literature only until about 1980, the 1985 reprint misses such important new developments like: long-term follow-up studies and the "panic" controversy in treatment of phobias and other anxiety disorders; "medication versus behaviour therapy" research in depression and obsessive-compulsive neurosis; recent behavioural-psychological approaches to addictive behaviours and their relapses; new developments in couple and family interventions—including the systematic, *in-vivo* family treatments for schizophrenics, now available; benefits and risks from the increasingly popular self-help manuals for a wide variety of problems and disturbances; research into "failures in behaviour therapy"; and, last but not least, the general "strategy" of clinical behaviour therapy as the essential framework for the application of its techniques and procedures.

For the teacher in (clinical) behaviour therapy, this low priced reprint is by no means a substitute for the efforts and the rewards of a carefully selected and regularly updated reading list for his students. For the research worker, the experienced clinician and the librarian, a low priced sale of the original edition, at least to some extent, would have been a substitute for a revised complete edition.

IVER HAND, *Professor of Psychiatry,
Hamburg University Clinic*

Handbook of Intelligence: Theories, Measurements and Applications. Edited by BENJAMIN B. WOLMAN. Chichester: John Wiley. 1985. Pp 985. £66 45.

Handbook of Intelligence is a solid American piece of work in several senses. It is written in grammatically

correct academic style, and its thousand pages are very well edited. The inside cover describes it as being written by "leading international authorities", though only a single non-American chapter out of the 23, seems to have merited that accolade, and almost all references are to American work. Maybe they mean inter-state, as in the World Series baseball league. No other book, it claims, has "examined intelligence with the depth and breadth of this work", and for sure there is plenty of it, yet there is no reference to personality vis-à-vis intelligence in the limited subject index, and creativity is only considered as a minor aspect of giftedness. How can the publishers justify the price?

However, there are some gems within. Robert Sternberg's chapter explaining his thesis on the relationships between cognition and intelligence is scholarly, thought-provoking, and valuable; he describes how psychometric and cognitive theories complement and enhance one another, and proposes an ideographic approach to human abilities. Nathan Brody argues well from evidence that what most of us understand by intelligence is about right, and that the tests do measure it validly. But in direct contradiction, Michael Lewis and Margaret Wolan Sullivan, in their excellent survey of infant testing, stress that intelligence cannot be unitary, and see the IQ as a tool of social control.

The overview chapter on the neurological foundations of intelligence is useful, though the writer's assumption that 'g' with its subsidiaries describes intelligence is somewhat simplistic. But then, John Horn in his elegant piece on models of intelligence, finds the idea of 'g' "almost certainly incorrect and not very useful". Another contributor provides statistical evidence that intelligence is polygenic. Guilford and his cube of 120 potentially distinguishable abilities take a bow. His system, he says, is the first to detail the functioning of intelligence on a scientific foundation, and he suggests that teachers should plan their lessons round it.

The Max Planck team follow a developmental, life-span perspective of intelligence, focussing on adulthood, and distinguishing between its practical and cognitive aspects. Looking at research findings, they conclude that plasticity is paramount in the first part of life, giving way to multidirectionality and multidimensionality within the wider experience of adulthood. Adequate coverage is given to environment in IQ, the history of testing, clinical assessment, including some pertinent comments on assessing minorities, and to group and individual testing. The chapter on giftedness is poor.

Intelligence to the psychologist is like schizophrenia to the psychiatrist. I quote one of the con-

tributors—"The nature of intelligence is a highly complex issue", and a psychiatrist at a recent meeting on schizophrenia—"it is very difficult to understand".

JOAN FREEMAN, *Honorary Tutor, Department of Education, University of Manchester*

Guide to the Use of Psychotropic Drugs. By DONALD GILLIES and MALCOLM LADER. Edinburgh: Churchill Livingstone. 1986 Pp 532. £14 95.

This book is primarily addressed to pharmacists. With the current proposals to encourage a greater advisory role for community pharmacists it is important that they have a broad understanding of psychiatry as well as possessing a sound working knowledge of psychotropic drugs. The book is divided into three unequal parts. The first deals with psychiatric conditions, the second with drug usage and therapy, while the third, much the largest, partly lists alphabetically all the psychotropic drugs in current usage, providing relevant information about each.

The first section which takes the form of an elementary introduction to clinical practice is a model of its kind. All the major psychiatric conditions are described with helpful notes on aetiology and management. I did, however, think it unfortunate to state that schizophrenia was an illness for which "there is an increased representation of lower social classes..." this implies causation rather than consequence. Despite such occasional blemishes I would wholeheartedly commend this section to any non-clinician looking for a succinct up to date introduction to psychiatry.

The next section can be best described as an introduction to clinical psychopharmacology. In my opinion this is rather too terse. A general brief description of synaptic transmission and the sites of action of psychotropic drugs would have been helpful. There are three main categories of drugs considered (antipsychotic, antidepressant and anti-anxiety drugs) plus lithium and "miscellaneous". One objection in this section: I cavill at the collective term tricyclic antidepressants; the tricyclic structure is not exclusive to antidepressants, nor does it include all non MAOI antidepressants. Another quibble, pimozone is not really very closely related to haloperidol, and being a much more selective dopamine receptor blocker is less likely to cause drowsiness and to promote weight gain.

The third section which takes up 470 of the 532 pages takes the form of a psychotropic mini-Martindale. There is a great deal of repetition. For example the mode of action of the monoamine

re-uptake antidepressants is solemnly repeated word for word for each drug in this category. The same is true for side effects. Without such repetitions the book could be made much slimmer.

At the end one is left with the question: What advantage for pharmacists does this book have over the National Formulary, a regularly updated work which is widely distributed by the DHSS? There are two. Firstly it is an advantage to have the relevant pharmaceutical information placed in a clinical context; secondly it must be more convenient for pharmacists working in a psychiatric setting, as well as for students of pharmacy to have all the psychotropic drugs placed together. In addition it would be a useful reference work for nurses on psychiatric wards.

TREVOR SILVERSTONE, *Professor of Human Psychopharmacology, German Hospital, London*

Mind, Brain, Body: Toward a Convergence of Psychoanalysis and Neurobiology. By MORTON F. REISER. London: Harper & Row. 1986. Pp 228. £17.50.

Psychoanalysis and neurobiology occupy different domains with different conceptual languages, and the relationship between them can seem obscure. Can they have anything to say to each other? Reiser believes so. He describes a hypothetical situation of a popular baseball match being shown on TV. As one team scores, three overweight middle-aged supporters of the losing team die of a heart attack. On the other hand, if the other team had scored, three different men might have died. The physical stimuli are the same in each case; only the meaning is different. The question is 'How is input at the level of meaning transduced into a physiological reaction?'.

Reiser draws on raw data from his own clinical psychoanalytic practice, to illustrate processing at the level of meaning. Describing two successful analytic treatments, he diagrams the complex network of ideas, memories and fantasies. He stresses the complexity of memory processes in the patient, in the analyst, and in the interaction between. For example, the patient's description of the onset of a recent phobia prompted a memory *in the analyst* of a dream from an earlier period of the treatment. Drawing attention to this dream led to recognition of the meaning of the phobia, with immediate symptom relief. Reiser notes that "case reports which deal only with the process in the analysand may be recording only one part—a solo version—of what is really a two part play—a duet".

In going on to discuss psychosomatic states, Reiser outlines some of the complex feedback systems

involving different areas of the brain at higher and lower levels, and the endocrine system. He takes anorexia nervosa as an example of a syndrome which demonstrates how social and cultural influences interact with psychological and biological processes. Emphasizing that the brain should be regarded as part of the body, he suggests that schizophrenic and affective disorders could be understood as stress disorders in which the brain is the target organ. These syndromes may begin with disturbance at the level of meaning, which may be conveyed in language, but once such a syndrome is fully developed, it may become therapeutically beyond the reach of words alone. This clearly has implications for the linking of verbal psychotherapy and psychopharmacological approaches.

There are many interesting and original ideas in this book, but overall I found it unsatisfying, seeming to promise more than it delivers. Despite Reiser's avowed aim of demonstrating points of contact between the disciplines, these possible links and their implications seem insufficiently elaborated.

PHIL MOLLON, *Psychologist, Dryden Road Hospital, Tyne and Wear*

The Frontal Lobes. By DONALD T. STUSS and FRANK D. BENSON. New York: Raven Press. 1986. Pp 303. \$65.50.

It is of interest that the frontal lobes, estimated to cover around one third of the total cortical surface of man, remains an enigma. We seem to know far less about the frontal lobes than other areas of the brain, although reports of disturbances of behaviour following damage to the frontal cortex have been recorded for over a century. These regions, in addition to other new well described areas of the brain, compromise association cortex, areas we now know that are intricately involved with higher cortical function in contrast to their presumed redundancy of early theorists who thought of them as silent areas.

This comprehensive review of the frontal lobes will come with great relief to clinicians who wish to grapple with some of the more important aspects of frontal lobes structure and function in relationship to their discipline, being succinctly written and timely in the sense that the frontal lobes have once again recently become of great interest to people of different disciplines, but in particular neuro-psychiatrists. Thus, the clear delineation of frontal lobe syndromes, alongside other neurophysiological abnormalities that occur secondary to focal damage, has

been important for the growth of behavioural neurology, and techniques for monitoring the frontal lobes and frontal lobe function have become more readily available recently with such technology as MRI and PET scans.

The book initially outlines neuroanatomical and neuropathological considerations, and then provides an overview of the methodology for monitoring frontal lobe activity both experimentally and clinically. Disorders which involve frontal lobe function are considered, and these authors raise the possibility that frontal lobe function not only is associated with the well known frontal lobe syndrome, but is also abnormal in such conditions as the Capgras syndrome, reduplicative paramnesia and a confabulation.

Frontal lobe abnormalities in more purely psychiatric syndromes such as schizophrenia and depressive illness do not receive much attention, in spite of the considerable evidence that now exists showing that at least a sub-group of patients with schizophrenia have clearly demarcated abnormalities of frontal lobe function. Nonetheless, psychiatrists who wish to familiarise themselves with the complexities of frontal lobe influence over other neuronal systems, and the most appropriate ways to detect frontal lobe dysfunction clinically will gain considerably from reading this book.

Finally, the authors give their own theory of frontal lobe function, after reviewing those of others. The essence of it is based upon a hierarchical division of frontal lobe functions controlling other systems. The "pre-frontal cortex attends, integrates, formulates, executes, monitors, modifies, and judges all nervous activities", but further is also intimately involved in self-awareness and self-consciousness. Whether or not one accepts such a view, the fact that all those who have studied the frontal lobes believe that they are involved in man's highest cognitive functions and are related to the organisation and planning of behaviour would mean that they become a natural subject for study by all psychiatrists. This book is recommended as an excellent volume for anyone's library.

MICHAEL R. TRIMBLE, *Consultant Physician in Psychological Medicine, The National Hospital for Nervous Diseases*

The Father: Contemporary Jungian Perspectives.

Edited by ANDREW SAMUELS. London: Free Association Books. 1985. Pp 266. £22.50 (hb), £8.95 (pb).

Except for one Freudian paper, this book is a collection of papers on or relevant to the actual father and

the father-archetype by Jungian analysts. Several of these are trying to begin to remedy the dearth of psycho-analytic writing on the role of the father in personality development. Samuels feels that in the past decade or two the patriarchal castrating he-man, if he ever existed, has given way to the kinder, loving, sensitive father. Although basically a positive change, the 'new' father may be unhappy because he has not freed himself sufficiently from the mother, so we still have an unbalanced situation. A paper by David Kay describes a patient whose father had mothered or indeed over-mothered him for his first six months of life, and then left him to go away on business. There were difficulties in establishing a personal identity and gender confusion which (on the evidence of the changes in the analysis) were the result of these early passionate experiences with this unusual father. As I happened to have had this father in therapy I can vouch for Kay's completely accurate reconstruction of what the father was like from Kay's transference/countertransference emotions with the son and their evolution during analysis. For example, Kay says "For a man who has serious doubts and confusion about his sexual identity and potency, the arrival of an infant, particularly a first-born beautiful male child, can have a very powerful and crucial effect. The father, in the example quoted, saw and experienced his son as the 'Divine child hero'—living proof that his self doubts were at an end." When I saw the father in therapy thirty years later he was deeply involved in one more of several relationships of identical nature with boys and young men, on to each of whom in turn had been projected this same semi-divine image. It is a very comforting validation of modern analytical technique and of Kay's skill in particular that Kay had reconstructed the feeling of this patient's early relationship with his father so accurately.

In another excellent clinical paper Eva Seligman describes a series of 'half-alive' patients for whom the father had been in some sense absent, and therefore unable to help the patient free himself from a mutually collusive embrace with the mother which had nourished an illusory 'one-ness' with her.

John Beebe contributes a thoughtful article about the effect of the father's emotional attitude towards his son on the son's psychology and destiny. Andrew Samuels' article on 'The image of the parents in bed' discusses the primal scene both in terms of how the infant experiences the parent relationship and how he projects the archaic, archetypal parental images. He hypothesizes that his images of two-ness (first mother and baby, then the parents together) create a climate in which images of three-ness can begin to exist and the paternal image can emerge. Allenby's

paper on father fixation balances Layland's on the need of a loving father. There are three further articles mainly on the archetypal aspects of the child-father relationship, including Jung's seminal paper originally written in 1909.

I think that bringing these papers together in one volume, with the valuable linking explanations of the main introduction and the subsidiary introductions to each paper, was a very worthwhile undertaking for which Samuels deserves credit. In this book the father comes to life as a whole human being in analytical theory.

J. W. T. REDFEARN, *Director of Training,
Society of Analytical Psychology, London*

The Nightmare: The Psychology and Biology of Terrifying Dreams. By ERNEST HARTMANN. London: Harper & Row. 1986. Pp 294. £16.95.

Ernest Hartmann is both a psychoanalyst and a leading American sleep researcher. This book, the fruit of many years research in both fields, demonstrates an exemplary integration of clinical insight and experimental data, the results of which have important implications well beyond the specific phenomenon of terrifying dreams.

Hartmann distinguishes between three separate conditions which have often been grouped together under the single heading "Nightmare". These are 'ordinary' or typical nightmares, post-traumatic nightmares and night terrors. The major research concerns a group of lifelong sufferers of 'ordinary' nightmares. On the basis of several comprehensive studies he proposes the concept of "thin boundaries" as the essential characteristic shared by these people, i.e. they have a strong tendency to blur the distinctions between different categories of experience such as sleeping vs. waking, fantasy vs. reality or self vs. other. Hartmann is at pains to consider the non-pathological indications of these findings alongside the pathological ones. Thus, while such people are unusually defenceless, open and vulnerable and this may constitute a susceptibility to schizophrenia, thin boundaries may also be an advantage in both artistic and scientific endeavour.

Although hardly conclusive, the evidence which he has accumulated in this area is a valuable contribution towards understanding the link between creativity and madness.

The remainder of the book concerns clinical and biological aspects of the nightmare. Nightmares are interpreted as expressions of basic childhood fears, especially of helplessness and catastrophic loss. Post-traumatic nightmares occur when these fears

have been realised but only in circumstances where mental boundaries are unusually vulnerable. Normally, these nightmares gradually become integrated with ordinary dream material but in extreme cases the traumatic memory becomes encapsulated and can be "set off" by a variety of physiological or emotional disturbances.

The biology of the nightmare is thought to involve an increase in dopaminergic activity at the forebrain. Hartmann hopes that further investigations might lead to the establishment of a biological basis to the concept of mental boundaries. If so, an important bridge between biology and psychology will have been created.

WARREN COLEMAN, *Psychotherapist,
Institute of Marital Studies, London*

The Provision of Mental Health Services in Britain: The Way Ahead. Edited by GREG WILKINSON and HUGH FREEMAN. The Royal College of Psychiatrists: London. 1986. Pp 197. £7.50.

This volume brings together the contributions of twenty speakers and the edited discussions of a conference organised by the College and the DHSS.

The editors' introduction draws comparisons with a similar conference in 1972. At that time informal and piecemeal developments in district psychiatric services were being embodied in government policy. The present volume illustrates something of the scope of subsequent change and obstacles to future development. The contributions range from a few blandly hopeful restatements of policy, which suggest that with more resources, flexibility, leadership and teamwork all will be well, to the hackneyed criticisms of mental hospital closure. Between these extremes there are reasoned appreciations of the inevitable difficulties and dangers of change. There is, for example, an interesting analysis of the differing structures and organisation of Health Services and Social Services and how these impede continuity of care.

Old age psychiatry and services for the mentally handicapped are well covered, while the views of consumers and those in disciplines other than psychiatry receive attention, but less space.

As in most recent accounts of change in psychiatric services, one detects an uneasy and rather reluctant acceptance of proposed developments. There are anxieties about the rhetoric and the wishful thinking, and how, with the decentralisation of services and the dispersion of responsibility, it will be possible to maintain control of policy. There are doubts too about the difficulty of joint planning and financing and the inevitable queries about cost.

Excellent services in many parts of the country still have serious gaps. Progress is slow and "the sheer inertia of mind and matter" which Enoch Powell foresaw, has not yet been overcome. Since history has no ready-made answers for us, our slow advance gives us time to follow the innovations and to avoid some of the pitfalls which this book describes.

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Therapeutic Factors in Group Psychotherapy. By SIDNEY BLOCH and ERIC CROUCH. Oxford: Oxford University Press. 1985. Pp 342. £16.00.

Group psychotherapy is well established as a treatment mode in modern psychiatric practice. Once regarded as the poor relation of individual psychotherapy, the experiences of those involved in the Northfield Military Hospital work during World War II led to increasing recognition of group therapy as a powerful tool in its own right, and a body of theory developed with various 'schools' promoting their particular approaches. Therapeutic communities represent one aspect of applied group therapy, but ignorance of group dynamics may limit both the potential and the effectiveness of in-patient units and day services alike, whereas their exploitation offers well-recognized benefits in many forms of treatment.

Although many of the benefits of group therapy have been recognized clinically, and therefore become enshrined in practice, the actual way in which group psychotherapy works remains poorly understood. A considerable body of research work has been published, much of it in the U.S.A. but too often studies have been badly designed or carried out on experimental volunteer populations, so that extrapolation of the findings has contributed little to our understanding of group psychotherapy with patients.

Therapeutic Factors in Group Psychotherapy is a most welcome and long overdue addition to the literature in this field. The authors have reviewed systematic research in group psychotherapy over the past thirty years, and have assembled the knowledge that has emerged on how and why group therapy works, setting out to dispel the vagueness generally associated with this subject. In the first chapter they consider the important matters of definition and classification, leading to their own selection of ten therapeutic factors and the methods which have been used to study them. Subsequent chapters are devoted to each therapeutic factor in turn, with definition, theoretical aspects and empirical research covered in detail, and a comprehensive list of references. The

final chapters deal with therapeutic factors overall, their relative neglect, their importance, and the prospects for their future study. A useful appendix of experimental research setting out the findings of cited studies in tabular form, and separate author and subject indexing ensures that this book is easily accessible for reference by prospective researchers and all who seek to become better informed about group psychotherapy.

At a mere £16.00 for a well-printed text in hard covers, this book is a real bargain. It should be available in every psychiatric library, and recommended reading for all who use groups in the course of their work. Trainees embarking on group psychotherapy for the first time, as well as experienced therapists whose clinical knowledge is unlikely to be equalled by their researches in the field, will find much food for thought in this readable book, which may well serve to stimulate group therapists into more and better therapeutic factor research with patient groups. The authors have distilled their subject matter into a compact volume which is likely to be an important source of reference for the next decade; it is one for all group therapists to buy and consult.

PAMELA ASHURST, *Consultant Psychotherapist,
Royal South Hants Hospital, Southampton*

Prediction in Criminology. Edited by DAVID P. FARRINGTON and ROGER TARLING. New York: State University of New York. 1985. Pp 278. \$39.50 (hb), \$14.95 (pb).

This is mainly a book for criminologists and those others who are both interested in telling the future and well versed in the nomenclature and methods of statistics. In addition to British contributors there are several from the USA and one from Canada. The predictions covered include recidivism, absconding, and risk and failure among parolees. There is a very interesting chapter on 'The Politics of Prediction' which highlights the folly of the doctrine that taking specific action against individual offenders has an influence on crime in general. Throughout the book a blindingly obvious theme recurs: that predictor variables will never be refined until the criterion variables of, for example recidivism, are something more than simply reconviction and the type of offence committed.

There are two chapters for the psychiatrist. The first looks at self injury in a population of female Borstal detainees, several variables were found to discriminate well between self-injurers and non-injurers but they were clearly independent: a history of psychiatric treatment, institutional experience, self-injury; high scores on neuroticism and aggression, low scores on happiness and self-esteem. In a prospective study these and other variables combined to form a risk rating which accurately predicted 83% of all self injurers.

The second chapter of interest is from Broadmoor Hospital's Psychology Department. It describes a cohort of

discharged patients studied retrospectively and claims to be the first work of its kind to use data collated from clinical assessment. The authors express the hope that developing their method will lead to the application of empirically validated criteria to patients under consideration for discharge and to the evaluation of treatment programmes. But the authors do little to temper the pessimism which surrounds predictive research in the psychology and psychiatry of dangerousness and there is little new in their showing that previous convictions, previous psychiatric admissions and types of offences were most highly related to the four outcome measures: committed further assaults, subsequent court appearances, re-admission to Broadmoor, and not remaining in the community for the entire five year follow-up.

The Broadmoor study does make a plea for discrimination between the treatment needs of patients. It emphasises the lunacy of treating over-controlled homicidal offenders who are so inhibited that they have no behaviour, other than the catastrophic, for coping with stress alongside undercontrolled offenders who need to increase these same inhibitions.

PAUL BOWDEN, *Consultant Forensic Psychiatrist, Bethlem Royal and Maudsley Hospitals*

Clobazam: Human Psychopharmacology and Clinical Applications. Edited by I. HINDMARSH, P. D. STONIER and M. R. TRIMBLE. Oxford: Oxford University Press for the Royal Society of Medicine. 1985. Pp. 215. £8.00.

This book derives from a symposium on the 1,5 benzodiazepine clobazam. It consists of 30 separate papers almost all of which directly concern the drug. A number refer to studies of performance tests on volunteers, others concern anxiolytic effects and the latter part of the book is about the anticonvulsant effects of clobazam. Several of the studies tend to go over similar ground and indeed this is almost inevitable when so many groups of authors are dealing with a relatively limited subject. Some investigations are well done but many are very small and a few have defects of methodology. They are inclined to be reported in rather excessive detail, for example several authors specified that subjects who were known to be pregnant or suspected of being so were excluded from the study, a precaution that I would have expected to be so customary that it could go without saying. On the other hand the topic of the risks of benzodiazepine prescription does not receive as much emphasis as one might have hoped in a volume entirely concerned with a benzodiazepine for illicit ingestion by polydrug abusers and another mentions a difficulty in obtaining the planned sample size because of local changes in attitude to benzodiazepine prescribing but otherwise the question of the possible problems associated with benzodiazepine use in neurotic patients is scarcely mentioned. The papers on anticonvulsant effects generally concern the addition of clobazam to standard regimes in epileptic patients whose control is poor. The results are conflicting but fortunately the last paper reviews the use of clobazam in epilepsy. Many readers might find a review on the other aspects of

clobazam use more rewarding than the perusal of this volume which at times seems a little like promotional literature. At its price of £8 however some may find it a worthwhile purchase in view of the few very good chapters and perhaps for the insight it offers into the conduct of clinical studies.

EVE C. JOHNSTONE, *Consultant Psychiatrist, Northwick Park Hospital, London*

Family Management of Schizophrenia: A Study of Clinical, Social, Family and Economic Benefits. By IAN R. H. FALLOON *et al.* Baltimore: Johns Hopkins University Press. 1985. Pp. 207. £25.45.

It is now a truism that for all that the major advances of medicine over the last half-century have been based on the intense development of specialist expertise, this has its inevitable limitations. So we move on to the whole world of the individual, to an advocacy of holistic medicine that varies from the trite to the crackpot, only to find that here again there are limitations. Because of the social nature of man, the involvement of the family is a crucial element in any, but especially in any long-term, illness. Not only can the family provide an elaborate mechanism for humiliation or ill-treatment, or indifference, as well as for support, but the family exists in a changing world.

The family as the unit of care is certainly more popular with politicians, for it is likely to be cheap. But when we come to research the performance in family management of an illness like schizophrenia, the difficulties are nothing less than awesome. The fluctuations of the illness require a long-term approach now only attainable in this country with difficulty.

In this important, academically sound and fascinating volume we see the fruits of a decade's study. Dr Falloon describes four major aims: to compare behavioural family therapy and individual supportive psychotherapy after the treatment of a florid exacerbation in patients who lived in a stressful family environment; to assess the changed functioning of family members; to conduct an analysis of the costs of the two approaches; and to analyse the variables that might influence outcome.

There seems, despite suggestions to the contrary of earlier work, to be a series of important advantages accruing from the factual education and enhanced problem-solving skills in families helped to cope with a schizophrenia member. The social benefits for the family were combined with improved functioning of their patient when compared with individual treatment.

The authors provide an admirable review of the literature, keep well away from excessive enthusiasm and provide an elegant and instructive monograph that merits attention and respect from colleagues of all grades of seniority. And like all important research, it raises more fascinating problems for the future.

ERIC WILKES, *Emeritus Professor of Community Care and General Practice, University of Sheffield*

Making Sense of Psychiatric Cases. Edited by MAURICE GREENBERG, GEORGE SZMUKLER and DIGBY TANTAM. Oxford: Oxford University Press. 1986. Pp. 258. £9.95.

This book fills a gap for psychiatrists in training. As the author states, it is necessary to become practised in history taking and mental state examination. The Institute of Psychiatry booklet *Notes on Eliciting and Recording Clinical Information* already provides a detailed structure for history taking. However, until now there has not been a book giving illustrative case histories to complement this.

The authors point out that while at places like the Maudsley much time is spent on history taking, in other places with a more busy work load, junior doctors may find a text book helpful. Following an introductory case concentrating on how to organise and select information there are eleven further case histories covering a wide spectrum of commonly encountered conditions such as depression, schizophrenia, alcoholism and overdoses. Each case history is founded on a genuine clinical case and presented under headings of presenting complaint, family history, personal history, past medical history, past psychiatric history, previous personality, mental state examination, physical examination, summary formulation, diagnosis and etiology, further investigation, management and prognosis. It is rightly emphasised that if the patient is in a too disturbed state on admission to give a family history this should be obtained later and be included in making a comprehensive formulation, a thing junior doctors often neglect.

A criticism of the case histories is that the headings are then repeated twice as a means of following further development of the case. As a result, at times, the descriptions become somewhat obsessively repetitive. For example, in the case of anorexia nervosa, after going into great detail in the initial formulation, as to why the diagnosis was anorexia nervosa, there were two further reformulations in the chapter using the same headings as previously, saying each time that the diagnosis still remained one of anorexia nervosa. Another concern is a more personal one. As both a practising general psychiatrist and analyst, I feel that psychiatrists can get incarcerated in the history taking technique with over application of text book theories in not only making formulations, but then applying them in a logical manner as answers to management. This might stultify development of their own individual sensitivity.

However, it is important to be fully conversant with history taking, mental state examination and making comprehensive formulations including a diagnosis. Often junior doctors try to opt out of committing themselves for formulations. In this context the book is a necessary contribution and should become standard reading for all junior psychiatrists approaching their MRCPsych. examination.

RICHARD LUCAS, *Consultant Psychiatrist, Claybury Hospital, Essex*

Sharing Psychological Skills: Training Non-Psychologists in the Use of Psychological Techniques. A special issue of the *British Journal of Medical Psychology*. Guest editors HILTON DAVIS and PETER BUTCHER. Leicester: The British Psychological Society. 1985. Pp. 96. £6.95.

This 'book' is an edition of the *British Journal of Medical Psychology* published in hardback. It has neither a preface nor a foreword and although a brief index has been added it retains the feel of a journal which for this reader dilutes the enjoyment of the read irrespective of the contents. The common theme of all the contributions is the teaching of psychological skills to non-psychologists with each of the authors actively involved in running training courses.

Some of the papers are essentially a review of the literature while others are anecdotal reports of specific educational programmes. In all there are nine contributions as well as an introduction by the guest editors. Three are on the training of patients or relatives in self-help methods while the remaining six discuss the dissemination of psychological treatment skills to other professional and voluntary caregivers including general practitioners, nurses, hospital medical staff and marriage guidance counsellors. The contents of the teaching programmes outlined in the book range from behaviour modification to basic counselling and from adaptive skills in the elderly to a broad based, 12-week adult education course on 'strategies for living'. In general there is a welcome emphasis on the practical rather than the theoretical aspects of psychological skills training.

While inevitably there is some overlap in a collection of papers of this nature the final selection does have a somewhat arbitrary appearance. Given the constraints on space I would have liked the book to have concentrated either on psychological skills training for various patient groups or alternatively on training for professionals. The book would also have benefited from an introductory paper on the organisational and practical constraints which may substantially affect the quality and quantity of teaching within the Health Service over the next few years. A related issue is discussed in, arguably, the most stimulating paper by Keith Nichols who reminds us that no matter how well we teach psychological skills to general medical and paramedical staff it is only through changes in organisation and practice at ward level and integration of psychological ideas into the medical subculture that the influence of such teaching can be maximised and maintained.

In spite of the rather arbitrary choice of material this book has much to recommend it with most of the contributions being clear, concise and convincing. It will doubtless prove a useful reference source for all of us who are faced with the daunting task of organising, implementing and evaluating a training programme on psychological treatment methods for either patients or colleagues.

ROBIN DAVIDSON, *Top Grade Psychologist, Holywell Hospital, Co. Antrim*

The Psychology of Gifted Children: Perspectives on Development and Education. Edited by JOAN FREEMAN. Chichester: John Wiley. 1985. Pp. 414. £24.75.

This is a volume in the Wiley Series on Developmental Psychology and its Application. The Series Editor, Professor Connolly, describes the book as "... a remarkably diverse set of essays", and this comment rather neatly sums it up. Twenty-two chapters, two of them contributed by Dr Freeman, cover topics as diverse as identifying gifted infants, sleep and mental efficiency, musical and mathematical giftedness, emotional aspects and delinquency, giftedness in Israel and China, and problems in the education of gifted children.

But what is "giftedness"? It still comes as a surprise to this reviewer that the former British Grammar Schools, catering for the most academically able 15 to 20% of the population, were regarded by foreign educators as "schools for the gifted". Are we talking of this able 20% or the most able 1 to 5%? Is giftedness a general ability or are isolated talents to be similarly regarded. As yet, there is no clear consensus, but Eysenck's chapter on 'The Nature and Measurement of Intelligence' provides a masterly, succinct overview of the complexities of the area.

Freeman's own chapter on 'Emotional Aspects of Giftedness' provides the background to and outcome of her own studies. She found that whereas the children whose parents had joined the National Association for Gifted Children were significantly more disturbed than normal controls, equally gifted children identified in schools (but not Association members) were no more deviant than the normal. Nevertheless, children who spend hours studying, practicing musical instruments and the like do have special needs, and these are sensibly discussed.

Less satisfying is Brooks' résumé of his study of 'Bright Delinquents'. His sample appeared to be underachieving academically and to have a high rate of father absence. Otherwise, there was little to distinguish them from the control group.

Overall, the book provides an interesting introduction to an important area. Many questions are raised: few are answered. But in the discussion some fundamental views on our attitudes to the gifted are challenged.

WILLIAM YULE, *Senior Lecturer in Psychology,
Institute of Psychiatry, London*

Behavior and Cancer. By Sandra M. Levy. London: Jossey-Bass. 1985. Pp 257. £28.50.

Although there is still no convincing evidence that psychological factors predispose to the development of cancer in man, animal and epidemiological research increasingly indicate that many factors related to ways of life may be major factors. Psychiatrists have tended to concentrate on a narrow range of psychological variables and have shown less interest in the ways in which personality, mental state and social circumstances affect established primary risk factors, such as diet, smoking and acceptable hazards and sexual activity. It is a virtue of this book that it takes

the wider view. Dr Levy begins from epidemiological and animal evidence and sets behavioural factors in context. Successive chapters review the major risk factors, diagnosis and screening, immune and hormonal mechanisms, behavioural factors contributing directly to the progression of cancer and behavioural implications.

Dr Levy uses standard medical and psychological references, case examples and a somewhat unhappy choice of rather pretentious literary quotations to provide a clear summary of behavioural influences on the onset and progression of cancer and to draw clinical conclusions about screening and treatment. This is a useful exercise and, allowing for some stylistic irritations, a readable introduction to a subject and to literature which has become almost inaccessible to the non-expert. It is, however, a relatively short book and, as is evident in the presentation of the medical background and in the glossary, it is intended for a wide non-medical audience. Psychiatrists will find it informative and a great deal easier to read than other less comprehensive reviews.

RICHARD MAYOU, *Clinical Reader in Psychiatry,
University of Oxford*

Dictionary of Psychiatry. By H. Walton. Blackwell Scientific Publications. 1985. Pp. 170 £12.50

As communication is so fundamental to psychiatry I wonder why we have waited so long for a book of this kind, a compact but reasonably comprehensive dictionary of the subject. The editor has been assisted by a panel of psychiatric dignitaries (mostly professors) so that the definitions are authoritative. The text is also a model of conciseness and clarity. It was a pleasure to range through the definitions, from *birth trauma*, through *dasein* and on to the *death instinct*.

Most of the terms included are much in current use, but requiring definition for all that. However, a few pleasures for lexicologists lurk among the pages. I now know what *geloplegia* and *hypnobia* are. Of course one can always quibble over what should be included in or omitted from a dictionary of this sort. Although the intended readership is wide, including the lay public, some of the inclusions seemed superfluous. Again, certain drugs are included, but the grounds for their selection were unclear to me.

Sometimes the text goes beyond mere definition. For example, we are told that psychiatric treatment is associated with social class, intensive psychotherapy or psychoanalysis being prescribed more often for class 1 and electroconvulsive therapy for class 5. Also brief but fascinating biographical notes are provided on famous figures. Under B the following appear: Bateson, Beers, Binet, Binswanger, Bleuler, Brentano, Breuer, Brill, Barrow, Burt.

I wonder if £12.50 is a little expensive for a book of this size, but none the less I predict this will be a popular book, and deservedly so. I recommend it.

ROGER FARMER, *Lecturer, St Mary's Hospital, London*

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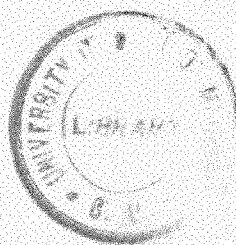


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The standard of statistical work in papers submitted for publication has proved to be very variable. Authors using any statistical methods at all are strongly advised to study the paper 'Statistical guidelines for contributors to medical journals' (D. G. Altman *et al.*, *British Medical Journal*, 7 May 1983, **286**, 1489-1493) at the earliest possible stage in preparing their work for publication.

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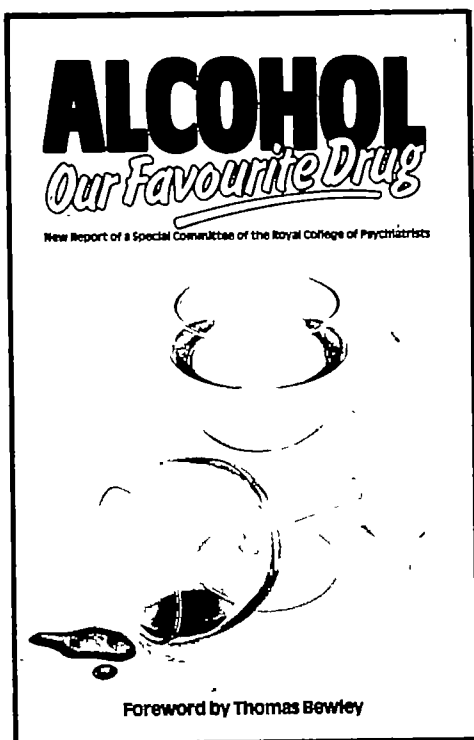
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Parasuicide and Unemployment

S. PLATT

The current economic recession in Europe and North America has led to a renewal of interest in the possible consequences of mass unemployment for the nation's health. This annotation assesses the relationship between unemployment and one indicator of morbidity: parasuicide (non-fatal deliberate self-harm). A previously published review of the literature is supplemented by more recent research, particularly from Edinburgh and Oxford.

The relationship between parasuicide and unemployment among individuals

At least twenty studies have been published which include information on the proportion of parasuicides who are unemployed on admission to hospital. A number of these (e.g. Bruhn, 1962; Edwards & Whitlock, 1968; Hawton *et al.*, 1982; Bille-Brahe *et al.*, 1985; Hawton & Rose, 1986) draw attention to the fact that the unemployment rate among parasuicides is considerably in excess of that in the general population. Investigators have also calculated the incidence of parasuicide by employment status, consistently finding higher rates among the unemployed. Thus, in 1982, the parasuicide rate among unemployed men in Oxford was 708 per 100 000, while the equivalent figure for employed men was 46; the relative risk (the ratio of the parasuicide rate among the unemployed to that among the employed) was 15.4 (Hawton & Rose, 1986). In Edinburgh in the same year the parasuicide rates were 1345 and 114 respectively, giving a relative risk of 11.8 (Platt & Kreitman, 1985a). While there has been a downward trend in the parasuicide rate among the unemployed in Edinburgh over the period 1968-1984, the relative risk has fallen below 10 in only three years of the series.

The parasuicide rate among the unemployed tends to increase with longer durations of unemployment. In recent years the relative risk of parasuicide among men unemployed for more than a year (compared with employed males) has ranged between 26 and 36.6 in Oxford (Hawton & Rose, 1986) and 13.5 and 20.4 in Edinburgh (Platt *et al.*, 1985). However, even recent job loss has been shown to be associated with relative risks of considerable magnitude. In Edinburgh the relative risk among those unemployed for less than four weeks has not fallen below 4.3 during the years 1982-84.

The Edinburgh and Oxford data sets also permit the calculation of parasuicide rates among males after controlling for possible confounding effects,

such as age, social class and area of residence. In both cities, unemployed men of all age groups show elevated relative risks compared with the employed. The highest parasuicide rates among the unemployed are found in the 30-49 age group in Oxford and in the 25-54 age group in Edinburgh (Hawton & Rose, 1986; Platt, 1985). Among both employed and unemployed in Edinburgh an inverse relationship between parasuicide and social class was evident, with highest rates in social class V (Registrar-General, 1980). The relative risk of parasuicide among the unemployed was significantly high in each social class group, ranging from 8.4 in class IV to 29.0 in classes I and II. While there was considerable variation in parasuicide rates by employment status across different geographical areas of Edinburgh, the lowest relative risk (7.4 in 1970-72 and 4.4. in 1980-82) was significantly greater than zero (Platt & Kreitman, 1985a).

It is noteworthy that in Edinburgh the highest relative risks are found in the years (1968-74), social classes (I and II), age groups (55 years plus) and areas (with above average proportions of professionals and owner-occupiers) characterised by the lowest rates of unemployment; i.e. there is a marked tendency for the relative risk to fall as the unemployment rate rises. Nevertheless, an analysis of trends in population-attributable risk shows that in recent years as much as half of the overall parasuicide rate in Edinburgh may be "attributable" to unemployment, since the declining rate of parasuicide among the unemployed over the years has been more than offset by the increase in the numbers at risk. In consequence, the absolute numbers of unemployed parasuicides and the proportion they form of all hospitalised parasuicides have increased steadily over the period (Platt & Kreitman, 1985a).

Personal characteristics of employed and unemployed parasuicides

In their study of male parasuicide patients admitted

to the general hospital psychiatric service in Oxford, Hawton & Rose (1986) showed that, compared with the employed, the unemployed were significantly more likely to be psychologically vulnerable, i.e. to have received psychiatric treatment in the past, to be in treatment at the time of the parasuicide episode, to be diagnosed alcoholic, and to be parasuicide repeaters. Platt & Duffy (1986) examined correlates of joblessness in two cohorts of first-ever male parasuicides: one admitted to the Regional Poisoning Treatment Centre (RPTC) in Edinburgh during the years 1969–71 (a period of relative prosperity), and the other admitted to the RPTC during 1980–83 (a period of economic recession). Overall, the unemployed were significantly more likely to be unmarried, to live alone or in an institution, to have experience early separation from their mother, to have received psychiatric treatment, to be given a diagnosis of abnormal personality, to have a criminal record, to be in serious debt, and to misuse drugs habitually. These differences appeared to be unaffected by the prevailing economic climate.

A further examination of variation in personal characteristics among parasuicides was undertaken by Platt (1986). In a cohort of unemployed male first-ever parasuicides admitted to the RPTC in 1982–83, significant differences by duration of unemployment were found on four of the response variables: civil state, age, personality diagnosis, and receipt of psychotropic medication. While there was no clear duration-related trend in respect of civil state or age, longer durations of unemployment were significantly associated with a greater prevalence of personality abnormality and receipt of psychotropic medication. Hawton & Rose (1986) also state that in their cohort of male parasuicides admitted in Oxford, significantly more of the long-term unemployed (compared with the short-term unemployed) are psychologically vulnerable. However, no data are provided.

In a third study from Edinburgh, Platt & Dyer (1986) measured variation in psychological features associated with unemployment among a sample of economically active male parasuicides admitted to the RPTC in 1979. Employed and unemployed individuals did not differ in the degree of suicidal intent, but the unemployed were significantly more likely to be depressed and to describe feelings of hopelessness. Even after partialling out the effect of depression, the unemployed remained more hopeless.

The ecological relationship between parasuicide and unemployment

Analyses of the relationship between unemployment and parasuicide rates across geographical areas have

revealed a significant positive correlation in Brighton, 1966–68 (Bagley *et al.*, 1973) and in Edinburgh, 1968–73 (Buglass & Duffy, 1978). Platt & Kreitman (1984, 1985a) correlated male unemployment and parasuicide rates across 23 electoral wards (1970–72) and 31 regional electoral divisions (1980–82) in Edinburgh. In both periods the ecological correlations were highly significant ($r=0.76$, $P<0.001$ in 1970–72; $r=0.95$, $P<0.001$ in 1980–82); the increase in correlations over the decade was also significant ($P<0.001$). Platt & Kreitman (1985a) further examined the relationship between unemployment and parasuicide to determine if the connection between them could be ascribed to common third factors such as social class and poverty. Partialling out the effect of variations in the social class composition of the city districts reduced the magnitude of the observed correlations (from 0.76 to 0.51 in 1970–72 and from 0.95 to 0.81 in 1980–82), but the resulting correlations remained significant for both periods. On the other hand, after controlling for the level of poverty the partial correlation in 1970–72 fell to a non-significant level ($r=0.13$) (data were not available to perform this analysis for 1980–82.) Thus unemployment appears to be associated with parasuicide only in so far as it relates to poverty or to some other variable closely connected with poverty.

Forster & Frost (1986) report a substantial (but not statistically significant) correlation between the medical self-poisoning rate (based on persons age 15–44 years admitted to hospital because of adverse effects of medical agents) and the unemployment rate across 12 regional health authorities in England and Wales in 1978 ($r=0.57$, NS). A multiple regression analysis performed on the medicinal self-poisoning rate as the dependent variable and the psychotropic prescription rate, unemployment rate and admissions capacity rate (a measure of bed availability) as the independent variables demonstrated that only the psychotropic prescription rate met the criteria for retention in the equation.

Only three reports are available on the relationship between unemployment and parasuicide rates over time. Among Edinburgh male residents the temporal association was found to be significantly positive overall ($r=0.62$, $P<0.02$) during the period 1968–83. It should be noted, however, that the relationship between unemployment and parasuicide over the first eight years of the period was strikingly different to that found over the final eight years: it was positive between 1968 and 1975 ($r=0.92$, $P<0.01$); and negative between 1976 and 1983 ($r=-0.67$, NS) (Platt, 1985). In Hartlepool during the period 1974–83 the unemployment rate quadrupled while the parasuicide rate (as measured by

hospital admissions diagnosed as principally suffering from toxic effects of drugs or chemicals) remained fairly constant ($r = 0.23$, NS) (Furness *et al.*, 1985). In Oxford, the correlation between the male unemployment rate and the male parasuicide rate over the period 1976–82 was negative, though not significant ($r = -0.69$) (Hawton, personal communication.)

Discussion

The ecological studies provide somewhat contradictory results. The correlation between parasuicide and unemployment rates across geographical areas is positive, if not always significant, and is markedly reduced when controls for the level of poverty and the psychotropic prescription rate are introduced. Data from Hartlepool and Oxford show that there was no temporal relationship between parasuicide and unemployment from the mid-1970s onwards. In Edinburgh the correlation was found to be positive until 1982, when the parasuicide rate declined precipitately while the unemployment rate continued to rise. This reversed pattern, maintained in 1983 and 1984, might have been anticipated on theoretical grounds: as economic recession worsens, the stigma associated with unemployment would be expected to be less marked, unemployed people would be more integrated in the social order, and their parasuicide potential would decrease. Each unit increase in unemployment above a certain level would be likely to bring about a smaller unit change in parasuicide. When mass unemployment becomes endemic, the change might indeed become negative (such a polynomial equation has been shown by Stack (1981) to fit the relationship between divorce and suicide.

Other explanations of the recent fall in parasuicide incidence include a reduction in the number of prescriptions for hypnotics and tranquillisers (Brewer & Farmer, 1985), a decline in *per capita* alcohol consumption, and a change in cultural attitudes to parasuicide. We also have to consider the suggestion that the fall in the hospital-treated parasuicide rate may be an artefact of changes in medical approaches to the treatment of parasuicide. There is a growing belief among researchers in the field that a smaller proportion of parasuicides are admitted to hospital than formerly. However, there are no empirical data to test this possibility.

The individual-level studies produce more consistent findings and clearer evidence of an association between unemployment and parasuicide. Parasuicide rates among the unemployed are markedly higher than those among the employed, the relative risk rarely dropping below 10 in the Edinburgh series. Relative risk remains significantly high after con-

trolling for social class, age and area of residence. While the impact of recent job loss has been shown to be quite marked, the highest rates (and risks) are found among the long-term unemployed. Finally, the attributable risk analysis shows that over 50% of the overall parasuicide rate may be attributable to unemployment in recent years.

The epidemiological analyses highlight an inverse relationship between the rate of parasuicide among the unemployed (and relative risk) and the prevailing unemployment rate. To account for these findings, Platt & Kreitman (1985a) speculate that the unemployed individual considers himself less socially stigmatised and personally deviant in social situations or contexts of high unemployment than in situations of low unemployment. Consequently, the psychological impact of unemployment may be significantly less marked in high rate unemployment areas, in years of economic recession, and in age groups and social classes where unemployment is a relatively common experience. Discussing the negative temporal relationship between changes in the parasuicide rate among the unemployed and changes in the unemployment rate, Platt & Kreitman (1985a) also suggest that the high rates in the earlier years may reflect a marked degree of psychological and social impairment among those comparatively few individuals who remain unemployed in times of full employment. The decline in the parasuicide rate over subsequent years probably signals the dilution of the 'unemployable' group by others whose status is decreasingly a consequence of personal handicap and increasingly the outcome of impersonal, economic factors.

Platt & Kreitman (1985a) conclude that their findings "are entirely compatible with the hypothesis that unemployment is a cause of parasuicide", although they explicitly recognise that "parasuicide results from the complex interaction of many factors". Their conclusion has been questioned by Shapiro & Parry (1985) and by Hawton & Rose (1986). Shapiro & Parry claim that their finding, that unemployed parasuicides rarely mention unemployment as an important or secondary current problem, gives "no support to the suggestion that those who commit parasuicide see unemployment as being relevant to their action". They go on to warn of the danger of interpreting the higher risk of parasuicide among the unemployed as evidence of a "casual connection". In reply, Platt & Kreitman (1985b) point out that they nowhere suggest that unemployment is a *precipitant* of parasuicide. Indeed, given the finding that parasuicide risk increases with greater durations of unemployment, it would make no sense logically to do so. However, prolonged unemployment may be

a significant *predisposing* factor in suicidal behaviour because it leads to an increase in family tensions, arguments and violence; more depression and hopelessness; increasingly isolation from others; changes in role structure within the family; financial hardship and material deprivation; loss of self-esteem and self-confidence; feelings of reduced self-worth; or any combination of these sequelae. A provoking agent (e.g. argument with spouse) would be more likely to lead to parasuicide in the presence of unemployment (and its associated behavioural correlates) than in its absence (Platt, 1984). Economic recession may also have an indirect effect on psychological well-being and parasuicide by increasingly labour market marginalisation. Those in work are subjected to higher levels of stress, e.g. by enforcing pay restraint, reducing job insecurity and demanding higher productivity. Individuals who in times of full employment are only partially integrated into the workforce and tend to move in and out of work on a regular basis (the subemployed) may find that the opportunities for this lifestyle are severely limited during times of economic instability. Among male parasuicides admitted to the RPTC the subemployed, previously constituting about a quarter of the total, now account for less than 10%. Virtually all of the 'missing' group will have joined the ranks of the long-term unemployed.

In their discussion of the research evidence from Oxford and Edinburgh, Hawton & Rose (1986) consider the possibility of an indirect causal link whereby unemployment may "precipitate or exacerbate factors known to increase the risk of suicidal behaviour, such as social and interpersonal difficulties, poverty and psychiatric disorders". The findings of a higher prevalence of psychiatric treatment and alcoholism among the unemployed

and higher risks of parasuicide in the long-term unemployed would be predicted by such an indirect causal link. However, the findings are also compatible with another type of explanation: that both unemployment and parasuicide are causally related to a third factor. Hawton & Rose postulate that the psychologically vulnerable may be more likely to become and remain unemployed, particularly during a recession, and to engage in self-harmful behaviour. The long-term unemployed may be most at risk of parasuicide because people with psychological vulnerability are over-represented in this group, rather than as a result of the unemployment experience itself. In their conclusion, Hawton & Rose state firmly that we are not currently in any position to decide between these two types of explanation and urge more research on this topic. Clearly their plea deserves to be heard, for both scientific and practical reasons. There are still many areas of ignorance which require illumination, including the link between unemployment and parasuicide among women, the mechanisms by which prolonged unemployment may raise the risk of parasuicide in marginal subgroups, the importance of hopelessness as an intervening social-psychological variable, the temporal relationship between onset of personal difficulties and unemployment among parasuicides, and the role of personality disorder as a possible common link between the two variables. In the meantime, however, we are faced with incontrovertible evidence that the long-term unemployed suffer an unacceptably high risk of parasuicide and that the overall level of parasuicide could be reduced even more if there were a return to fuller employment. Further research is unlikely to alter these facts and their obvious implications for national social and economic policy.

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Genetics of Fear and Anxiety Disorders*

I. M. MARKS

From protozoa to mammals, organisms have been selectively bred for genetic differences in defensive behaviour which are accompanied by differences in brain and other biological functions. Studies of twins indicate some genetic control of normal human fear from infancy onwards, of anxiety as a symptom and as a syndrome, and of phobic and obsessive-compulsive phenomena. Anxiety disorders are more common among the relatives of affected probands than of controls, especially among female and first-degree relatives; alcoholism and secondary depression may also be over-represented. Familial influences have been found for panic disorder, agoraphobia, and obsessive-compulsive problems. Panic disorder in depressed probands increases the risk to their relatives of phobia as well as of panic disorder, major depression, and alcoholism. The strongest family history of all anxiety disorders is seen in blood-injury phobia; even though it can be successfully treated by exposure, its roots may lie in a genetically determined specific autonomic susceptibility. Some genetic effects can be modified by environmental means.

Defensive patterns vary greatly among species (e.g. rabbits are more timid than humans) and among individuals within species (some people are more timid than others). The range of stimuli which a species fears, and its defensive response patterns to those stimuli, reflect natural selection of genotypes over the generations and cultural transmission among members of that species. If a category of predators drops out, so do the defences of their prey against them. For example on the Galapagos Islands, where predatory mammals are lacking, hawks, marine iguanas and penguins allow themselves to be touched by humans when they are on land, but in the water the iguanas and penguins are threatened by sharks and flee from a swimming man (Eibl-Eibesfeldt, 1970). Decades of contact with man have not greatly increased these species' defensive behaviour, which presumably derives largely from genetic make-up. Where predators have long been present, animals have been selected for their ability to show and learn defensive behaviour and to transmit it to one another.

Individuals within a species vary in their degree of fear of particular stimuli as a function of their individual genetic make-up and the environment in which it is expressed. The product is a phenotype which describes what is, not what could be. As Plomin & Rowe (1979) state, "even if most of the extant phenotypic differences for a particular trait

were determined by genetic differences, that does not mean that the trait is immutable environmentally".

Other sources of variation within individuals are due to age (each species being more fearful at certain ages than at others), to natural selection of particular fear stimuli into a species' repertoire (prepotent stimuli and prepared associations), and to the cultural transmission and individual learning of fear. This review will concentrate on individual variation within humans in genetic predispositions to fear—the raw material on which natural selection acts. Other species are dealt with in more detail elsewhere (Marks, 1986).

Fear and anxiety are not unitary features but assemblies of numerous characteristics that relate variably to one another; they have many different meanings for the behavioural geneticist. In animals, fear and anxiety have been called 'emotionality' (referring to exploratory behaviour or defaecation scores), 'avoidance conditioning', 'freezing', or 'startle'. In humans, the terms have been equated with changes in skin conductance or heart rate, measures of vigilance, self or observer ratings of fear, worry, or autonomic symptoms in the present (state) or over some enduring period (trait), or with criteria for various anxiety syndromes. Whatever the criterion for fear or anxiety, numerous experiments have demonstrated genetic influences on them in animals and humans.

Each species has characteristic patterns of fear behaviour, which vary slightly among individual members of that species. It is not yet known whether genetic control is on a finite number of discrete basic

Adapted from Chapter 6 of *Fears, Phobias and Rituals*, by Isaac Marks. Published by Oxford University Press, New York, 1986

behavioural units of which those action patterns are composed, or is continuous, although the former seems more probable. Whichever the case, there must be "many pathways, each with its own set of detours, from a locus on a strand of DNA to behaviour" (Carey & Gottesman, 1982). The pathways are especially confusing, firstly because many genes are pleiotropic (each gene affects more than one trait), secondly, because variation in any trait is affected by many genes interacting with each other as part of the genotypic system, and thirdly, because the expression of those genes is variable, leading to more than one phenotypic manifestation of any trait. The goal is to link a gene and its biochemical product with its consequences in the ontogeny of the nervous system and resultant neural function and behaviour.

Defensive behaviour develops out of a complex interaction of multiple genes that takes place in the embryonic and subsequent environment. A hint of a strand in that interaction comes from work on the human pro-opiomelanocortin (POMC) gene, which codes for several polypeptide hormones (Whitfield *et al.*, 1982). POMC is processed in the pituitary into adrenocorticotrophic hormone and other hormones that affect defensive behaviour. The pattern of similarity of POMC protein among different species is reflected in the nucleotide sequence of the coding regions of the gene.

Studies in Non-human Species

Protozoa and Invertebrates

Although genetic variation in normal and abnormal fear in mammals is mostly produced by combinations of genes interacting with each other, for the most part in non-additive fashion, some understanding of components in these interactions may be obtained from single-gene analysis of defensive reactions in unicellular organisms and invertebrates (Fuller & Thompson, 1978; Gould, 1982). At least 20 single-gene mutations are known in the pathways mediating avoidance in *E. coli*, and single-gene analysis in *Paramecium* has revealed some of the DNA bases that affect its membrane potential and resultant avoidance behaviour. In the fruitfly *Drosophila*, avoidance learning and habituation are affected by different types of mutation. In a mutant form called *dunce*, twin failures to learn to avoid and to habituate to stimuli occur with a single genetic defect. This results in the lack of one of the two enzymes which metabolise cyclic adenocine monophosphate, postulated by Kandell (1979) as central for habituation in the sea snail *Aplysia*.

Genes can modify escape behaviour by preventing the normal stimulation required during sensitive

phases for the neural substrate of escape to develop. If that stimulation is not experienced at the appropriate developmental period, then the brain will fail to respond to it later, and can thus affect escape. Unusual predominance of one class of stimuli during the sensitive period can permanently destroy or attenuate responses to others; this may result from competition among incoming cells for synaptic space on the target cells in the cortex. A genetic effect of this kind was shown in mutant crickets that lacked sensory hairs on their vibration-sensitive cercus in the tail; such crickets failed to develop normal escape responses. The same defect in defensive behaviour could be produced in normal crickets by non-genetic means, namely by waxing the hairs (Gould, 1982; Bentley, 1975).

Selective breeding of mammals

Most forms of behaviour can be enhanced or reduced over generations by appropriate selective breeding. The number of generations needed to produce a strain with the desired features depends upon their degree of heritability. In these experiments, man replaces nature as the architect by choosing individuals with particular characteristics and mating them together, to the exclusion of other members of the species who show these characteristics less. Animals have thus been bred for tonic immobility (Gallup & Maser, 1977), for emotionality, and for conditioning of active and passive avoidance, escape, and heart rate (Fuller & Thompson, 1978; Plomin *et al.*, 1980).

In well-known breeding experiments, rodents were reared with companions in a cage and then placed, one at a time, in an unaccustomedly wide, brightly-lit arena (open field), upon which the rodent defaecated, urinated, and explored. This behaviour was taken to indicate 'emotionality' or 'reactivity', and high and low scorers can each be selectively bred. Selection soon yields two strains – reactive and non-reactive – and these have been maintained to the 60th generation of selection in rats (Broadhurst, 1975). The two strains differ on numerous measures of emotionality and learning. Non-reactive rats are also more liable to seizures (Gray, 1979), and have more benzodiazepine receptor binding sites in the brain (Robertson *et al.*, 1978). Controlled experiments showed that the differences in defensive behaviour are genetic, and not due to the pre-natal or post-natal environment.

In another experiment, mice were selected for open-field activity (Plomin *et al.*, 1980; DeFries *et al.*, 1978). Over 30 generations, a 30-fold difference developed between the high and low lines, the low-active strain nearly reaching the bottom limit, and

the high line running the distance of a football field during two three-minute test periods. Although the mice were selected for open-field activity, their defaecation rates in the open field also diverged seven-fold. Open-field inactivity and defaecation correlated (0.80), suggesting that they are influenced by many of the same genes. Heritability was moderately low.

Mice have also been selectively bred for active avoidance, scores for shuttlebox avoidance learning in different strains ranging from near zero to 50% or more. Such inbred lines are useful experimentally, as they yield less variable results than does random-bred heterogeneous stock (Plomin *et al.*, 1980). The mouse strains differ not only in learning to avoid, but also in learning to run through a maze to obtain food. Rat strains have been similarly bred to show 14-fold differences in escape-avoidance conditioning (Broadhurst, 1981).

Pointer dogs, too, have been selectively bred into two separate strains, one excessively fearful, the other reasonably stable (Dykman *et al.*, 1965, 1969). By the second specially bred generation, excessively fearful behaviour was shown by 90% of one strain and stable behaviour by 80% of the other; a visitor would cause the timid pointers to hide and freeze, and the others to approach wagging their tails. Timid dogs showed many additional differences in defensive behaviour. Given the association of freezing with bradycardia (Marks, 1986), it is noteworthy that in timid dogs, which froze often, the heart rate was low from birth onwards, averaging 80 bpm and not varying with petting, nor declining much even after extensive handling; in stable dogs it averaged 120 bpm and declined during petting. Hippocampal theta activity (trains of relatively high voltage waves at 4–8 Hz) were absent in alert timid dogs (Lucas *et al.*, 1974), which is interesting given that in normal dogs, hippocampal theta activity reduces with orienting and avoiding, and returns as they habituate to a situation.

Since the pointers' environments were constant, the differences between strains were best accounted for by genetic factors, the mode of inheritance probably being polygenic. Behaviour of the timid and the stable dogs did not differ when they were in their home cages and in the absence of human or novel stimuli. Maturation was important, activity scores and freezing time to noise becoming typical by the age of three or four months.

The development of five breeds of dog and their hybrids was studied over two decades (Scott & Fuller, 1965; Fuller & Thompson, 1978): within breeds, there was substantial genetic variability in defensive behaviour. Such behaviour also differed markedly between breeds, varying with age, and actually being accentuated rather than attenuated by

life experiences. Most of the strain differences in emotional reactions seemed situation-specific, e.g. basenjis were more afraid of strange apparatus than of other dogs. Selection for confidence in one situation did not necessarily affect timidity in another; unwanted timidity occurred in almost every breed, its very universality suggesting that it is necessary for existence.

Other differences in defensive behaviour among various breeds of dogs were noted in passive-defensiveness, this being increased further in all breeds by rearing in a kennel rather than in a home (Krushinskii, 1966). Others found contrasts between dog breeds in persistently raised heart and respiration rate, salivation, high energy metabolism and urinary catecholamines, and vasopressin release (Corson, 1977; Corson & Corson, 1983).

Tonic immobility is another defensive behaviour under genetic influence (Gallup & Maser, 1977). Even after rearing under identical conditions, after only one generation chickens derived from parents that had long reactions remained immobile four times longer than chicks from parents that exhibited brief immobility. Similar strain differences and effects of selective breeding were reported for tonic immobility in rats (McGraw & Klemm, 1973).

Very little research has examined the genetics of fear in primates. In rhesus monkeys there may be some genetic influence, since heart rate deceleration and cortisol response to challenge in the first month of life, which predict fearfulness, separation anxiety and depression over the next three years, were more similar among sibs and half-sibs than among unrelated monkeys, even though they were reared together under similar conditions (Suomi, 1983).

Work in Humans

Normal fear and anxiety

As in animal research, different aspects of human defensive behaviour show considerable specificity. The various tests of fear and anxiety (e.g. the numerous measures of 'neuroticism') are only grouped together to convey a broad view of the many indicators of negative emotional states that have been examined. It is not meant to imply that they denote a unitary dimension, still less that there must be continuity between normal fear and syndromes of morbid anxiety.

Siblings differ from birth onwards in the intensity of their startle response and fears (Valentine, 1930). Over the first year of life, the development of smiling and of fear of strangers was more similar among monozygotic (MZ) than among dizygotic (DZ) twins (Freedman, 1965). At the age of about 22 months, in

interaction with a stranger but not with the mother, the degree of apprehension was more similar among MZ than among DZ twins (Plomin & Rowe, 1979). Slightly older twins, aged about 3.6 years, showed a substantial genetic influence on their emotionality (Plomin & Rowe, 1977). In school-aged twins, with a mean age of 7.6 years, emotionality, tension, and shyness were significantly more similar among MZ and DZ twins (O'Connor *et al.*, 1980). At the age of seven, MZ twins significantly more concordant than DZ twins for separation distress, emotional reactivity, and being fearful and inhibited (Goldsmith & Gottesman, 1981).

An adult twin series was given a neuroticism questionnaire about emotionality, nervousness, and shyness (Shields, 1962). Again, MZ twins were more similar than DZ twins; moreover, MZ twins reared apart were more similar than were DZ twins reared together. Furthermore, in this and in two other studies (Newman *et al.*, 1937; Juel-Nielsen, 1965, cited by Murray & Reveley, 1981), the degree of resemblance between the MZ twins reared apart was close to that between MZ twins reared together. In contrast, however, among 5000 Finnish twins of whom 167 were reared apart, neuroticism had a lower heritability among those reared apart than together; environmental factors explained at least 60% of the variance in the twins reared apart (Koskenvuo *et al.*, 1983).

Other studies, too, found substantial genetic contributions to personality traits related to timidity (McGuffin & Reich, 1984); this was the case for social introversion in normal adolescent twins (Gottesman, 1963) and for neuroticism in adults (Eysenck & Prell, 1951). Another investigation found that genetic factors contributed about half the variance on all dimensions of personality, judged by the responses of 850 pairs of twins to the California Psychological Inventory (Loehlin & Nichols, 1976). Further twin data with normals have found significant heritability for: being highly-strung, easily upset, and 'a worrier', whether measured by the Minnesota Multidimensional Personality Inventory (MMPI), the Eysenck Personality Inventory (EPI) or the Differential Personality Questionnaire; and for specific forms of this anxiety, e.g. rapid changes in mood, constant worrying, etc. (Carey & Gottesman, 1981).

Physiological responses associated with emotionality are also more alike in MZ than in DZ twins, e.g. habituation of skin conductance responses and their number of spontaneous fluctuations, pulse and respiration rate, blood pressure changes, and EEG variables (Carey & Gottesman, 1981; Lader & Marks, 1971).

Four sets of data concerning self-reported fears in normals and their relatives deserve mention. The first three come from twin studies. In one, a fear survey schedule was administered to 91 MZ and 60 DZ twin pairs and to 66 pairs of parents and their twin offspring (Rose *et al.*, 1981); analysis revealed "consistent evidence of heritable variation". In another study (Rose & Ditto, 1983), responses to a fear survey schedule were examined in 354 pairs of like-sex twins aged between 14 and 34. For all fear factors the co-twin's score and the interaction of co-twin's score with pair zygosity significantly predicted a twin's fearfulness, but the power of this prediction varied among different fears, being twice as great for fear of misfortune in a loved one as for fears of dangerous places. Age also affected the contribution of heritability, fear of death being equally similar among MZ and DZ twins during development, but more similar among MZ and DZ twins by the time they reached maturity.

Based on data from the foregoing two studies, two fear factors (social criticism and leadership) were subjected to critical path analysis in order to separate correlated genetic and cultural effects (Neale & Fulker, 1984). This analysis showed that both fear factors had about equal environment and genetic components, with only a small amount of negative genetic-environmental covariance induced by parental phenotypic influences on their offspring.

In a third investigation (Torgersen, 1979), 99 same-sexed twin pairs (11 of the pairs having one twin as an in-patient for neurosis) were interviewed and given a fear questionnaire. MZ twins were more similar than DZ twins for strength and type of fear, except for separation fears (agoraphobia).

The fourth study (Carey & Gottesman, personal communication) was of 151 families (654 individuals) of psychology students, unselected for the presence or absence of psychopathology. All subjects completed a questionnaire about fears and phobias; ten agoraphobia-like cases were selected, and the prevalence of phobias calculated in their first-degree relatives and in the whole study sample. Compared with the whole sample, the relatives of agoraphobia-like probands had significantly more phobias (any phobia, animal phobia, non-animal phobia, multiple phobias, agoraphobia, and obsessions), fears (animals such as snakes, rats, spiders, and bats, and health and social fears) and orderliness. No difference was found for fears of 'normal' animals (dogs, cats, horses, birds, fish) and of physical situation (heights, storms, water and swimming, doctors, hospitals, and enclosed places). There was thus a familial aggregation of many phobias and fears, although this need not be genetic in origin.

Anxiety syndromes

Undifferentiated anxiety disorders and neurosis

Varying diagnostic labels and methods of study over the years and in different countries make it hard to compare investigations and to work out the heritability of each syndrome recognised today. Data will therefore be included concerning not only phobic and obsessive-compulsive disorders, but also panic disorder, generalised anxiety, and neurosis (Carey, 1982; Gottesman, 1963; Carey & Gottesman, 1981; Murray & Reveley, 1981).

Family and family history studies. Although heritability of a problem requires it to run in families, familial prevalence need not imply heritability. Adoption and twin studies are more conclusive on that issue, but only the latter have been made for neuroses: between 1869 and 1948, at least 19 separate reports noted a familial predisposition to anxiety disorders (Cohen *et al*, 1951). Studies of anxiety disorder were reviewed by Carey (1982) (Table I): except for the most recent studies, samples with 'anxiety neurosis' include an unknown number of cases with agoraphobia and with panic disorder without agoraphobia. Most of the earlier reports were of family history studies in which the proband with anxiety neurosis was interviewed about the presence of the disorder in family members. Fewer cases were likely

to be missed by the family studies, in which all relatives were interviewed directly.

About twice as many cases of anxiety neurosis are found among female as among male relatives (Table I). A similar ratio is found in the study by Cloninger *et al* (1981) if one adds to their low prevalence of definite cases included in Table I the questionable anxiety neurotics blindly diagnosed by the research team (19% of females, 8% of males) (Carey, 1982). This female preponderance among relatives reflects that seen for anxiety neurosis in the general population and in general medical practice, but is greater than that in psychiatric practice (Lader & Marks, 1971).

Table I shows that the prevalence of anxiety disorder among first-degree relatives in family history studies is 15–18%, two to three times more than that in two of the three non-twin family studies (except for that of Cloninger *et al*, 1981). Relatives of anxiety neurotics have a far higher prevalence of the disorder than the available controls. The lifetime risk of anxiety neurosis was 6% among the controls of Wheeler *et al*, (1984) – only one-eighth of the prevalence found in the children of their probands, diagnosed as having 'neurocirculatory asthenia' 20 years previously. Among the relatives of the surgical controls of Noyes *et al* (1978), the risk of anxiety neurosis was 3% – merely one-sixth of that among the

TABLE I
Prevalence of anxiety neurosis among relatives of anxiety neurotics¹

Study	Degree of Relationship	Type of Study ²	Prevalence in Relatives: %			
			Males	Females	Total	Controls
Brown (1942)	Second	FH			3	
Pauls <i>et al</i> (1979)	Second	FH	5	14	10	
McInnes (1937)	First	FH			15	
Brown (1942)	First	FH			16	
Cohen <i>et al</i> (1951)	First	FH	12	20	16	
Noyes <i>et al</i> (1978)	First	FH	13	24	18	3
Wheeler <i>et al</i> (1948)	First	FS			49	6
Alanen (1966)	First	FS			41	
Crowe <i>et al</i> (1983) ³	First	FS	17	33	25	2
Cloninger <i>et al</i> (1981)	First	FS	2	13	8	
Slater & Shields (1969)	DZ twins	FS			4	
Torgersen (1979)	DZ twins	FS			9	
Schepank (1976)	DZ twins	FS			17	
Slater & Shields (1969)	MZ twins	FS			41	
Torgersen (1979)	MZ twins	FS			30	
Schepank (1976) ⁴	MZ twins	FS			33	

1 Adapted from Carey (1982)

2 FH = family history; FS = family study

3. Panic disorder only

4. Neurotic symptoms

relatives of their probands with anxiety neurosis. In a subset of their sample, the first degree relatives of 41 probands with panic disorder had an age-corrected risk of anxiety disorder of 25%, compared with 2% in first degree relatives of controls (Crowe *et al*, 1983). In the general population, the prevalence of anxiety disorder is about 4% (Lader & Marks, 1971; Weissman *et al*, 1984).

Of a proband's genes, half are shared with first degree relatives and a quarter with second degree relatives. The pooled risk of anxiety disorder is 15% for first degree and 6% for second degree relatives (Table I). However, the correlations in liability (i.e. the degree to which combined genetic and environmental factors are producing anxiety disorder in the relatives of probands when compared with the population prevalence) based on a population prevalence (K_p) of 0.04 suggest that the data could reflect the common environment shared by first degree relatives (McGuffin, personal communication).

The risk to first degree relatives is greatest when both parents have anxiety disorder and least when neither have it (Table II). Prevalence was also higher in relatives of probands with chronic as opposed to acute anxiety neurosis—22% compared with 5% (Cohen *et al*, 1951). Such findings make polygenic-multifactorial inheritance plausible (McGuffin & Reich, 1984). More than genetic factors may be involved, as most studies find that parents of probands have higher rates of illness than do siblings (McGuffin & Reich, 1984). However, this could also come about because siblings have not lived through as long a period of risk as their parents have.

Are other disorders over-represented in the families of anxiety neurotics? Alcoholism may be more common (Noyes *et al*, 1978; Weissman *et al*, 1984),

and children have a greater risk of major depression or phobia if their parent proband has both major depression and either agoraphobia or panic disorder (Weissman *et al*, 1984). The minor congenital cardiac anomaly of mitral valve prolapse (MVP) is more common among anxiety neurotics than among controls (Crowe *et al*, 1981). However, the familial incidence of panic attacks is no higher among probands with MVP than among controls (Crowe *et al*, 1982). A higher familial incidence of panic attacks in the 12 MVP probands who also had panic attacks than in the 38 without them suggests independent segregation of MVP and of panic attacks.

Twin studies. Four twin studies of neurosis, of variable quality, were reviewed by Fuller & Thompson (1978). The concordance rate was 56% for MZ and 40% for DZ twins, excluding the twins reported by Slater & Shields (1969). Much greater differences between MZ and DZ pairs were found in two more series of twins with anxiety neurosis, in which probands were ascertained more systematically (Slater & Shields, 1953; Torgersen, 1978). Starting with one affected twin, concordance rates were respectively 41% and 30% for MZ twins, but only 4% and 9% for DZ twins (Table I); the differences between MZ and DZ twins were significant in both studies. Using the criterion of marked anxiety rather than a diagnosis of anxiety state, the concordance rate for MZ twins rose from 41% to 65% and for DZ twins from 9% to 13% (Slater & Shields, 1969), suggesting a genetic predisposition to anxiety both as a symptom and as a syndrome.

The DZ twin data yield a much lower risk for anxiety disorder (4–9%) than the other studies (15–49%) of first degree relatives. This may partly reflect differing diagnostic criteria. High rates were found among first degree relatives where probands had typical and severe anxiety (DSM-III type panic disorder) (Crowe *et al*, 1983), and lower rates where probands were unselected (Cloninger *et al*, 1981).

Among 229 same-sex neurotic adult twins who received psychiatric treatment, greater concordance of MZ over DZ twins for neurosis was absent in females and present only in twins who were male or had been psychiatric in-patients (Torgersen, 1983). Greater familial tendencies among males than among females have also been found for phobic fears among same-sexed twins (Torgersen, 1983) and for neuroticism on the Maudsley Personality Inventory (MPI) of neurotic in-patients (Coppin *et al*, 1965).

Such sex differences might be explained by cultural variables such as sex-role stereotypes pressuring women more than men; men may show the disorder only when the genetic loading is high (Reich *et al*, 1972).

TABLE II
Increase in prevalence of anxiety neurosis among siblings of anxiety neurotics with number of affected parents¹

Study	Number of parents with anxiety neurosis		
	Neither	One	Both
McInnes (1937)	8	27	
Cohen <i>et al</i> (1951)	16	22	35
Noyes <i>et al</i> (1978)	9	24	44
Crowe <i>et al</i> (1983) ²	18	42	

1 Adapted from Carey & Gottesman (1981)

2 Panic disorder only

Defined anxiety syndromes

Broad diagnostic labels such as 'neurosis' or 'anxiety' overlap in variable degree with reports of more detailed diagnostic categories, discussed below.

Panic disorder. A pedigree analysis of the relatives of 19 probands with panic disorder suggested that the disorder is familial (Pauls *et al*, 1980). The same was found in a larger study from the same centre, of the relatives of 41 probands with panic disorder, of whom 11 also had agoraphobia as a diagnosis and a further 11 had it as a symptom (Crowe *et al*, 1983); controls were matched by hospital personnel for age and sex. The lifetime morbidity risk of definite plus probable panic disorder for first degree relatives of panic disorder probands was 25% (33% for women, 17% for men), compared with 2% for relatives of controls ($P < 0.001$), corresponding figures for definite panic disorder alone being 17% and 2% respectively. Either definite or probable panic disorder was found in at least one first degree relative in 61% of panic disorder probands, compared with only 10% in control families ($P < 0.001$). Probands' relatives had an excess of generalised anxiety disorder or primary depression, and only a trend to more alcoholism, although secondary depression was increased (16% versus 7%). Of interviewed relatives of panic disorder probands, 55% had received treatment for their anxiety; in 14% this was psychiatric treatment. Crowe *et al* thought that this evidence was compatible with both multifactorial and single gene models.

Relatives of 77 probands who had major depression plus panic disorder were compared with those of probands who had major depression without any anxiety disorder (Leckman *et al*, 1983). Relatives of the former had more than twice the risk of major

depression, panic disorder, phobia, and alcoholism.

Phobic disorders. Most family data have concerned agoraphobia and one type of specific (simple) phobia – that of blood and injury. Very little has been reported about other specific phobias, social phobia, or illness phobia.

Agoraphobia has been examined systematically: a high familial prevalence of anxiety, phobic and depressive disorders and sometimes alcoholism has usually been found (Tables III and IV). An exception was the study of Buglass *et al* (1977); among the relatives of agoraphobic women they found little familial prevalence compared with normal controls, apart from an increase in non-phobic psychiatric disorders among brothers (17%). Most other workers reported increased familial morbidity. A high rate of phobic disorders was found among mothers (31%), along with an increase in other neuroses among both parents (55% of mothers, 24% of fathers) (Solyom *et al*, 1974), and a higher than expected rate of school phobia among the children (14% for ages 11 to 15) (Berg, 1976). More neurosis was noted in the families of agoraphobics (33%) than of temporal lobe epileptics (Harper & Roth, 1962). Increased family histories of depression and of alcoholism were noted among the relatives of agoraphobics, compared with those of social and simple phobics (Munjack & Moss, 1981).

In a family study of agoraphobia, panic disorder, and non-anxious controls (20 probands per group), the morbidity risk for all anxiety disorders among first degree relatives was respectively 32%, 33%, and 15%, the risk for relatives of controls being lower (Harris *et al*, 1983). Risk of anxiety disorder was increased by more than twice among female relatives of ill probands, while male relatives of agoraphobics were also at higher risk for alcoholism. Relatives of agoraphobics were at increased risk not only for

TABLE III
Percentage of illness among first degree relatives by proband group¹

Proband group	Major depression	Diagnosis of relative Anxiety disorders	
		Concurrent with depression	Separate from depression
Normal	6	1	4
Major depression			
No anxiety disorder	10	1	8
With anxiety disorder	18	4	10

¹ Adapted from Weissman *et al* (1984)

TABLE IV
Percentage of illness among first degree relatives by proband group¹

Proband group	All first degree relatives			Diagnosis of relative			Children of probands	
	Alcohol abuse	Major depression	Total anxiety	Major depression	Total anxiety	Any phobia	Separation anxiety	Panic
Normal	8	6 ³	5 ⁴	0		1	0	0
Major depression								
No anxiety	9	11	9	11		0	0	0
With agoraphobia ²	10	12	8	22		11	11	6
With panic	21	20	16	26		12	37	5
With general anxiety disorder	11	20	14	3		0	6	0
Panic alone ³	6	4	25					25

1. Adapted from Weissman *et al* (1984)

2. Any phobia for children of probands

3,4. Crowe *et al* (1983) give 7% and 7%

5. Figures from Crowe *et al* (1983)

agoraphobia, but also for panic disorder and for other phobias, while those of panic disorder only had a raised risk of panic disorder. Major depression and other psychiatric disorders were not increased in the relatives of ill probands. The increased rate of alcoholism in families of agoraphobics accords with the findings above (Munjack & Moss, 1981) and with those in anxiety neurotics (Cohen *et al*, 1951; Noyes *et al*, 1978; Crowe *et al*, 1980).

In another study, the familial risk of agoraphobia was ascertained by questionnaire and interview data from 60 agoraphobic probands (52 women, 8 men) (Moran & Andrews, 1985). Among all parents and siblings, the risk of current or past agoraphobia was 12½%; the risk was twice as great among female relatives. Proband tended to have either a parent or a sibling affected, not both; the risk to parents was not less than that to children, contrary to what would be expected with transmission from a single recessive gene. Direct transmission through three generations, which is usually regarded as a feature of dominant inheritance, was reported in only two families. The overall risk was greater than the estimates of population incidence of agoraphobia in the Epidemiological Catchment Area study in New Haven, St Louis and Baltimore of 4.1%; correlation in liability was on the low side (0.26 to 0.46), depending on the sex of proband and of first-degree relatives.

Why agoraphobia runs in families, and especially among female relatives, remains unclear. The higher risk among female relatives could reflect cultural differences in the mode of expression of agoraphobia. Men might experience similar panic but show less avoidance and be more likely to drink alcohol—as we saw, the risk of alcoholism is raised among the male relatives of agoraphobics, and the female preponderance is distinctly less in anxiety states (panic disorder without agoraphobia). In obsessive-compulsive disorder, too, fewer men have washing rituals in which avoidance of dirt is prominent, but more men have checking rituals in which avoidance is less obvious (Marks, 1986).

The familial prevalence of agoraphobia is unlikely to result solely from modelling within the family (Moran & Andrews, 1985). Many of the agoraphobic probands had never heard of agoraphobia, let alone met a sufferer. Of the probands, 85% came from a family in which neither parent was reported to be agoraphobic.

In another family study of anxiety disorders (Cloninger *et al*, 1981), the greatest familial prevalence of anxiety neurosis was among the kin of six probands who had severe chronic agoraphobia. Among twins with panic disorder or agoraphobia

with panic attacks, anxiety disorders were more than five times as frequent in MZ as in DZ co-twins (Torgersen, 1983). The degree of similarity of the twins' childhood environment did not affect the degree of concordance within the MZ or DZ groups.

Blood-injury phobias have the strongest family history of all the various forms of anxiety disorder (Marks, 1986). Unlike most other phobias, the majority of blood-injury phobics report other family members with a similar problem (Connolly *et al.*, 1976; Marks, 1981; Yule & Fernando, 1980). Among blood-injury phobics, fully 68% had biological relatives who were blood phobic, a rate three to six times higher than the frequency of corresponding phobias among the relatives of agoraphobias or social, dental, or animal phobics (Mark, 1969; Marks & Herst, 1970; Öst *et al.*, 1984; Öst & Hugdahl, 1985). The concordance of blood-injury-illness phobias was higher among MZ than among DZ twins.

This strong family history suggests that blood-injury phobias may originate in a genetically-determined extreme autonomic response. Unlike the tachycardia which is the usual response of most phobics on encountering their phobic stimuli, the cardiovascular response of blood-injury phobics to their phobic stimuli is biphasic, consisting of initial tachycardia followed by profound bradycardia, even to the point of asystole and fainting (Marks, 1986). The degree of bradycardic response of blood-injury may be normally distributed in the general population for genetic reasons (just as are height, intelligence, and many other features that are polygenetically influenced). Presumably only those who have an extreme bradycardic response faint readily, and are thus liable to develop the phobia. This would yield a blood-phobic population with a strong family history, as is found. The bradycardic response is probably present from an early age, as blood-injury phobias start selectively in childhood rather than later (Marks, 1986). It remains to be demonstrated that relatives not only faint frequently, but also have an extreme bradycardic response to blood-injury stimuli.

Perhaps other phobias, too, may partly arise from other autonomic specificities under genetic or environmental influence. For example, when anxious, those with labile blood flow in the cheeks might blush more readily than others, and so be liable to become phobic about blushing; those with undue gastric motility might, when anxious, vomit easily after eating and thus be prone to develop a phobia about that. In addition, blushing may be an autonomic response specifically tied to social cues in the same way that bradycardia with fainting seems linked to blood-injury stimuli, so that blushers

would become phobic of social rather than of other situations.

Cary (1982) investigated 21 twin probands with phobic disorders; of these, seven of the eight MZ co-twins had either a phobic disorder or phobic features, compared with only five of the thirteen DZ co-twins. Several observations argued for a genetic rather than a modelling effect. Firstly, even twins raised apart were concordant. Secondly, in at least two of the concordant MZ pairs the type of phobia present in the one twin was unknown to the other. Thirdly, in one pair of female identical twins, both twins broke down while they were apart in different countries. Fourthly, several pairs were concordant in having agoraphobia-like features, but discordant in the details of the particular stimuli which they feared (e.g. darkness and enclosed places for one, leaving the house alone for the other).

The MMPI on these and obsessive-compulsive twins also suggested genetic effects (Carey & Gottesman, 1981). The mean profile reflected a dysthymic personality, with much tension, obsessive-compulsive worry, feelings of personal inadequacy, and autonomic symptoms of anxiety. This profile suggested someone quiet, seclusive, conventional, anergic, sensitive, and socially submissive. Profiles from MZ co-twins paralleled those for probands, but were less extreme. A similar but less marked profile was seen in DZ co-twins, but lacked a peak on the psychasthenia scale.

Further evidence of the heritability of phobias comes from the MMPI of 26 twins, reared apart on average from the age of four months to 24 years. The intra-class correlations were 0.50 for phobias and, on clinical scales, 0.52 for psychasthenia and 0.56 for A (Welsh) (Gottesman *et al.*, 1982). (An intra-class correlation compares variability within twin pairs with that between twin pairs; implications of genetic effects are strong with values of 0.6 to 1.0, and moderate to weak with values of 0.3 or less).

Obsessive-compulsive disorders. (Murray & Reveley, 1981; McGuffin & Reich, 1984).

Excluding the atypical report of Lewis (1935), some psychiatric abnormality was found in a high proportion of relatives: 40 to 50% of parents and 19 to 39% of siblings (Table V), and 16% of children (Carey, 1978). Obsessive-compulsive illness was found in 2 to 10% of parents and sibs (Table IV), and in a lower proportion (but still eight times more than in the general population) by Rosenberg (1967). Responses of 404 normal twin pairs to the Leyton Obsessional Inventory indicated that the hereditary proportion of the variance was 44% for obsessive traits and 47% for obsessive symptoms (Murray *et al.*, 1981).

TABLE V
Percentage of disorder in first-degree relatives of obsessive-compulsive patients¹

Study	Obsessive-compulsive disorder		Obsessive-compulsive personality		Any noteworthy psychiatric condition	
	Parents	Sibs	Parents	Sibs	Parents	Sibs
Lewis (1935)	37	20	18	10		76
Brown (1942)	7	7	33	20	50	36
Rudin (1953)	5	2	3	3	40	19
Kringlen (1965)		10				
Carey (1978)		5	14		48	39

1. From McGuffin & Reich (1984)

Among obsessive twins, Carey (1978) found reports of 30 concordant and 13 discordant MZ pairs, and of no concordant but 14 discordant DZ pairs; this could reflect a tendency to report concordant pairs more often. Interesting discordance was found in identical triplets who all became psychotic, but only one of whom developed obsessive-compulsive disorder as well (subsequent to the report) (McGuffin *et al*, 1982). The largest twin series (15 MZ, 15 DZ) is that systematically investigated by Carey (1982). Obsessive symptoms or features were found in 87% of MZ and 47% of DZ twin pairs.

Discussion

Genotypic variation contributes to differences in defensive patterns among individuals within a species. Genetic influences on a trait need not mean that it cannot be altered by environmental effects: its phenotypic expression depends on the surroundings in which the individual develops. Potential paths from genes to defensive behaviour are hinted at by single-gene analysis of avoidance and escape behaviour in invertebrates; how genes interact together and with environmental factors to result in defensive responses in vertebrates is still unknown.

Animals have been selectively bred for many types of defensive behaviour. Genetic differences in defensive behaviour have been found to be accompanied by differences in the number of benzodiazepine receptors in the brain, hippocampal theta activity, in heart, respiration and salivation rates, and in energy metabolism, cortisol response to challenge, urinary catecholamines, and vasopressin release.

Normal human fear is partly under genetic control from infancy onwards. Compared with fraternal (DZ) twins, identical (MZ) twins are more similar as infants in their fear of strangers, and as children

and adults on measures of emotionality, emotional lability, neuroticism, shyness, social introversion, worry, tension and fears, and on physiological variables such as pulse and respiration rate, blood pressure, EEG, and skin conductance.

Among the relatives of probands with anxiety disorders, such problems are far more common than in control families. Twice as many cases are present among female than among male relatives, and among first than among second-degree relatives. The more parents that have anxiety disorder, the greater the risk to first-degree relatives. Alcoholism and secondary depression may also be over-represented among relatives. Twin studies also suggest a genetic predisposition to anxiety both as a symptom and as a syndrome; this may be less evident in women than in men, as sex-role stereotyping may lead women to display fear and anxiety more readily.

Using more refined diagnostic categories, the highest familial incidence of all is in blood-injury phobia, which may originate in a genetically influenced autonomic vulnerability from childhood onwards to have bradycardia and faint at blood-injury stimuli. Familial and genetic influences are also evident in panic disorder, agoraphobia, and phobic disorders as a whole. The presence of panic disorder in depressed probands increases the risk to their relatives of major depression, panic disorder, phobia, and alcoholism; MZ twins are more concordant than DZ twins for phobias and on MMPI profile. Internal evidence from agoraphobia and from phobias as a whole argues for a genetic rather than a direct modelling effect, although other cultural factors presumably interact with genetic ones. Obsessive-compulsive problems are far more common among the relatives of obsessive-compulsive probands than in the general population, and more concordant MZ than DZ twin pairs have been reported.

Conclusion

There is some evidence for genetic effects on normal fear and anxiety, on anxiety disorders as a whole, and on panic and obsessive-compulsive disorders, agoraphobia, and blood-injury phobia. Apart from the possible origin of blood-injury phobia in a specific autonomic susceptibility, little is known about how these genetic factors might operate, about potential biological markers for them, or about how they might be modified by environmental means.

Exposure therapy produces rapid and lasting improvement in blood-injury and other phobias and in compulsive ritualisers, even when they have a strong family history; whether this fact can lead to useful preventive principles in bringing up genetically vulnerable individuals is worth exploring.

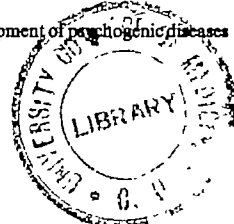
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The Continuum of Psychosis and its Implication for the Structure of the Gene

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Three observations challenge Kraepelin's binary view of the functional psychoses: a bimodal distribution of the clinical features of manic-depressive illness and schizophrenia has not been demonstrated; affective illness appears to predispose to schizophrenia in later generations; and 'schizoaffective' illnesses cannot be separated in family studies from either of the prototypical psychoses. The alternative concept is that psychosis is a continuum extending from unipolar, through bipolar affective illness and schizoaffective psychosis, to typical schizophrenia, with increasing degrees of defect. According to this concept the genes predisposing to psychosis have a degree of stability that ensures that the form of the psychosis tends to remain the same within families, but there is also the possibility of change, implying that the genetic mechanisms themselves are variable. It is proposed that quantal changes in the 'virogene' are due to replications within the genome (e.g. the generation of tandem repeats of the element or a component of it); that such replications occur at a critical stage (e.g. gametogenesis, fertilisation, very early embryogenesis) in the course of reproduction; and that the 'season of birth effect' reflects the operation of the mechanism responsible for these replications.

Contemporary classifications of psychiatric disease owe much to Kraepelin (1899). At the end of the last century he distinguished the organic from the functional psychoses¹ and, in the latter category, separated manic-depressive insanity from dementia praecox (schizophrenia). The separation was achieved on the basis of outcome—an episodic course with recovery from individual episodes was regarded as characteristic of manic-depressive illness, while in dementia praecox an element of persistence and even progression was to be expected. Although exceptions are recognised, this generalisation on outcome has survived relatively unchallenged.

Kraepelin's 'binary' view has dominated subsequent work to the extent that the entities of schizophrenia and manic-depressive illness are widely regarded as separate diseases, each with a different genetic background, treatment and outcome (Gershon & Rieder, 1980; Loranger, 1981). The earlier view (Guislain, 1833; Neumann, 1859; Griesinger, 1861) that there is a unity in the psychoses (the 'einheit-psychose') has yielded to common observation that typical cases of manic-depressive illness and schizophrenia have distinct psychopathological features, usually follow their predicted courses, and are associated with an incidence of similar illnesses in first degree relatives.

This paper presents an alternative to Kraepelin's

binary concept. Doubts about the binary hypothesis arise firstly from the failure of Kendell and co-workers (Kendell & Gourlay, 1970; Kendell & Brockington, 1980) to achieve a bimodal separation of the two conditions on the basis of symptoms or outcome. Secondly, the similarity of season of birth and season of onset effects suggests that the psychoses share aetiological determinants. Thirdly, in family studies two findings indicate that the conditions are more closely related genetically than is generally believed: several investigations have shown an excess of children with schizophrenia among the offspring of parents with affective disorder, and studies of 'schizoaffective psychosis' have failed to separate this entity from either affective disorder or schizophrenia.

The alternative concept is that there is a continuum of psychosis extending from pure affective disorder to schizophrenia with the defect state. The existence of such a continuum would imply that the genetic mechanisms postulated to underlie it are subject to more rapid change than is associated with most human genes.

Limitations of the binary concept

A persistent problem for the binary view has been the undoubted occurrence of psychoses with both manic-

depressive and schizophrenic symptoms. Kasanin (1933) introduced the concept of 'schizoaffective psychosis' and initiated discussion as to whether such illnesses are variants of one or other of the prototypes, or constitute a 'third psychosis'.

It is generally assumed that 'true' schizoaffective psychoses, which cannot be placed in either category, are relatively rare, and that the majority of psychotic illnesses can be classified as either affective or schizophrenic. It would thus be expected that a discriminant function based on the characteristic features would separate the two conditions with relatively few intermediate cases. In testing this prediction on data from the US/UK Diagnostic Project, collected using the Present State Examination and a semi-structured history schedule, Kendell & Gourlay (1970) on the contrary found a distribution with a maximum of cases at the mid-point between the two typical pictures (Fig. 1). A discriminant function which

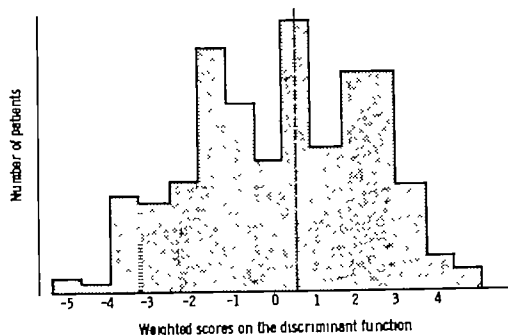


FIG. 1 The distribution of the weighted scores of 146 patients with schizophrenia and 146 patients with affective disorder from the US-UK study on the discriminant function derived by Kendell & Gourlay to maximise separation of the conditions (redrawn with permission from Kendell & Gourlay, 1970)

included all those items which best distinguished patients with a project diagnosis of affective disorder from those with a diagnosis of schizophrenia did not separate two groups at either end of the distribution.

In further sample ($n=217$) Kendell & Gourlay again failed to demonstrate bimodality—this time the distribution on the discriminant function was uni-modal rather than tri-modal. Such findings cast doubt on the common clinical assumption that most cases of psychosis can be relatively easily classified as either affective or schizophrenic.

Returning to the attack, Kendell & Brockington (1980) developed a method for ascertaining a non-linear relationship between symptomatology and

outcome, but were unable to demonstrate such relationships in samples of 127 unselected psychotic and 105 schizoaffective patients. They wrote: "no firm conclusions can be drawn about the relationship between schizophrenic and affective psychoses, though it has to be noted that yet another attempt to demonstrate discontinuity between them has failed."

The diagnostic boundaries of schizophrenia have for long been debated. Nuclear symptoms (e.g. thoughts experienced as alien or as auditory hallucinations) were advanced by Schneider (1957) as pathognomonic, but they define a restricted range of illnesses, and are not directly related to poor outcome (Bland & Orn, 1980). An alternative strategy adopted in recent research criteria (e.g. the DSM-III criteria (American Psychiatric Association, 1980), and the earlier criteria of Feighner *et al.*, 1972) is to include duration as a defining feature. Such criteria are successful in predicting outcome. Outcome for the schizoaffective psychoses, whether studied before (Hunt & Appel, 1936; Clark & Mallett, 1963) or after (Croughan *et al.*, 1974; Brockington *et al.*, 1980) the introduction of operational criteria, is found to be intermediate between schizophrenia and affective disorder. Thus, although bimodality of outcome has been claimed in one study (Cloninger *et al.*, 1985), experience with schizoaffective illness does not appear to support it.

The two prototypical psychoses share some salient features. Both show a tendency to recur, more striking in the case of manic-depressive psychosis but present also in schizophrenia. Onset before puberty is rare in either case, but the disease can occur at any point in adult life, the mean onset of schizophrenia being earlier (by a decade or more) than affective illness. The lifetime prevalence of both conditions approaches 1%. In neither case have characteristic histopathological changes in the brain been established, although recent neuroradiological (Johnstone *et al.*, 1976) and post-mortem studies (Brown *et al.*, 1986) suggest structural changes (e.g. cerebral ventricular enlargement) which are more marked in schizophrenia. Seasonality of onset and of date of birth have been widely discussed as a lead to the aetiology of schizophrenia; such illnesses are more likely (by about 5–10%) to have an onset in the early summer months than at other times of the year, and patients with these illnesses are more likely to have been born in the winter months (Dalen, 1975). These trends follow the seasons in the north and south hemispheres. Similar effects have been found for manic-depressive illness (Hare & Walter, 1978) (Fig. 2).

Two explanations of the similarity of these findings in affective and schizophrenic psychoses suggest

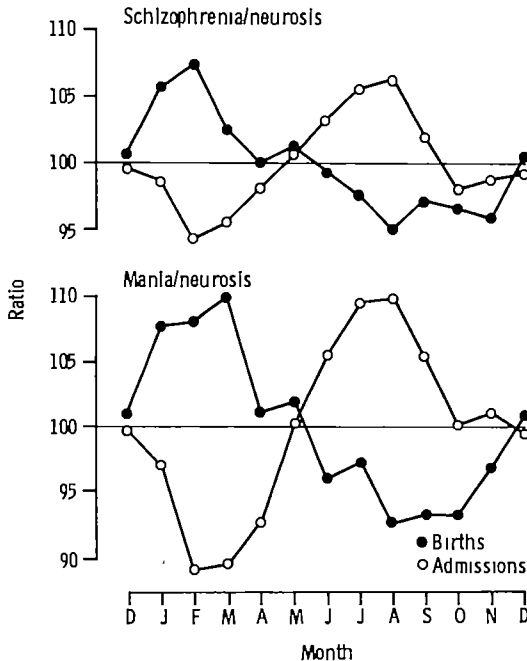


FIG. 2 Monthly births and admissions for schizophrenia and mania expressed as a percentage of the number expected on the basis of overall numbers of births and admissions for neurosis: points represent three-monthly moving averages (redrawn with permission from Hare and Walter, 1978)

themselves: that a statistical artefact has an influence on both sets of data, or that the conditions share a fundamental relationship to the seasons. Such artefacts as have been suggested have been effectively dismissed (Watson *et al*, 1982; Pulver *et al*, 1983), and

therefore the second possibility must be seriously entertained.

The prophylactic value of medication is established in both conditions—lithium in preventing relapse in affective illness (Davis, 1976), and neuroleptic medication in schizophrenia (Davis, 1975). Although such drug specificity suggests differences in the underlying disease processes, some schizophrenic illnesses respond to lithium (Biederman *et al*, 1979; Delva & Letemendia, 1986), and in addition to their efficacy in mania, neuroleptics may be of value in depression (Hollister *et al*, 1967). Thus no unequivocal demarcation of the functional psychoses can be made on the basis of symptoms, outcome or response to treatment, and the seasonality effects suggest an underlying aetiological unity.

Are schizophrenia and affective psychosis genetically related?

It is widely held that the genetic factors which predispose to the two typical psychoses are unrelated. Thus according to Gershon & Rieder (1980), "evidence from twin and family studies suggests that bipolar manic depressive illness and chronic schizophrenia are distinct entities", and according to Reich *et al* (1982) "the genetic diathesis for affective disorders is independent of that for other psychiatric disorders". Several findings suggest that such conclusions are too dogmatic. Rather, the two conditions appear to be related in a way which reveals something about the genetic mechanisms. Specifically, affective illness in one generation may predispose to schizophrenia in the next.

In a survey of the risk of other psychiatric illness in first-degree relatives of patients with affective disorder, Rosenthal (1970) noted an excess of schizophrenia in children (a mean of 2.3% in 5 studies),

TABLE I
Psychotic offspring of psychotic parents classified by diagnosis

Offspring	Parents	
<i>Penrose (1968)</i>	<i>Affective disorder</i>	<i>Schizophrenia</i>
Affective disorder	232	34
Schizophrenia	205	150
<i>Powell et al (1973)</i>	<i>Manic-depressive psychosis</i>	<i>Schizophrenia</i>
Manic-depressive psychosis	10	0
Schizophrenia	15	9

while the risk in parents and siblings remained within the 0.8% lifetime prevalence for the general population. He asked: "From the genetic point of view, why should schizophrenia have occurred at all in these families?"

Two studies have examined parent-child pairs in which both members suffered from psychosis (Table I). Penrose (1968) identified 621 such pairs in a series of 5456 pairs of relatives with psychiatric disease collected over a period of 18 years in the Ontario Mental Hospitals. Among the ill children of schizophrenic parents a diagnosis of schizophrenia preponderated over affective disorder in a ratio of almost 5:1. Among the children of affectively ill parents the ratio was a little less than 1; schizophrenia was almost as common as affective disorder. In a similar study on the Aberdeen case register Powell *et al* (1973) found schizophrenia in the children of schizophrenic parents and affective illness among the children of parents with affective disorder as expected. However, among the latter they found that for 10 cases of affective disorder, 15 cases of schizophrenia were also present.

These findings suggest an excess of schizophrenia among the children of affectively ill parents²; this seems unlikely to be due to a secular trend toward diagnosing more schizophrenia, or to selective factors (e.g. hospital admission) leading to over-inclusion of patients with schizophrenia, as the two samples were

collected 30 years apart on either side of the Atlantic, one from in-patient records and the other from a case contact register.

In a series of 25 children of two manic-depressive parents, Schulz (1940) reported that 7 suffered from affective illness but 3 suffered from schizophrenia. In applying more restrictive diagnostic criteria to a larger literature, Elsasser (1952) found that of 169 children of two parents with manic-depressive or atypical psychoses (i.e. with schizophrenia excluded), 18 suffered from definite affective illness and 6 from definite schizophrenia.

Pollock & Malzberg (1940) collected family histories of psychosis over three generations. In relatives in preceding generations (parents, grandparents, uncles and aunts) of patients with schizophrenia they found an excess of affective illness: 15 cases of affective illness compared with 11 of schizophrenia (Table II).

Slater (1953) reported similar findings. In his study of twins he recorded psychiatric illness in other relatives, and found the ratio of affective disorder to schizophrenia (4:3) particularly high in the parents of patients with schizophrenia. The corresponding ratio (3:5) for siblings similarly showed more affective disorder than would conventionally be expected (Table II). No excess of schizophrenia was found in the parents or siblings of patients with affective disorder.

TABLE II
Psychotic illness in the preceding generations and siblings of patients with affective disorder or schizophrenia

Relative group	Proband diagnosis	
	Affective disorder	Schizophrenia
<i>Pollock & Malzberg (1940)</i>	(n = 155)	(n = 175)
Preceding generations (parents, uncles, aunts, grandparents)		
Manic-depressive psychosis	7	15
Schizophrenia	3	11
Siblings		
Manic-depressive psychosis	11	2
Schizophrenia	3	8
<i>Slater (1953)</i>	(n = 38)	(n = 156)
Parents		
Affective disorder	9	16
Schizophrenia	0	12
Siblings		
Affective disorder	12	15
Schizophrenia	1	26

TABLE III
Risk of psychosis in the first-degree relatives of probands with schizoaffective illness. percentages of relatives affected

Type of psychosis	Angst et al, 1979 (n=150)	Tsuang et al, 1977 (n=52)	Baron et al, 1982 ¹ (n=50)	
			SA-A	SA-S
Schizophrenia	5.26	0.9	0	4.1
Schizoaffective psychosis	2.97	—	3.2	1.4
Affective psychosis	6.70	11.8	28.1	10.9

1 Schizoaffective illness was subdivided into mainly affective (SA-A) and mainly schizophrenic (SA-S) subtypes

In an earlier study of manic-depressive illness (Slater, 1936) "a surprising feature had been the number of schizophrenics among the children . . . In 10 of the 15 cases where manic-depressive subjects had been found by Dr Slater to have schizophrenic children he had been unable to find schizophrenia in other members of the patient's family or that of the husband or wife".

Each of these observations, therefore, is consistent with a one-way movement between generations from affective disorder to schizophrenia. On the basis of his own family studies Myerson (1925) summarised what he believed to be the inheritance of mental disease: "that the manic-melancholic diseases are in the main followed by manic-melancholic diseases, but in a certain number, especially of doubtful cases by dementia praecox . . . that the manic-depressive states of involution trend toward manic-depressive and dementia praecox, especially the latter . . . that dementia praecox in an ancestor trends toward dementia praecox in the descendants with a certain scattering incidence of imbecility."

Such a notion is an echo of the concept of the degeneration psychosis as formulated by Morel (1860) and Magnan (1893). As a genetic theory it is unorthodox, but on the accumulated literature looked at from an intergenerational standpoint it appears it cannot be easily dismissed.

The genetics of schizoaffective disorder

On account of the historical origins of the concept of schizoaffective psychosis and its pivotal position in nosology, particular interest attaches to the genetics involved.

In the largest study to date, Angst *et al* (1979) found the risk of schizophrenia and affective disorder to be approximately equal in first degree relatives of schizoaffective probands, and that of schizoaffective

illness to be appreciably less than that of either of the prototypical psychoses (Table III). Two other surveys (Tsuang *et al*, 1977; Baron *et al*, 1982) found schizoaffective disorder to be more closely related to affective illness than to schizophrenia. Each group of researchers inferred that schizoaffective illnesses are not genetically separate from the major (particularly affective) psychoses. The conclusion that schizoaffective illness is not a genetic entity was also reached by Tsuang (1979) in a study of pairs of siblings with psychosis. When diagnoses were blindly allocated to the categories of schizophrenia, affective disorder, and schizoaffective disorder, a deficit of schizoaffective $\times$ schizoaffective pairs was observed relative to the number of such diagnoses in the sample. Eight schizoaffective $\times$ affective, five schizoaffective $\times$ schizophrenic, and two schizophrenic $\times$ affective pairs were present in a sample of 35 pairs.

The relationship of schizoaffective illness to the affective disorders is illuminated by Gershon *et al*'s (1982) study with modified Research Diagnostic Criteria of 1254 relatives of patients with major affective disorder: "These data were compatible with the different affective disorders representing thresholds on a continuum of underlying multifactorial vulnerability. In this model schizoaffective illness represents greatest vulnerability, followed by bipolar . . . then unipolar (affective) illness". These authors thus espouse a continuum concept which extends up to schizoaffective illness but excludes schizophrenia.³

If schizoaffective disorder does not breed true and has a relationship (as suggested by Tsuang *et al* (1977), Baron *et al* (1982) and Gershon *et al* (1982)) to affective disorder, the question arises whether an excess of schizoaffective illness (or affective disorder) is seen amongst first degree relatives of patients with schizophrenia when schizophrenia is narrowly defined as in recent operational criteria. Kendler *et al*

TABLE IV

Morbid risk of schizophrenia, affective disorder, schizoaffective disorder and atypical psychosis (determined by DSM-III criteria) in blindly assessed first degree relatives of patients with schizophrenia and surgical controls (from Kendler et al, 1985)

Diagnosis	Relatives affected. %		P
	Schizophrenics (n=332)	Controls (n=318)	
Schizophrenia	3.7	0.2	7.9×10^{-8}
Schizoaffective disorder	1.4	0.1	0.008
Atypical psychosis	2.5	0.3	0.0006
Affective disorder			
Unipolar	6.0	7.6	NS
Bipolar	1.2	0.3	NS

(1985) reanalysed a consecutive series of 510 patients with a chart diagnosis of schizophrenia from the Iowa Hospitals with the DSM-III criteria, and compared rates of illness in the relatives of 332 patients with a DSM-III diagnosis of schizophrenia with those in relatives of 318 surgical controls. In their analysis of personal interview or hospital records these authors found that while the risk of affective disorder was not significantly increased, the risk of schizoaffective disorder and atypical psychosis was (Table IV). Even with a restrictive definition of schizophrenia, therefore, no 'genetically pure' syndrome is isolated, and a relationship with the schizoaffective spectrum remains.⁴

Thus it seems that schizoaffective illness occurs in the relatives of patients with both schizophrenia and affective disorder even when these illnesses are restrictively defined. Unless schizoaffective disorder can be partitioned into schizophrenic and affective types of illness (and since the concept originated in the failure of such an enterprise, this seems unlikely), parsimony requires the conclusion that schizoaffective disorder is but the bridge between affective disorders and schizophrenia.⁵ The psychoses constitute a genetic continuum rather than two unrelated diatheses (Crow & Cooper, 1986).

The genetics of a continuum

However, there is also a degree of stability of clinical picture within families; without this, the Kraepelinian dichotomy could hardly have survived.⁶ Within families (perhaps particularly within generations) the form of the psychosis tends to breed true. Such a trend is accentuated when (as often in genetic studies) cases which do not fit the two classical pictures

are excluded from consideration. A more faithful representation is obtained when the series of cases is unselected and intermediate diagnoses of probands and relatives are allowed. Thus in his series of consecutive admissions with psychosis Ødegaard (1972) included schizophrenic psychoses of three grades of defect, and the categories of schizophrenia without defect, reactive psychosis and atypical affective psychosis cover most of what would be described as schizoaffective illness in other classifications. The incidence and type of illness in relatives (Fig. 3) is

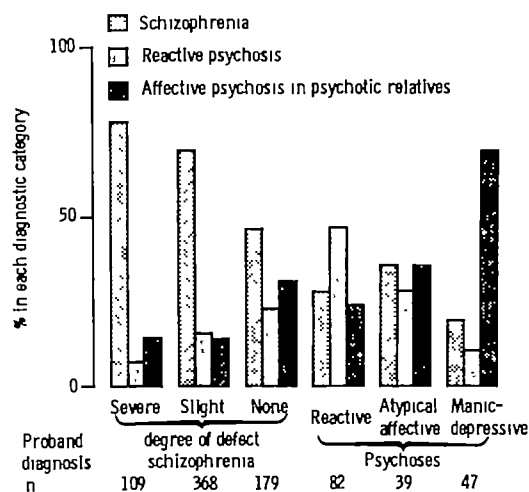


FIG. 3 Relative risks of psychiatric disorder, classified from case histories, in the first and second degree relatives of a consecutive series of admissions of patients with psychosis (data taken from Ødegaard, 1972)

compatible with the concept of a genetic continuum along which conditions are distributed with increasing severity from typical manic-depressive psychosis to schizophrenia with severe defect. Within this continuum particular forms of illness are genetically related to those nearby. The inter-relatedness of the two ends of the continuum, however, is limited.

The hypothesis of a continuum of psychosis is a development of the concept of the 'unitary psychosis' espoused in the last century by Guislain, Neumann, and Griesinger and more recently by others (Llopis, 1954; Menninger *et al*, 1958; Karlsson, 1974; Renner, 1982; Flor-Henry, 1983). The refinement is that the psychoses are represented on a continuum (Fig. 4) from pure affective illness to deteriorating schizophrenia.⁷

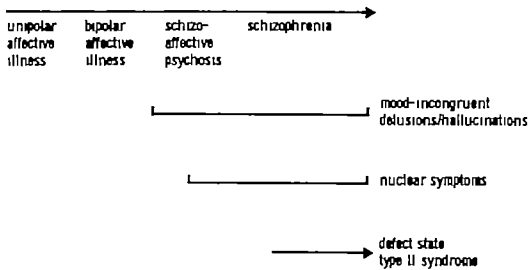


FIG. 4 The continuum concept. Brackets indicate the range over which mood-incongruent psychotic phenomena and nuclear symptoms may occur

That movement along this continuum occurs is documented by Ødegaard's study and the studies on schizoaffective disorder; that such movements take place in the course of reproduction is suggested by the systematic trend in the intergenerational studies. For the children of parents with one type of psychosis the risk of an illness of greater severity is increased. These phenomena cast some light on the nature of the underlying genetic mechanisms. They intimate that in the psychoses we observe the manifestations of quantum changes in a variable gene. One possibility is that at some critical stage of the reproductive process (e.g. meiosis, fertilisation) tandem repeats of a mobile genetic element (or of some component of that element) are generated.

The nature of the psychotic continuum

The persistence of the psychoses in spite of a fertility disadvantage, well documented in the case of

schizophrenia but possibly also associated with manic-depressive psychosis (Stevens, 1969), raises the question of whether the gene confers advantages on relatives of patients with psychotic illness. In Iceland, Karlsson (1984) noted that such relatives were over-represented, as recorded in the Icelandic *Who's Who*, in public life and the professions. Karlsson (1978) also drew attention to the frequency of a history of mental illness in compendia of those distinguished in literature, mathematics and philosophy. Others (Andreasen & Powers, 1975; Jamison, 1986) have documented the prevalence of significant affective disturbance in writers and poets. The nature of the relationship between the two typical psychoses and achievement is illuminated by Noreik & Ødegaard's (1966) study of Norwegians with a background of higher education. Among high school graduates they found admissions increased with respect to general population expectation for a diagnosis of manic-depressive psychosis in both sexes and for schizophrenia in females. In studying admission rates in relation to occupation, they found that the group comprising the liberal professions, teaching and higher officials had first admission rates for manic-depressive psychosis twice those of the general population (151, compared with 73), but admission rates for schizophrenia were reduced (305, compared with 357 for the general population).

Such associations suggest that the gene has beneficial as well as adverse consequences, and that the balance between them is seen only when the psychoses are considered together. However, it remains to be demonstrated that these effects are related to gene survival. There appear to be two possibilities—that the gene or a component element promotes fertility in (as yet unidentified) relatives of psychotic individuals, or that the element is able to replicate between haploid genomes, as suggested for mobile genetic elements by Hickey (1982), and thus to persist against a fertility disadvantage.

A clue to the nature and functional significance of the variable underlying psychosis is Flor-Henry's (1969) observation that the psychoses associated with temporal lobe epilepsy are commonly schizophrenic when the lesion is on the left side and affective when it is on the right side of the brain. Recent (and some earlier) studies of brain structure support the view that schizophrenia itself is a disease with a degree of selectivity for the left hemisphere (Crow, 1986). Such observations raise the possibility that the genetic determinants of brain lateralisation and psychosis are related.

Asymmetries in the human brain are now well established (Galaburda *et al*, 1978): structures in the superior temporal lobe, including the auditory

association cortex, are better developed on the left than on the right side of the brain (at least in right-handed individuals). These asymmetries are a relatively recent evolutionary development being shared by man with the gorilla (Groves & Humphrey, 1973) the orang utan (Le May & Geschwind, 1975) and the chimpanzee, but not the rhesus monkey (Yeni-Komshian & Benson, 1976). Presumably they are related to the cerebral mechanisms of communication and speech. In man the asymmetries are associated with cerebral dominance and handedness, a genetically determined attribute whose persistence as a polymorphism is as yet unexplained.

Since differential brain growth occurs, one must suppose that the genes involved code for one or more growth factors. These genes, e.g. the 'cerebral dominance gene' or 'right shift factor' (Annett, 1978, 1985), could be the location of the genetic changes underlying psychotic illness. One possibility is that a viral gene (e.g. a retrovirus or other type of mobile genetic element or 'transposon') is integrated at this site (Crow, 1984). If the presence of such a gene were associated with increased growth but the gene was sometimes expressed as viral particles this could explain the postulated benefits as well as the disruptive cerebral effects, and possibly the episodic nature of the illness.

Conclusions

It is concluded that the affective psychoses and schizophrenia are related to each other on a continuum, and that this continuum has a genetic basis. This concept is founded in the failure of Kendell & Goulley's attempts to separate the conditions by symptomatology and in the similarities in season of birth and season of onset effects for the two prototype disorders. It is sustained by observations that schizoaffective psychoses (states defined as intermediate between the major psychoses) do not breed true and cannot be excluded from the relatives of patients with affective disorder or schizophrenia when these are defined by stringent criteria. Therefore, by an inductive (in a mathematical sense) argument as well as from the evidence of studies of symptomatology and outcome, there is a case for a continuum of psychosis which increases in severity from unipolar through bipolar affective disorder to schizoaffective disorders and schizophrenia with increasing degrees of defect. The existence of such a continuum and the excess of individuals with schizophrenia among the descendants of patients with affective disorder provide clues to the nature of the genetic mechanisms. The genes responsible appear to

be subject to quantum changes. Perhaps these genes survive because they are closely related to growth factors responsible for the development of cerebral asymmetry and the mechanisms of dominance. Such relatively late evolutionary developments may be associated with a high degree of genetic variation.

Notes

1. According to G. E. Berrios (in preparation), the concept of functional psychosis is unlikely to have originated with Kraepelin, although it was adopted by him. It may have been introduced by E. Mendel.
2. Cammer (1970) also commented on the incidence of schizophrenia in the children of manic-depressive parents. In a personal case record study he judged that 148 (26.6%) of 533 children of 273 manic-depressive parents were "schizophrenics", but the diagnostic criteria adopted are broad and the age structure and hospitalisation histories of this series were not given.
3. It is of interest that in Gershon *et al.*'s (1982) investigation there was an excess ($P < 0.001$) of schizophrenia among the relatives of patients with schizoaffective illnesses in comparison with those having other types of affective illness. Also of note with respect to inter-generational effects is the finding that the risk of schizoaffective illness in the siblings of the generation succeeding the proband is increased (at 3.9% age-corrected lifetime risk) in comparison with siblings of the proband (0.8%) and siblings of members of the generation preceding the proband (0%).
4. It is also of interest that an earlier study (Tsuang *et al.*, 1980) of the Iowa series, which did not allow schizoaffective diagnoses, supported the distinction between schizophrenia and affective disorder "although the distinction between schizophrenia and mania was not clear-cut". Subtyping of paranoid and non-paranoid schizophrenia and of unipolar and bipolar affective disorder on the basis of familial associations was not supported. These conclusions do not appear to rule out a continuum concept.
5. A number of studies have related outcome in schizophrenia to incidence of atypical or affective psychoses in relatives. Thus Kant (1942) studied family histories of 50 deteriorated and 50 recovered schizophrenic patients and found that while overall rates of psychosis were similar in the two groups, the ratio of schizophrenia to affective disorder was 5:1 in the former and 1.5 in the latter. However, he notes that "none of the manic-depressive patients among the relatives of the recovered group actually belong to the manic-depressive nucleus... there are several whose clinical pictures remind one strongly of the corresponding atypical types in the recovered schizophrenic group". With a parallel strategy, Kendler & Hays (1983) studied patients with schizophrenia diagnosed by DSM-III criteria, and identified a group of 18 with a first-degree relative with unipolar affective disorder and 10 with a relative with bipolar affective disorder, whom they compared with a group of 98 with no affective disorder in first degree relatives. These authors commented on their own and some other authors' apparently high rates of affective disorder in relatives as "probably representative of most families of schizophrenics". Patients with a family history of affective disorder were more likely to have suffered from affective symptoms on follow-up. Kendler & Hays concluded that "even when DSM-III criteria are met, hesitation is indicated in diagnosing schizophrenia in patients with a first degree relative with bipolar illness". Similarly, Pope & Lupinski (1978) review 15 studies of "good prognosis", "remitting", and "recovered" schizophrenia and "atypical", "schizophreniform" and "schizoaffective" psychoses and draw attention to the high frequencies of affective illness in the relatives. They warn that "over-reliance on (clinical) symptoms alone results in over-diagnosis of schizophrenia and under-diagnosis

of affective disorder, particularly mania. This compromises both clinical treatment and research".

Underlying the views of Kendler & Hays and Pope & Lipinski is the conviction that the binary concept is correct, but that the location of the borderline has strayed from its position of Kraepelinian rectitude, if this borderline were replaced, symptoms and outcome would separate in the prescribed bimodal manner. The work of Kendell & Gourlay (1970) and Kendell and Brockington (1980) gives no support to this notion. Moreover, in the recent literature the border has moved far in one direction and then far in the other without finding a satisfactory resting place. It is clear from the work of Kendler & Hays (1983) and Kendler *et al.* (1985) that the DSM-III criteria do not locate it. It has to be considered that no such natural resting place exists.

6 A recent independent challenge to the Kraepelinian viewpoint is the suggestion that the clinical picture in the individual patient is less constant than is often thought. Thus Sheldrick (1975) and Sheldrick *et al.* (1977) draw attention to a group of patients who present with apparently typical schizophrenic illnesses but later suffer from episodes of illness which are affective in form. Conversely, Kendler & Tsuang (1982) report a pair of monozygotic twins, each of whom progressed from typical affective illness to 'process' schizophrenia. They suggest that the progression is genetically determined. Most unexpected, from a Kraepelinian and a genetic viewpoint, is the

report of McGuffin *et al.* (1982) of identical triplets discordant for psychosis according to binary typology. Each of these findings appears to be exceptional (although the precise frequency of such exceptions is of considerable interest), as can be seen from the findings of the major studies (e.g. Tsuang *et al.*, 1980; Gershon *et al.*, 1982; Kendler *et al.*, 1985), which document the relative stability of clinical picture within families.

7. Ødegaard's (1963, 1972) conception of a "multifactorial" genetic background was based on observations of overlap in family studies in series of consecutive admissions. The notion was particularly well developed in the 1972 paper, in which the range of diagnosis in the proband was extended (Fig. 3). A 'spectrum' concept of the psychoses was also formulated by Beck (1972), without reference to genetic studies. Beck considered that two variables were involved—a schizophrenic variable linked to a poor prognosis, and an affective variable linked to a good prognosis.

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The Attachment Dynamic in Adult Life

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A case is presented for regarding the Attachment Dynamic as a theoretical model for hypotheses on the aetiology and management of neuroses and personality disorders. It is proposed that man has a number of natural propensities, including: (1) attaining 'companionable interactions' with peers, in which competence is affirmed, self-esteem enhanced, and a state of well-being ('assuagement') is promoted. (2) when hindered in the pursuit of interests and companionable interaction, an urge to seek support through proximity to, and often 'supportive interaction' with support-givers. (3) severe psychological distress ('disassuagement') when support-givers cannot be induced to act effectively, with a propensity to devise defensive strategies, supplemented by psychological defence mechanisms; when maladaptive, these strategies are the source of neurotic symptoms and antisocial traits. Through these propensities, movement between companionable interaction and effective support sustains the urge to develop skills and explore interests creatively. However, this movement is hindered by the experience of repeated disassuagement and the need to maintain maladaptive defensive strategies.

Clinicians working with adults continue to look for more reliable and testable theoretical models that can be used as a basis for examining and treating patients suffering from various forms of psychopathology, notably neuroses and personality disorders. Much interest has been aroused by Bowlby's Attachment theory (Parkes & Stevenson-Hinde, 1982) but clinicians have had difficulty in using it as a developing conceptual framework (Bowlby, 1980) or as an open-ended model (Ainsworth *et al.*, 1978). There are three major impediments to the fuller use of Attachment concepts in clinical work. Firstly, as it stands, the theory is not sufficiently comprehensive to be used as a basis for the assessment and management of adult patients. Secondly, the predominant emphasis on objective observation in the way the theory is framed, while constituting an attraction for behavioural scientists, under-emphasises the significance of a patient's experience, which is an essential currency for clinicians. The third impediment is the absence of a sufficiently formulated definitive framework that can act as a bridge between attachment phenomena in children, on which the theory was developed, and those phenomena in adults.

The theoretical model which we outline here, is a minimum first step to deal with these limitations. It is a development of the concept of the Attachment Dynamic (Heard, 1978, 1982), reformulated to generate hypotheses regarding the aetiology, management, and treatment of neuroses and personality disorder. Clinical findings can then be used to

correct the theoretical statement, and to determine its explanatory power.

Basic Concepts

The concept of the Attachment Dynamic (Heard, 1978, 1982) emerged from integration of the theoretical innovations of Bowlby (1973, 1980, 1982) with the experimental findings of Ainsworth (1969, 1977) and the insights of Winnicott (1971, 1972). The first version outlined an interpersonal dynamic which sets out some of the situations in which three kinds of behaviour relate to one another, namely (1) care-giving, (2) care-seeking (attachment behaviour) and (3) exploratory behaviour or play, as conceptualised by Winnicott (1971).

The transition to this second version came about after the concepts on which the first was based were integrated with concepts of: (1) peer interactions; (2) competence, mastery, and self-esteem (Hendrick, 1947; White, 1959; Frank, 1979; Phillips, 1967); (3) demoralisation (Frank, 1979); (4) the closed sado-masochistic system (Fairbairn, 1952; Rubens, 1984), and (5) qualities of interactions (Hinde, 1979).

Two experiences led to the transition. The first entailed clinical observations on adult clients who showed distress over the absence of like-minded companions of similar experience and stamina with whom to engage in mutually interesting and enjoyable activities. This evoked the concept of 'companionable interaction' as an essential component of exploratory behaviour. The second experience arose during the translation into operational terms of the concept of ego strength (Lake, 1985) by rating observable abilities in terms of personal and social competence. This brought together ideas from a number of different schools

of thought, and led us to focus on the commonplace concepts: (1) that an individual's monitoring of his/her own effectiveness is essential for the attainment and maintenance of selected goals, (2) that such monitoring is markedly influenced by the views expressed by those he holds as significant, and (3) that the recognition of potential and actual competence by others, as well as by oneself, is commonly associated with a sense of increased well-being. Lack of such recognition is associated with loss of autonomy and with a sense of being devalued.

This new emphasis prompted us to distinguish (1) two interpersonal, continuing goals for the dynamic of companionable interaction, and of supportive interaction; and (2) the conditions which in everyday life lead individuals to seek one goal rather than the other. It is important to distinguish the set goals of the dynamic from its function. The function enables man to survive by developing exploratory propensities, executive skills, and autonomy, within a limited but expanding network of trusted relationships. In so doing, man maintains his "environment of evolutionary adaptiveness" (Bowlby, 1982), which enables him to evolve features distinguishing him as a species. An essential feature of man's environment of adaptiveness is the continuing presence, in the life of each individual, of a hierarchy of preferred relationships through which a state of 'assuagement' can be maintained.

Attachment Dynamic in Adults

Intrapsychic events will be described from the point of view of an individual's experience; as the dynamic describes movement between interpersonal aims, we have to break into a "story" (Medawar, 1967), which is circular.

Companionable Interaction and the state of 'assuagement'

The concept of companionable interaction is supported by research on the social behaviour of children (Mueller, 1979), Harlow & Harlow's (1965) concept of peer-peer affectional systems, and Hartup's (1980) observations on peer interaction and the growth of social competence. It has an affinity with Murray's (1938; 1959, Murray & Kluckhohn, 1953) concept of affiliative relationships, Sullivan's (1953) conceptualisation of the relationship between 'chums', and corresponds closely to Weiss's (1982) concept of affiliation.

The exploratory nature of companionable interaction

We postulate that individuals have a natural propensity to have 'companionable interactions' in specifiable circumstances with peers, i.e. people of broadly equivalent intelligence, stamina, capacity to handle fear and panic, and competence in the pursuit of interests. Companions are peers who have similar commitments to sharing one or more interests. Companionable interactions may take place in twosomes, threesomes, or in small or larger groups. It has the function of providing both shared awareness and understanding of an interest, and the recognition of competence; such interactions are therefore episodes in an exploratory

endeavour. 'Interest' is used in the most general sense for work and leisure activities, e.g. dancing, football, teaching, cooking, engineering, and literature; some are pursued alone, with episodes of companionable interaction, while others are more social, and the interest is pursued in small groups.

Factors evoking companionable interaction

Factors which evoke the seeking of companionable interactions include: (1) reaching new levels of understanding in regard to an interest, or of achievement in an associated skill; (2) observing other individual(s) doing likewise, (3) recognition that a companion requires support in the area of the interest, or requiring such support oneself.

Emotive messages and the goal of companionable interaction

The goal of companionable interaction is reached during the sharing of new information about an interest and the skills associated with it; this comes about through the exchange of 'emotive messages', which relate to the command aspect of a communication (Bateson, 1951). In these each participant conveys, in non-verbal as well as in verbal language, the feelings, thoughts, and wishes each has about the behaviour of the other(s). They are the means by which each participant in the interaction conveys whether or not he is anticipating, or experiencing the goal of the interaction. We describe here the emotive messages associated with reaching or failing to reach the goals of the two interactions of the dynamic. We do not discuss details of facial expressions, gestures, tones of voice, and syntax that may be used in conjunction with verbal information to communicate emotive messages, but paraphrase the spirit of the messages.

The goal of companionable interaction is reached whenever an individual construes from the emotive messages sent by companions, that they are taking interest in and showing appreciation of his contribution and the way he has made it; and at the same time, realises that the interest he is taking in the contribution of his companions is also appreciated.

'Assuagement' as a consequence of effective companionable interaction

As a consequence of experiencing the goal of the interaction, a state of mind is reached which, for want of a better term, we call 'assuagement'. In this state, there is: (1) an affective experience of mental and physical well-being, a feeling of satisfaction and enjoyment in what one is doing; (2) a sense of confidence and self-esteem in the ability to achieve short or longer-term aims which are consistent with stages of life, abilities, and interests; and (3) awareness that one's personal supportive environment is secure and that opportunities for companionable and supportive interactions are available.

In the state of assuagement, an individual is also able to: (1) consider the consequences of his actions, (2) see issues from another's point of view; (3) reappraise past experiences; and (4) integrate new approaches with known

routines to carry out short-and longer-term plans. There is also the disposition and readiness to develop ventures and pursue interests creatively, in ways that expand meaning and widen relationships with others. This purposive dimension embodies the motivation to engage in companionable interaction and so to maintain a state of assuagement. The state can consequently be considered as experience of basic trust, autonomy, and initiative (Erickson, 1965). In the contrasting state of 'disassuagement', all these abilities tend to be over-ridden.

Support and the maintenance of creative exploration

Circumstances which evoke movement towards psychological support

Support-seeking is evoked when an individual finds himself hindered in pursuing an interest and engaging in companionable interaction. This may occur through the recognition that competence and effectiveness are, or are about to be diminished (for physical or psychological reasons), or that companions are not available. Once pursuit of an interest has been threatened, psychological support is required before an individual is able to become sufficiently self-reliant to deal with the interfering circumstances. This support restores the individual's confidence in his ability to: (1) review his goals concerning a particular interest, and his relationship with companions; and (2) make and carry through plans likely to be effective; in so doing, loss of confidence is reduced, hope of acting competently and autonomously increased, and self-worth and self-esteem raised. A state of assuagement may be anticipated at this stage but support, however effective, does not in itself bring about assuagement until companionable interaction over an interest is attained.

Psychological support for adults and the use of internal representational models of experience in relationships (IMERs)

Psychological support for adults comes from two interdependent systems. An internal system interconnects with an external one which is used whenever the support provided by the internal proves insufficient. The internal system comes into being during infancy, and continues to evolve throughout life; it is derived from the experience of the interactions an infant has with principal care-givers (attachment/care-giving interactions described by Bowlby, 1982). These interactions with people in the external environment are continuously encoded into internal representational models which the individual constructs of himself and his environment. This concept of representational models has been extensively explored (Bartlett, 1932; Piaget & Inhelder, 1948; Fairbairn, 1952; Craik, 1943; Cantlil, 1950; Miller *et al.*, 1960; Young, 1964; Bowlby, 1982). We postulate that some representational models, viz. of experiences in both companionable and supportive relationships (IMERs) form the basis of an individual's internal supportive system. IMERs store information (see Appendix) in ways that enable the lessons continuously learnt from interactions to be integrated with other related experiences, so

that it can be used to: (1) construct and correct attributions about causes for success or failure in such interactions; and (2) predict how competence can be increased, and companionable and supportive relationships be found and maintained. Each relationship an individual has with others is recorded in different IMERs as a sequence of episodes, during which different kinds of interaction between that individual and another take place. Any information about the other(s) already encoded on other IMERs is added to the record, so that personal histories of particular relationships are reconstructed over time. For example, a man after marriage adds an IMER of himself relating to his wife, later an IMER of himself relating to his child, and then a third IMER of himself relating in a threesome with his wife and child. These histories include beliefs about what can be expected to take place within each relationship.

People who feature in a personal history of a relationship appear in various roles, covering every combination of companion, support-giver, or support-seeker. Thus, someone may feature in a history of a relationship as a reliable support-giver, seeking minimal support but only marginally acting as a companion; another may appear in a more reciprocal relationship, weighted towards seeking and giving support or perhaps towards companionship. The combination of roles tends to change as each partner in the relationship develops greater psychological maturity and deals with the vicissitudes of fate. When the search for support is evoked, an individual automatically uses his IMERs to provide memories of analogous situations in which he has experienced effective support, and then adds to his knowledge of the availability of effective support-givers. If these internal resources are insufficient, external support is sought.

The external supportive system: use of the personal supportive environment, attachment network and supportive interaction

The external supportive system in adults has three components. The 'personal supportive environment' describes the ambience in which an individual feels secure: this has affinities with Lewin's (1952) life space, and covers the concept of transitional and/or attachment phenomena (Winnicott, 1972; Boniface & Graham, 1979). It includes activities, recreations, personal routines, pace of life, disposition of belongings, kind of terrain and house found to be congenial; the degree of light, colour, warmth and physical comfort one enjoys; and the spatial and psychological distance from and closeness to, companions, support-givers and support-seekers found comfortable. This environment is constructed through the integration of information derived from memories of: (1) contexts in which an individual has experienced harmonious forms of companionable and supportive interaction; (2) natural phenomena and works of man which evoke a sense of wonder and often awe; and (3) religious experience.

Such memories are distilled from information in the IMERs, and act as a vision of a secure environment which an individual sets about creating in tangible form by means of the style in which he lives his everyday life. Whenever an individual is unable to maintain enough of his personal

supportive environment to supply the reminders necessary to supplement the internal supportive environment, he checks over the availability of preferred relationships of his 'attachment network'. Finally, if availability alone is not sufficient, supportive interaction – the third component of the external system – is sought.

'Preferred relationships' refers to relationships in which individuals regularly expect to find opportunities for companionable and/or supportive interactions which are experienced as more rather than less effective. People who are so classed constitute an individual's attachment network, and the degree to which they are preferred establishes a hierarchical order of preference. The concept of preferred relationships in the attachment network circumvents difficulties in describing attachment relationships and affectional bonds in adults (Parkes & Stevenson-Hinde, 1982; Ainsworth, 1982; Weiss, 1982; Cohen, 1974; Svejda *et al.*, 1982). Preferred relationships are experienced in twosomes, threesomes, or small groups; the people concerned will tend to be members of an individual's family and close friends, though we are inclined to include well-loved pets in the network of some people. An individual's attachment network varies in size and composition throughout life, as opportunities for interaction come and go, as commitment to different interests wax and wane, and as locality changes. In response to such changes, those people who remain in the network over a considerable time may move up and down the hierarchical scale. The place a person occupies on this scale at any one time appears to be reviewed regularly on the basis of how: (1) available and enjoyable that person is as a companion; (2) available and effective as a support-giver; (3) congenial as a support-seeker in situations in which support may be sought; and (4) the degree to which defensive strategies and the personal supportive environments of the individuals concerned are compatible.

Effective supportive interaction, its participants, and associated emotive messages

Supportive interaction takes place between one or more individuals who seek support and one or more willing to give it. Support-givers who are able to fulfill the role have greater experience, stamina, and judgment in the areas in which support is sought, as well as a greater capacity to handle the anxiety and fear that may be generated by the circumstances in which support has to be given. The aim of support-givers is to provide the support-seeker with the appropriate mixture of comfort, protection, and relevant educative information that enables the seeker to choose the lines of action that best suits his needs and aims. The aim of the support-seeker is to elicit the mixture that enables him to reappraise his goals and his attitudes about himself, and to remake plans, so that he can return with some confidence to old or new interests and companionable interaction. This mixture is found and given during one or more episodes of interaction, over the period required for the support-seeker to make his reappraisal. The giver finds the effective mixture by being attentive to the seeker's emotive messages and, guided by his empathic understanding of the messages, modifies his behaviour so that the seeker approaches the goal of the interaction. In supportive interaction, the

seeker's emotive messages signal to the giver whether the mixture on offer is good enough, whilst the giver's emotive messages signal his willingness or refusal to adapt the mixture until the goal is reached.

Emotive messages and psychological proximity

To discuss issues of agreement or conflict over the messages expressed by the giver or seeker, we have extended Bowlby's concept of the need for physical proximity to preferred persons in defined circumstances, by introducing the notion of the need for comfortable psychological closeness and distance from such persons; and we contrast it with uncomfortable or distressing psychological distance or closeness (impingement). Comfortable psychological closeness is experienced by individuals whenever they consider their interactional goals are being approached or attained. Uncomfortable psychological closeness or distance is experienced whenever the behaviour of the other(s) is antithetical to the goals of the particular interaction.

Emotive messages exchanged between giver and seeker, during a period in which comfortable psychological closeness is experienced by both, show that each is prepared to relate to, be interested in, and value (although maybe not agree with) the feelings, ideas, and actions expressed by the other. Furthermore, each shows he is prepared to relate to the other, whatever aspects of personal supportive environment or form of defence the other is using. Finally, the support-giver shows pleasure and enjoyment in the seeker's achievements. In effective support, the giver notices the messages from the seeker which indicate uncomfortable closeness or distance, and adapts the mixture of support he offers until comfortable psychological proximity is re-established. Furthermore, the giver monitors his own requirements for support, and if necessary interrupts giving in order to gain support for himself elsewhere.

Consequences of experiencing effective support

Whenever a support-giver recognises that the seeker is able to carry out effective plans that enable the interest(s) about which support has been sought to be resumed or changed, the giver feels free to diminish proximity and to attend to other affairs. For the support-seeker, the end-point of the interaction is the realisation that he can make plans that are likely to enable him to find companionable interaction – through resuming the pursuit of a particular interest or engaging in another – and also maintain a comfortable physical and psychological distance from the support-giver. Provided the most effective members of an individual's attachment network remain available and the individual can maintain the most essential features of his/her personal environment, each experience of effective support increases expectations of finding such support in the future, by increasing the adequacy of his internal supportive system.

The integration of effective companionable and supportive interaction

Companionable and supportive interactions are seen in

combination throughout life. Integration can be seen from the time of the first social smile, when exploration of interests and companionable interaction needs to be enjoyed within a framework provided by support-givers, until a child is old enough to pursue mutual interests with peers. The roles taken in companionable and supportive interaction are influenced by epigenetic factors; they show different forms, commensurate with concurrent levels of psychological and physical development and interactional experience encoded in the IMERs. The most significant contribution from the IMERs which influences the effectiveness of the internal supportive system is the balance between experiences of effective and ineffective support-giving from the support-givers and companions. Experience of both effective and ineffective support is common to all, an individual draws on one or other of these types of experience in different contexts, using them as models of giving support both to himself and to others. Examples of the combination and integration of companionable and supportive interaction can be seen in the ordinary pursuit of interests with companions, in friendships, and sexual relationships based on companionship.

Support as part of companionable interaction

It is commonly observed that companions give each other support (effective or ineffective) whilst pursuing or attempting to pursue an interest, in either work or recreational settings. Interests are pursued most competently in adult life when companions show each other a sufficient degree of effective support in the development and practice of the requisite skills. However, it is not possible to give attention to the exploratory pursuit of an interest, and simultaneously give effective support in an area outside that interest, although the two activities may alternate with each other, as can be observed in friendships.

Friendship, shared interests and reciprocal support

We regard friendship primarily as a companionable relationship, within which interests are enjoyed and support given and received, not only in the pursuit of shared interests but embracing other aspects of life. The more issues over which companionship and reciprocal support can be expected and the more compatible the defensive strategies and personal supportive environments, the closer and deeper the friendship and the more its loss will be mourned. The weighting given to interest-sharing and reciprocal support as an aspect of friendship means that relationships with parental figures will tend to move down the hierarchy of the attachment network, as an adult grows older.

Sexual relationships as a form of intimate friendship

Another example of the integration of companionable and supportive interaction is found when peers express and explore sexual interests. We distinguish two categories of sexual interest: (1) sexually intimate friendships, characterised by shared interests and reciprocal support, to which shared sexual attraction is added, experienced as an

intensely enjoyable sense of physical and psychological closeness. The more sexually intimate the friendship, the greater its claims to the highest place in the hierarchy in the attachment network (2) defensive sexual relationships, characterised by an absence of friendship

Ineffective support, defense and disassuagement

We have considered how the Attachment Dynamic maintains and develops an individual's creative capacity, but should supportive interaction fail to take place (support figures not being available when needed, or available but regularly acting in ways that are antithetical to the goals of supportive interaction) the sense of inadequacy that evoked support-seeking in the first place is exacerbated, and a state of distress – 'disassuagement' – is experienced. Regular and/or intense experiences of such disassuaging interaction induce individuals to devise defensive strategies, in order to reduce intolerable distress and to restore self-esteem and the potential for competence; as a result, interests associated with ineffective support are either pursued with anxiety or abandoned. In these contexts, the supportive system has been put under stress, and the individual either evades companionable or supportive interactions, or else engages in them in maladaptive ways that are tormenting or damaging to himself and/or others.

A view of defensive processes in the attachment dynamic

Psychological processes of defence are a complex and contentious field of study; this discussion will be limited to a view which considers that defensive processes are designed to handle the pain of disassuagement and the fear of loss of autonomy. This derives from psychotherapeutic encounters with clients and from consultations over the management and treatment of patients suffering from neurosis. We find it useful to consider defensive processes as: (1) behavioural strategies which are, for the most part learnt; (2) processes which involve the concept of automatic selective exclusion of information (Bowlby, 1980), and the general paradigm that memory is structured in ways that allow us to learn from experience, using the phenomena of reminding in the process (Schank, 1982).

Disassuaging experiences stored in individual IMERs are partially or wholly segregated from conscious awareness, through automatic exclusion of information. A similar view was expressed, in different terms, by Fairbairn (1952), Guntrip (1961, 1968), and Kernberg (1976). These stores are kept under surveillance, in such a way that any current circumstances that seem similar to previous disassuaging events act as reminders of them; such reminders appear to be glimpses into segregated events, and evoke a wide range of conscious experiences. The glimpses range from re-experience of intensely distressing affects to an apparently inexplicable recall of some incident – with or without any particular affect – which, on later reflection in a supportive setting, is connected with a distressing event. They also elicit defensive patterns of behaviour, seen e.g. in transference and counter-transference phenomena. Using whatever degree of sophistication their cognitive development can command, individuals supplement automatic responses by

devising strategies to minimise each particular type of regularly repeated ineffective support. Once an effective strategy is learnt with particular support figures, it tends to become habitual with those figures, and generalises to analogous situations, so becoming a character trait.

Defensive strategies, although adaptive in the context in which they are devised, may be grossly maladaptive when used in contexts which only seem similar to the original. These strategies are recognised by the presence of the compulsive pursuit of closeness to or distance from certain people, settings, and activities; they include both compulsive sexual activities, many of which may be deviant, or a total inhibition of sexual activity. Common examples include (1) inducing ineffective support-givers in the network to interact in ways that are less disassuaging, through compulsively compliant or rebellious behaviour (2) excluding from supportive interactions persons predicted to be more successful in claiming the attention of a support-giver—a common aspect of sibling and other forms of rivalry (3) staying in contact with ambivalent support-givers by creating the illusion that they are effective, through the selective exclusion of contrary information. Common examples of defensive strategies associated with an individual giving up the pursuit of proximity to ineffective support-givers, include imagining effective interactions with idealised support-givers, or the ruthless exaction of retribution from punitive and rejecting figures

tend to reverse roles and seek support rather than give it, or mask their own disassuagement by defensive strategies.

Description of disassuagement

States of disassuagement are frequently masked by defensive strategies, and are revealed in adults when defences have been breached or when undefended losses are sustained, as in natural mourning. The clinical picture fits Frank's (1979) description of demoralisation and Engel's (1968) giving up/given up complex, in their somewhat less severe forms. Briefly, an individual suffers loss of a sense of confidence and competence in his ability to manage not only external circumstances, but his own feelings and thoughts, and tends to lose faith in what previously gave him a sense of security and significance. These losses reactivate memories of earlier losses and failures, and increase feelings of helplessness and hopelessness. A sense of low self-esteem is induced, frequently associated with guilt and shame, the individual loses sight of his long-term goals, and is preoccupied with avoiding further failure and loss of autonomy. Resentment is commonly felt because those from whom help is expected seem unable or unwilling to give it. In states of disassuagement, depending on the effectiveness of defences, the dominant moods are those of anxiety, irritability, and depression, ranging from mild to severe.

The emotive messages of ineffective support

Psychological distancing which is uncomfortable and distressing arises; firstly, when a companion or support-giver fails to recognise or acknowledge a request for supportive or companionable interaction, or having recognised it, devalues or rejects it; and secondly, when someone assumed to be either a support-giver or -seeker refuses to accept the expected role. In contrast, uncomfortable and distressing closeness arises when individuals, as seekers, are pressurised to accept unsolicited support and/or support they construe to be inappropriate and inadequate, or as support-givers, are subjected to intense demands which they cannot satisfy. In each instance, giver and seeker put each other under painful pressure to accept the meaning and point of view of the other. Giving in to such pressure is frequently experienced as submission, with loss of autonomy which increases the experience of disassuagement.

When psychological distancing or closeness becomes increasingly uncomfortable, messages from each participant express frustration, tension, and increasing fears of being hurt by or of hurting the other. If unacknowledged, such messages, tend to be repeated with escalating degrees of emotional intensity, and epitomise a struggle to regain comfortable psychological proximity or relief. Anger in such situations is frequently an expression of the intensity with which an individual endeavours to force the other to comply with his wishes and beliefs. During such struggles, each of the participants fails to be empathic, and the capacity to negotiate differences is severely limited. Adult support-givers are usually ineffective only when they find themselves having to give support when they themselves need supportive interaction. In such circumstances, they

Disassuagement and the three forms of the Dynamic

We distinguish three forms in which the Dynamic is expressed and disassuagement experienced—one adaptive and two maladaptive. Each is associated with different levels of an individual's exploratory capacity, defences, and the effectiveness of his internal and external supportive system. These factors lead to the development of three different approaches to the pursuit of interests—companionable interaction, support-giving, and support-seeking, and hence to three different forms of the Dynamic.

Disassuagement and the adaptive form of the Dynamic. This form has already been described. It is shown when individuals have enjoyed and anticipate sufficiently effective support and companionable interaction over a range of interests, so that few interests are obstructed by defensive strategies. Disassuagement is suffered by these individuals following the threat or actual loss of highly preferred and effective support-givers, e.g. following natural catastrophe or loss of contact. The experience is seen in pangs of homesickness, or in grief, when mourning is followed by natural resolution over time.

Disassuagement and maladaptive forms of the Dynamic associated with anxious exploration. These forms of the Dynamic occur whenever individuals have been exposed over long periods of time to the experience of ambivalent and/or highly conditional support with key figures. The experience of ineffective support in relation to a few interests is probably a universal experience. When, however, affected interests are widespread and the internal system is

relatively inadequate, the individual has to rely mainly on the external supportive system. Interests are pursued with anxiety, and only in so far as defences will allow, so that the capacity for effective support-giving and companionable interaction is limited.

Disassuagement and maladaptive forms of the Dynamic associated with pseudo-assuagement In these forms of the Dynamic, support has been less effective, more inappropriate, and less predictable than in those associated with anxious exploration. The support-seeker has few expectations of being able to gain realistic appreciation and/or recognition of his abilities and work, over the range of interests associated with disassuagement. In these areas, the goals of companionable and supportive interaction have been replaced by a secondary defensive goal – pseudo-assuagement – which is egocentric and markedly hedonistic. We adopted this term because individuals give up the search for supportive and companionable interaction associated with a creative pursuit of interests in work or recreation to become compulsively attached to various forms of excitement and relief.

When disassuagement is associated with almost everything an individual does, it is experienced as a severe form of demoralisation and associated with a pervading sense of futility. Defensive responses which have to be continuously maintained include: Firstly, types of maladaptive self-support which enhance various forms of self-comfort, geared to providing immediate relief from tension, e.g. common forms of addiction associated with denial and rationalisation; and secondly, forms of defensive companionship in which various modes of achieving excitement and/or status are idealised and pursued as ends in themselves. However, the search for effective support is seldom completely abandoned, and should appropriate skilled support be found, the primary goal of the Dynamic may be reinstated.

Individual styles within the attachment Dynamic

The forms in which the dynamic may be expressed in different contexts contribute to an individual's unique personal style, e.g. the way the Dynamic is expressed over a day may change from an adaptive form to forms associated with anxious exploration or pseudo-assuagement, as an individual moves from life at home to work, or to leisure activities. Similarly, individual styles in maintaining the goals of the Dynamic are likely to change over a lifetime, as different developmental stages are approached and life events affect the opportunity to maintain an effective attachment network and personal supportive environment. An observer of any one area or stage of an individual's life will therefore see only that one aspect of the total picture. However, it is possible, for clinicians to work out the style in which the Dynamic is currently, or has previously been expressed by examining the present nature and history of: (1) states of mind associated with companionable interaction, and both roles of supportive interaction; (2) the size, availability, and effectiveness of the attachment network; (3) emotive messages concerning psychological proximity regularly used

during companionable interaction, and in both roles of supportive interaction; (4) the level of commitment to, and effectiveness in the range of interests pursued in the various contexts of everyday life; and (5) the kind of personal supportive environment maintained and the degree of preoccupation in maintaining it.

Discussion

The outline we have presented represents a loom on which may be woven the rich, elusive and at times repellent tapestry of human relationships. Our concern has been to set up a sufficiently comprehensive warp to investigate the possibilities and limitations of human potentialities.

This model leads to the conclusion that men and women cannot be co-operative and look after themselves and others unless they can enjoy practising skills that reveal creative talents with their peers. But the model shows that this can best be achieved when individuals live in the secure knowledge that they have relationships with people who recognise their potential for creativity, and who can provide information, protection, and comfort that enables them to restore the experience of competence, whenever their skills are no longer effective. Whilst recognising the intensely destructive nature of certain defences against disassuagement, the model suggests that much hostile aggressive behaviour is a strategy to handle fear concerning survival, arising from failure to find effective support.

Experience of sustained disassuagement is associated with psychopathology, which can range from mild to severe forms of neurosis, personality disorder, and borderline state. In our view these disorders are manifestations of an interplay between constitutional factors, the history of past interactions in preferred relationships encoded in the IMERs at all stages of life, and 'here and now' experiences in social relationships. Therefore treatment should take into account both the history encoded in the IMERs, and the quality of the interactions with those who provide formal or informal treatment. Some of the features of the therapeutic relationship required are discussed by Frank (1979) according to the model of the Attachment Dynamic, these are highly specific elements of effective supportive interactions, and will therefore play a part in all forms of management, counselling, adult peer self-help groups, different forms of individual and group psychotherapy-psychoanalytic and cognitive behavioural, and family therapy.

It should not be thought that all psychological distress, particularly that associated with organic disorders, depressive illness and schizophrenic disorders, is primarily a manifestation of states of

disassuagement. Loss of competence is, however, associated with any illness, physical or mental, and because it evokes a desire for support, it will exaggerate co-incidental neurotic manifestations.

We conclude with a reminder that the aim of this model is to generate hypotheses regarding the aetiology, categorisation and treatment of neuroses and personality disorder and to modify the theory in the light of the findings.

Appendix

Information in each IMER is stored in chronological order, and relates to: (1) the content of verbal exchange during interaction and the perception of the meaning of emotive messages; (2) perception (from the five senses) of the environment in which interactions take place; (3) other environmental information preceding or following the interaction that is available for association with it. We class these three categories as external information, and think that most attention is given to information relating to one's value, the roles one is expected to take, the rules one is expected to follow, and the level of performance required.

Other information comes from internal sources: (4) affective responses to environmental information regarding oneself, others involved in the interaction, the surroundings, and perceived consequences. (5) information related to defensive strategies, including handling disassuaging

relationships through phantasies of revenge, retribution, idealised forms of pleasurable interaction, and/or attaining levels of unparalleled admiration and esteem. (6) the evaluation of one's worth and performance after comparison with: (a) information already stored in the IMERs of previous interactions with appearances similar to those currently evaluated; (b) external information from other(s) before, during, and after the interaction, concerning one's worth and performance; and (c) one's personal judgment of personal performance concerning immediate and/or long-term aims.

On the basis of these evaluations, beliefs about one's worth and capacity to perform effectively are revised or confirmed, and rules concerning conduct formulated. However, there are two instances when the opportunity for revision and/or reappraisal is severely restricted: (1) instances where similar events have been followed by similar consequences over lengthy periods of time, and habituation has occurred; (2) the defensive segregation and systematic exclusion of disassuaging information.

We conceptualise the self as a form of "overall dynamic structural matrix" (Sutherland, 1979; Kernberg, 1980), constructed so that an individual can: (1) relive his experiences within a particular IMER or some part of it; (2) compare and contrast himself in different relationships and different contexts by extracting information relating to his experience from a number of IMERs. We consider that the latter facility underlies the phenomenon of the observing ego, which probably develops after infancy, but is often overridden during experiences of disassuagement.

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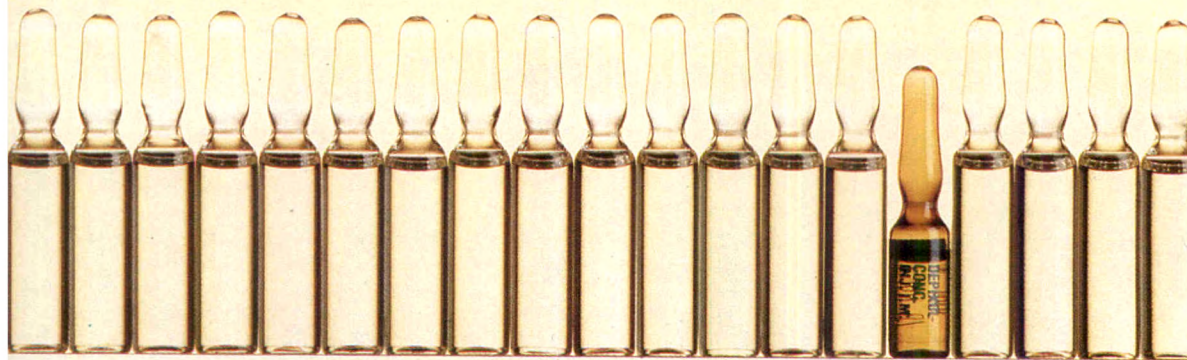
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Significance of Positive and Negative Syndromes in Chronic Schizophrenia

S. R. KAY, L. A. OPLER and A. FISZBEIN

Positive and negative syndromes were studied in relation to demographic, historical, genealogical, clinical, psychometric, extrapyramidal, and follow-up measures of 101 chronic schizophrenic patients. The criterion scales proved to be reliable, normally distributed, and strongly correlated with general psychopathology, but otherwise inversely related to one another. Multiple regression analysis identified sets of 4-6 independent variables that explained 74%-81% of the scales' variance. A positive syndrome was associated chiefly with productive features, family history of sociopathy, more previous hospital admissions, and longer in-patient stay during the 30-month follow-up period. A negative syndrome correlated with deficits in cognitive, affective, social, and motor spheres, higher incidence of major psychiatric illness but less affective disorder among relatives, lower education, and greater cognitive developmental impairment. The results underscore the importance of genetic and biodevelopmental variables for understanding schizophrenic syndromes.

The issue of heterogeneity has been at the heart of schizophrenia research since the writings of Bleuler (1911). Many efforts have been made to identify subgroups similar in phenomenology, with the hope of casting light on fundamental differences in aetiology, treatment strategy and prognosis. The early view of schizophrenia as constituting primarily a defect state along a deteriorative course (Kraepelin, 1913) has been gradually overtaken by the emphasis on productive features as pathognomonic of the disorder and even as a prerequisite for its diagnosis (Schneider, 1959; Feighner *et al*, 1972; American Psychiatric Association, 1980).

Since the beginning of this decade, Crow (1980a,b) and Andreasen (1982; Andreasen & Olsen, 1982) have proposed that these two aspects of schizophrenia may represent distinct syndromes of different aetiology. The Type 1 or 'positive' syndrome was defined by productive symptoms such as delusions, hallucinations and disorganised thinking. It was considered to issue from disturbance in dopaminergic transmission and be responsive to neuroleptic drugs, hence bearing a favourable prognosis. The Type 2 or 'negative' syndrome was characterised by deficits in cognitive, social, and affective functions, and was thought to be associated with structural abnormality in the brain, portending resistance to neuroleptics and a poor prognosis.

The fledgling research on positive and negative syndromes has provided some basis for their distinction, but the studies have generally been limited

by small samples, unstandardised rating methods, lack of long range follow-up, and no control for extrapyramidal drug side-effect (EPS) which can interfere in the assessment of motor activity, affect, and verbal productions (Rifkin *et al*, 1975; Van Putten & May, 1978). Therefore, many of the conclusions regarding aetiology and treatment response remain inferential.

Studies that relate these syndromes to differences in drug response and brain structure have yielded mixed results. Positive schizophrenic symptoms were reported to be improved by neuroleptic drugs (Johnstone *et al*, 1978a; Angrist *et al*, 1980) and worsened by anticholinergics (Johnstone *et al*, 1983), whereas the negative symptoms were essentially unaffected by either medication. In the Angrist study, however, one of the three negative parameters—emotional withdrawal—actually benefited from neuroleptics. The Johnstone group, in their assessment of 17 chronic schizophrenic patients also observed that those who had enlarged ventricles tended to have intellectual deficits and negative symptoms (Johnstone *et al*, 1976, 1978b). This finding was echoed by Andreasen *et al* (1982), although of the ten symptoms designated as positive or negative only bizarre behaviour was significantly related to ventricular size. They noted, however, greater deviation in the cognitive ratings of patients with predominantly negative symptoms, suggesting an intellectual deficiency. More rigorous experimental techniques have since found such persons slower in visual information processing (Green & Walker,



1984) but equally disordered in language production (Allen, 1983), although both these studies involved small samples (between 6 and 10).

In terms of prior functioning, patients with a mainly negative syndrome seem to have had poorer premorbid adjustment, including lesser education and work experience (Andreasen & Olsen, 1982; Andreasen *et al.*, 1982), which raises the possibility of pre-existing developmental failures. A similar finding emerged from a comparison of 39 schizophrenic patients with 33 unipolar depressives in the follow-up phase (Pogue-Geile & Harrow, 1984); negative symptoms in schizophrenia were associated with educational difficulties and poor social functioning both before and after initial admission, thus implying antecedent and sustained deficits. Since the positive-negative assessment was conducted only at follow-up, the analyses were retrospective in nature.

With expanded representation of cognitive and historical parameters, we recently carried out multi-dimensional comparisons of schizophrenic patients who showed predominantly positive syndromes with those showing predominantly negative syndromes during the acute phase of illness ($n=37$) (Lindenmayer *et al.*, 1984) as well as the chronic phase ($n=47$) (Opler *et al.*, 1984). In both studies, a positive subgroup was defined by a rating of 'moderate' or greater pathology along at least three out of seven positive dimensions and fewer than three out of seven negative dimensions (see below); a negative subgroup was defined by the reverse pattern. All other cases were disregarded.

The acute survey indicated that the negative syndrome was not secondary to the progression of the disorder, prolonged institutionalisation, severity of illness, neuroleptic dose, or EPS. Before the onset of illness, patients of this description had less schooling and poorer work adjustment. The study of chronic patients found the negative type to be older, less educated, and more often born in winter months. Despite comparable intellectual impairment and visual-motor deficit, they displayed a more primitive cognitive mode and greater psychomotor retardation. As a whole, these converging results have led us to suspect a cognitive developmental deficiency, possibly of organic basis, that underlies the schizophrenic defect state and its resistance to neuroleptic intervention.

One limitation of the foregoing studies was the typological approach, which presumes the debatable position (cf. Pogue-Geile & Harrow, 1984) that positive and negative syndromes are mutually exclusive. As a result, large proportions who failed to meet criteria for either definition (54% of the acute and 62% of the chronic group) were lost from the

analyses. The present investigation, by contrast, adopted a dimensional approach which enabled inclusion of the full sample while making no assumptions about a positive-negative bipolarity. Thus, based on the preceding assessment technique, measures were taken along three continuous scales to provide positive syndrome, negative syndrome, and composite (positive minus negative) scores. These data were studied among a larger group of 101 chronic schizophrenic in-patients in relation to a broader range of predictor variables, which encompassed demographic, historical, genealogical, clinical, psychometric, extrapyramidal, and outcome measures. By doing so we hoped to better elucidate the critical parameters underlying positive and negative schizophrenic phenomena by building into the design several methodological advantages over the previous surveys: (a) a substantial sample size; (b) the use of standardised psychometric instruments for assessing cognitive, developmental, attentional, and motor dysfunctions; (c) an expanded range of study; (d) provision of a follow-up outcome measure; (e) evaluation and control for the influence of EPS; (f) measurement of the positive and negative syndromes as both bipolar and unipolar constructs; and (g) assessment of the distribution and internal reliability of positive and negative ratings. We postulated, in line with our earlier work (Lindenmayer *et al.*, 1984; Opler *et al.*, 1984), that differences along the positive-negative dimension may be traced to premorbid developmental factors, are not mere artifacts of EPS, and may have direct implications for the future course of hospital treatment.

Method

Subjects

The medical charts of in-patients from long-term psychiatric units in a university-affiliated urban hospital were consecutively surveyed to select those having a formal DSM-III diagnosis of schizophrenia (American Psychiatric Association, 1980). Cases with questionable diagnoses, known organic disorders, or mental retardation were excluded. The final sample consisted of 101 subjects who ranged in age from 20 to 68 (mean = 36.81, s.d. = 11.16), and included 70 males, 31 females, 33 whites, 43 blacks, and 25 Hispanics. The mean age of first psychiatric admission was 22.39 years (s.d. = 8.63), and the mean duration of illness since then was 14.41 years (s.d. = 8.95). Over the previous 18 months, 67% had been continuously in hospital; for the remainder, the mean duration of in-patient stay was 195 days. All patients were receiving psychotropic medication when surveyed. Of the full group, 72-81 subjects participated in the various cognitive tests and 42 took the random number generation task.

Design and measures

All patients suited for study on the basis of initial chart

review were interviewed on their own ward by a research psychiatrist in order to independently confirm fulfillment of DSM-III criteria for schizophrenia. They then underwent a series of evaluations deriving from clinical interview, cognitive testing, physical assessment, and intensive examination of medical records. The data collection proceeded without knowledge of patients' status on positive and negative syndrome scales, which were scored only after all measures had been conducted.

Positive-negative evaluation Patients underwent a 30–40 minute semi-structured clinical interview with the research psychiatrist, after which they were rated on the 18-item Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1962) and an abbreviated 12-item Psychopathology Rating Schedule (PRS) (Singh & Kay, 1975), which provides a 1–7 point rating system in parallel to the BPRS and was included because it enabled a fuller assessment of deficit features. Furthermore, it presented criteria for each dimension and for four focal points on the scale so as to enhance objectivity and reliability.

Of the 30 psychiatric parameters from these two scales, 14 served for assessment of positive and negative syndromes, as detailed elsewhere (Lindenmayer *et al.*, 1984; Opler *et al.*, 1984). The positive symptoms were delusions (from PRS), and conceptual disorganisation, hallucinatory behaviour, excitement, grandiosity, suspiciousness and hostility (from BPRS), the negative symptoms were blunted affect and emotional withdrawal (from BPRS), and poor rapport, passive-aphetic social withdrawal, difficulty in abstract thinking, lack of spontaneity and flow of conversation, and stereotyped thinking (PRS). Positive and negative syndrome scores were generated from sums of ratings on the seven items constituting the respective clusters. Each scale thereby had a range from 7 to 49. In addition, a composite positive-negative score was calculated by subtracting the negative syndrome score from the positive, thus yielding a bipolar index that ranged from +42 to -42, which reflected the degree of predominance of one syndrome over the other.

Clinical assessment. After excluding the 14 items involved in the positive-negative scales, the remaining 16 items from the BPRS and PRS served as individual measures of schizophrenic symptoms, and their sum provided an index of general psychopathology.

Based on the same clinical interview, the research psychiatrist assessed patients also on the Manifest Affect Rating Scale (MARS) to permit a more detailed inspection of affective impairment, this 15-item scale, modified from the procedure developed by Alpert & Rush (1983), is composed of 4-point severity ratings, each with descriptive anchoring points along a range from 0 (normal) to 3 (extreme pathology). Principal component analysis has identified three factors underlying the scale: emotional relatedness (mean loading=0.80), expressive mobility (0.71), and appropriateness of affect (0.60).

Cognitive testing A series of objective psychometric tests was undertaken to examine the proposition that a negative syndrome in schizophrenia relates to cognitive and neurological deficits (Andreasen & Olsen, 1982; Andreasen *et al.*, 1982) that may be of developmental origin (Lindenmayer *et al.*, 1984; Opler *et al.*, 1984). These procedures were adminis-

tered by a research psychiatrist or specially trained and supervised medical students and then scored independently by a psychologist. All involved were unaware of patients' scores on the positive-negative scales.

Intelligence was gauged with the Quick Test (QT), a recognition vocabulary measure that correlates strongly with the standard individual IQ scales (Ammons & Ammons, 1962). This technique was selected for its relative invulnerability to cognitive and performance deficits associated with psychosis (Blatt, 1959) and for its minimal demands on prolonged attention and verbal productivity.

The Memory For Designs Test (MFD) provided a standardised index of neurological dysfunction based on impaired perception of configurations and short-term visual recall (Graham & Kendall, 1960); it was chosen because of its objective scoring criteria and adjustments for age and IQ. The hand used for holding the pencil in taking this test served as indication of hand dominance.

Disturbance of attention was quantified temporally using the Span of Attention Test (SOA), which assesses the average duration that one concentrates on a routine motor task before showing signs of distractibility (Kay & Singh, 1974; Kay, 1982). The procedure also yields a measure of spontaneous psychomotor rate, calculated as the ratio of test items completed to time elapsed.

Cognitive performance was studied on a random number generation test (Salierno *et al.*, 1984), an objective treatment-sensitive measure of success in purposeful goal-directed (rather than perseverative) responding, which has been considered a fundamental adaptational deficit in schizophrenia (Shakow, 1979). Instructions call for continuous recitation, over a 60 second period, of as many numbers as possible between 1 and 10 without using any particular order. Number fluency is scored as the ratio of random numbers (i.e. discounting sequences of three or more identical or consecutive integers) to total numbers recited.

To obtain a developmentally-based assessment of cognitive processes, four tests from the Cognitive Diagnostic Battery (Kay, 1982) were administered. The Colour-Form Preference Test (CFP) examines primitive bases for perceiving relationships and pre-representational modes of thinking (Kay & Singh, 1975; Kay, 1977). It consists of a 20-item similarity judgement task wherein a standard card is matched to one of three comparison cards that are alike in colour, form, neither cue, or both. The response strategy is scored for predominance of perseveration, random response, colour concept, form concept, and multilevel concepts, which correspond to successive developmental levels up to the 7–11 year old stage. The 20-item Colour Form Representation Test (CFR) extends the CFP assessment by adding the option of match by pictorial representation, which is a more mature criterion of similarity than colour or form (Kay *et al.*, 1975). As such, it focuses on the later verbal-symbolic stages of conceptual growth. The Egocentricity of Thought Test (EOT) consists of hierarchical subtests which assess the maturity of right-left positional concepts (Kay & Singh, 1975). These depict four major phases of development identified by Piaget (1928) as crucial to cognitive and social growth: preconceptual (<age 5 years), egocentric (5–8 years), socialized (8–11 years), and

objective (>11 years). The Progressive Figure Drawing Test (PFDT) delineates the level of perceptual-motor development in patients with combined developmental and psychiatric disorders (Kay, 1980). The procedure involves drawing from copy seven simple designs that children normally master at successive ages between 2 and 6.

These four developmental measures have shown high reliability and have differentiated schizophrenics from non-psychotics and retarded patients and among various diagnostic and prognostic subgroupings of schizophrenics (Kay & Singh, 1979; Kay, 1982).

Drug side-effects. The prevalence of drug-induced EPS was measured after complete examination using two 5-point severity rating scales: Abnormal Involuntary Movement Scale (AIMS) (National Institute of Mental Health, 1974) and Extrapyramidal Rating Scale (ERS), as originated by Alpert *et al* (1978). The 10-item AIMS assesses movement disorders involving the face, mouth, extremities, and trunk. The ERS, which is similar to Borison's (1984) Scale for Recognizing Acute EPS, evaluates bradykinetic and parkinsonian drug reaction along seven dimensions: abnormal gait (stooping, loss of associated movement, shuffling, propulsion), rigidity (resistance to passive stretch, cogwheeling), tremor of extremities (including choreiform movements and pinrolling), bizarre movements (torticollis, facial grimace, tongue tremor, tics, oral movements), expression (masked facies, dysarthric speech, loss of volume, loss of inflection), restlessness (constant walking, rocking, chewing, sitting), and reduced motor activity (fatigue, slowness, weakness, inactivity).

Historical review. The following variables were generated from information in patients' medical records and the hospital's computerised storage bank: demographic characteristics (age, sex, race, ethnic group, marital status); season of birth; years of formal education; age of onset and chronicity of illness, both as determined by date of initial psychiatric admission; and total number of hospital admissions.

Family history of illness. Based on interview with the patient and scrutiny of psychosocial, developmental, and family histories from the medical records, the following categories of psychopathology in the patient's first-degree relatives were identified: schizophrenia, where the family member was admitted to hospital with this diagnosis; affective disorder, where admission had occurred for depressive, manic, or bipolar illness; major psychiatric disorder, where schizophrenic illness, major affective disorder, or unspecified mental illness had led to admission; suicide, where such was reported; alcoholism, where a history of alcohol abuse or heavy drinking had existed; and sociopathy, where the records indicated criminal behaviour resulting in arrest and prosecution. Considering the probable bias of families against acknowledging mental disorders as well as the ever present possibility of errors of omission, these data are considered minimal estimates of familial illness.

Follow-up. With the help of the hospital's computerised information system, the subjects' status as in-patients was followed up for 2½ years after their participation in the study. The total length of subsequent in-patient stay provided an index of continued need for institutional care, and it thus permitted assessment of the long-range predictive validity of positive and negative syndromes.

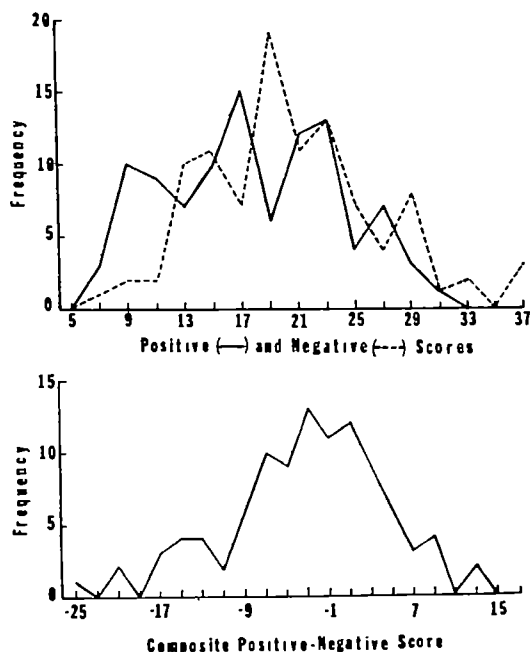


FIG. 1 Frequency distribution of the positive syndrome, negative syndrome, and composite positive-negative scales.

Results

Positive-negative scales

The scores on the positive, negative, and composite measures were found to be distributed fairly normally, without substantial skewness or kurtosis (Fig. 1). The positive and negative scales bore a modest but significant inter-relationship (Pearson's $r = +0.27$, $P < 0.01$), and both were strongly correlated with the composite scale ($r = +0.59$ and -0.61 respectively, $P < 0.0001$), whose scores centered around a median of -2.00 . Positive and negative scores also bore an exceptionally high relationship to the independent measure of general psychopathology ($r = 0.68$ and 0.60 respectively, $P < 0.0001$), suggesting that global severity of illness may have mediated the association between the two criterion scales. Indeed, a partial correlation which excluded the shared variance from general psychopathology revealed a significant inverse correlation between positive and negative scores ($r_{12.3} = -0.23$, $t = 2.37$, $d.f. = 98$, $P < 0.02$).

The reliability of the criterion measures was analysed using coefficient alpha for inspecting the internal consistency of the component scales. As detailed in Table I, each of the items comprising the positive and negative syndromes correlated significantly (at least $P < 0.01$) with the remaining scale total, and no perceptible gain could be achieved by discarding any item. The alpha reliability coefficients were 0.73 and 0.83 for the two scales respectively.

TABLE I
Reliability analyses for positive and negative syndrome scales

Individual scale items	Total scale with individual item deleted			
	Mean	s.d.	Item-total correlation	Alpha coefficient
Positive syndrome				
Delusions	15.11	5.02	0.65*	0.64
Conceptual disorganisation	15.27	5.58	0.27**	0.73
Hallucinatory behaviour	15.76	5.17	0.44*	0.70
Excitement	15.93	5.54	0.38*	0.71
Grandiosity	15.95	5.23	0.47*	0.73
Suspiciousness	15.57	5.46	0.45*	0.69
Hostility	16.17	5.53	0.45*	0.70
Negative syndrome				
Blunted affect	18.22	5.59	0.53*	0.81
Emotional withdrawal	18.11	5.35	0.69*	0.78
Poor rapport	18.57	5.14	0.62*	0.79
Passive-apathectic social withdrawal	18.36	5.26	0.70*	0.78
Difficulty in abstract thinking	17.20	5.42	0.44*	0.82
Lack of spontaneity and flow of conversation	18.28	4.93	0.78*	0.76
Stereotyped thinking	18.22	5.64	0.29**	0.84

* $P < 0.001$; ** $P < 0.01$.

Correlates of positive and negative syndromes

Correlational analyses were performed to evaluate the relationships between positive-negative syndromes and 65 variables representing different spheres of functioning and different methods of assessment. Following Ferguson (1966), Pearson analysis was used for continuous normally distributed data and point biserial correlation for the discrete dichotomous variables.

Results of the simple correlations found the positive-negative dimension statistically unrelated to such demographic and historical variables as race, cultural group, sex, marital status, age at first admission, and frequency or chronicity of hospitalisation. Likewise, no significant correlations were obtained with respect to depression ratings (BPRS depression, BPRS guilt feeling, and MARS sad affective tone). Positive and negative syndromes also bore no special relationship to variables that might be linked with neurological dysfunction, namely winter birth, left hand dominance, SOA attention span, MFD organicity score, and PFDT perceptual-motor development score.

Alternatively, the analyses indicated certain similarities between patients with pronounced positive and negative features. Both syndromes correlated significantly with general wealth of psychopathology, MARS ratings of inappropriate affect and poor eye contact, and clinical ratings of tension, unco-operativeness, disorientation, impaired attention, lack of insight, disturbed volition, poor impulse control, preoccupation, and social avoidance.

The significant covariates particular to either syndrome are indicated in Table II. The positive syndrome alone was

associated with excessive noncommunicative movements, affective lability, somatic concern, anxiety, and longer duration of in-patient care in the 30 month follow-up. Of special interest was its correlation also with sociopathy in the patient's family ($P < 0.05$), in stark contrast to the relationship of negative syndrome to family history of major psychiatric disorder ($P < 0.01$). The negative scale was furthermore uniquely associated with motor retardation, slowed SOA psychomotor pace, mannerisms and posturing, lower verbal intelligence, greater cognitive developmental deficits on the CFP and EOT, and more serious affective deficiency on most MARS scales as well as the MARS total. Thus, the negative syndrome seemed to entail a deficit state encompassing the cognitive, affective, and motor spheres.

Correlations with the composite scale identified those variables that were allied specifically with one syndrome in opposition to the other. The negative pole was significantly distinguished from the positive as involving patients who had less education, showed fewer signs of anxiety and bizarre thinking, proved slower on clinical and psychometric assessments of motor function, showed greater affective deficits on 9 of the 15 MARS scales plus MARS total, exhibited more rigidity in random number generation, and presented greater cognitive developmental impairment on the CFP, CFR, and EOT.

In the foregoing analyses, there emerged a significant association between negative polarity on the composite index and two control variables: age ($r = 0.25$; $P < 0.02$) and EPS ($r = 0.26$, $P < 0.01$). This raised the possibility that the

TABLE II

Significant non-overlapping covariates of positive and negative syndromes obtained by simple and second-order partial correlations that adjust for variance attributable to age and EPS (AIMS plus ERS total)¹

Variable	Simple correlation			Partial correlation		
	Positive	Negative	Composite	Positive	Negative	Composite
Demographic/catamnestic						
Male gender					0.21	
Years of education			0.27		-0.29	0.33
Number of hospital admissions				0.20		
Days in hospital during follow-up	0.25					
Family history of illness						
Sociopathy	0.22			0.21		
Major affective disorder					-0.21	
Major psychiatric disorder		0.30			0.29	
Cognitive/psychometric						
QT verbal intelligence		-0.23				
Random number fluency			0.31		-0.33	0.39
SOA psychomotor rate		-0.27	0.25			
CFP multi-level concepts		-0.33	0.34			0.27
CFR conceptual level			0.23			
EOT cognitive-social level		-0.35	0.23		-0.34	0.24
Affective (MARS impairment)						
Facial expression (wooden)		0.62	-0.45		0.54	-0.40
Expressive gestures (lacking)		0.54	-0.43		0.41	-0.37
Vocal emphasis (monotone)		0.61	-0.44		0.49	-0.40
Global relatedness (constricted)		0.62	-0.46		0.50	-0.42
Speech rate (retarded)		0.42	-0.44	-0.27	0.25	-0.39
Response latency (slow)		0.58	-0.38		0.47	-0.36
Voice level (soft)		0.40	-0.34		0.32	-0.30
Global mobility (sluggish)		0.55	-0.47		0.43	-0.43
Impoverished thought content		0.54	-0.51		0.52	-0.49
Non-communicative movements	0.24					
Affective lability (excessive)	0.33			0.31		
Angry affective tone	0.47	0.23		0.46		0.23
MARS total		0.74	-0.45		0.64	-0.41
Clinical (BPRS)						
Somatic concern	0.22					0.20
Anxiety	0.41		0.29	0.38		0.33
Mannerisms and posturing		0.25				
Motor retardation		0.41	-0.35		0.22	-0.28
Unusual thought content	0.73	0.21	0.42	0.73		0.50

1 Only values significant to $P < 0.05$ are shown. The full correlation matrix is available on request from the authors.

remaining correlations might have been mediated by older age and drug side-effects. To adjust for the variance attributable to these two parameters, therefore, the data were reanalysed using second-order partial correlations. The results, included in Table II, upheld the significance of lesser education and affective deficits in characterising the negative syndrome. Differences in the cognitive and motor spheres were to some extent diluted, although negative

symptoms were still significantly correlated with motor retardation, difficulty in random number generation, and developmental dysfunction on the CFP and EOT tests. The findings in regard to genealogy were upheld, with the further observation that the negative syndrome was associated with absence of family members who have had affective disorders. Other new findings included a preponderance of males with negative features. A positive

TABLE III
Multiple regression analysis of positive-negative scales

Variable	Coefficient	Standard error coefficient	Standard regression coefficient	Increase in R^2	F to enter	F to remove
Positive scale						
Unusual thought content	2.09	0.27	0.51****	0.57	117.53	61.99
General psychopathology	0.22	0.04	0.34****	0.13	38.75	23.34
Angry affective tone	1.44	0.51	0.18***	0.02	6.76	7.88
Sociopathy in family history	1.75	0.79	0.12*	0.02	4.91	4.91
Negative scale						
MARS total	0.59	0.08	0.53****	0.59	110.94	52.77
Lack of judgment and insight	1.30	0.25	0.29****	0.12	29.77	27.75
Major psychiatric disorder in family history	1.54	0.37	0.22****	0.05	13.78	17.42
EOT cognitive-social level	-1.12	0.34	-0.18***	0.03	9.16	11.16
Active social avoidance	0.65	0.29	0.12*	0.01	4.71	4.98
Impoverished thought content	1.21	0.55	0.16*	0.01	4.86	4.86
Composite scale						
MARS emotional relatedness	-0.85	0.19	-0.40****	0.30	32.28	18.88
Unusual thought content	2.50	0.35	-0.47****	0.26	42.86	51.45
CFR conceptual level	0.39	0.14	0.19**	0.07	13.66	7.34
Years of education	0.51	0.17	0.20**	0.04	7.68	8.65
Impoverished thought content	-2.22	0.89	-0.23*	0.02	5.51	6.19
Age	-0.10	0.05	-0.15*	0.02	4.83	4.83

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$; **** $P < 0.0001$.

syndrome was now associated with more previous hospital admissions, faster speech rate, and angry affective tone, whereas follow-up differences were not significant.

Multiple regression analysis

The significant correlates of the three criterion scales were subjected to multiple regression analyses to identify those parameters which principally account for the variance of the positive-negative dimension. The results (Table III) indicate the most important determinants, beyond which no significant variance is further explained.

The positive, negative and composite scales respectively yielded coefficients of multiple determination (R^2) of 0.74, 0.81, and 0.71, which converted to 0.72, 0.79, and 0.68 ($P < 0.0001$ in all cases) when adjusted for number of component variables. The positive syndrome was accounted for mainly by wealth of psychopathology and, specifically, by productive features added to the cognitive and affective spheres as unusual thinking and angry emotional tone. A family history of sociopathy also contributed significantly to the variance. The negative syndrome, in sharp contrast, was explained by impoverished cognitive and affective processes as well as deficits in social functioning and in the developmental underpinning of cognitive and social growth (EOT). In terms of genealogy, a family history of

major psychiatric disorder rather than sociopathy was a significant aspect of this syndrome.

Of particular interest for differential assessment was the composite scale, since its score delineated the two ends of the positive-negative polarity. From multiple regression analysis, unusual thought content emerged as the chief indicator of a uniquely positive syndrome. A negative syndrome, conversely, was explained by emotional unrelatedness (MARS Factor 1), older age, and cognitive deficits reflected in flawed conceptual development (CFR), poorer education, and impoverished thinking.

Discussion

Our project assessed, in a large sample of long-term schizophrenic in-patients, the significance of positive and negative syndromes in relation to a variety of parameters encompassing personal history, genealogy, epidemiology, mental status, cognitive test performance, and past and future clinical course. Preliminary analyses found the criterion scales to be reliable and normally distributed. A modest but significant correlation between scores for positive and negative syndromes suggested that these are not

independent, in consonance with the observations made by Pogue-Geile & Harrow (1984) during follow-up. When extracting the shared variance attributable to general psychopathology, however, the two syndromes had a significant inverse relationship. This supports the proposition (Andreasen & Olsen, 1982; Lindenmayer *et al*, 1984; Opler *et al*, 1984) that positive and negative symptoms are at opposite ends of a continuum, while justifying the present use of a bipolar composite index.

Multiple regression analysis demonstrated that between 71% and 81% of the total variance of the criterion measures could be explained by sets of 4–6 predictor variables representing separate dimensions. Clinically, the cluster of positive symptoms correlated with productive features involving the cognitive and affective spheres, particularly unusual thought content and inappropriate anger; correspondingly, the negative syndrome was associated with deficits in these spheres as well as in the social and motor realms. Patients with a negative profile generally emerged as socially isolated, vacuous in their affective and intellectual processes, and slow and perseverative in their responses, fitting Shakow's (1979) description of the schizophrenic with major adaptational difficulty. Contrary to Andreasen's assumption that disordered attention represents a deficit symptom of importance in distinguishing a negative syndrome (Andreasen, 1982; Andreasen & Olsen, 1982), clinical and psychometric measures found this parameter equivalently related to the positive syndrome. This result is not surprising in light of the significant association described between attentional impairment and measures of autonomic and cortical arousal on the one hand and social withdrawal on the other (Kay & Singh, 1974; Kay, 1981; Singh & Kay, 1982).

The present survey proceeded beyond concurrent functioning to examine areas not formerly probed in relation to the positive-negative dimension, notably family history of illness and prospective course of hospital treatment. Despite the likelihood that estimates of family illness were conservative, which enhances the risk of Type II error, genealogy appeared to be highly relevant. Thus, a positive syndrome was reliably associated with sociopathy in first-degree relatives, whereas a negative syndrome was linked to a family record of major psychiatric illness but absence of affective disorder.

In terms of the course of illness, a positive syndrome seemed to be anticipated by a greater number of prior hospital admissions and to portend more time subsequently spent as an in-patient, perhaps suggesting stormy episodes with decreased tractability in the more chronic phase. These

observations, however, were not independent of covariance with age and EPS. The finding that older patients tended to rate higher on negative features could signify enhanced representation of these symptoms in later years as the florid manifestations subside, although such an interpretation can be confirmed only by longitudinal study. As moderating variables, age and EPS were shown to have some mitigating effect on the findings in regard to motor activity and cognition, but overall did not obviate the key distinctions between positive and negative syndromes: differences were upheld on the historical, genealogical, and affective dimensions, and conclusions regarding clinical, cognitive, and motor functioning were not substantially modified. Similarly, the control variables, such as ethnic origin and chronicity of illness, had no significant bearing on any of the positive-negative indexes.

The data on the negative syndrome supported the hypothesis of premorbid impairment in cognitive maturation. Patients scoring higher on this dimension had achieved less education and were more impoverished in thought, as judged by independent clinical and psychometric procedures. On cognitive developmental tests they showed more fundamental deficiency in elementary conceptual and representational modes of thought and in the flexible use of socialised and objective perspectives. Poorer premorbid functioning by schizophrenic patients with negative features has been documented elsewhere in regard to schooling (Andreasen & Olsen, 1982; Lindenmayer *et al*, 1984; Opler *et al*, 1984; Pogue-Geile & Harrow, 1984), social relationships (Andreasen & Olsen, 1982; Pogue-Geile & Harrow, 1984), and occupational history (Andreasen & Olsen, 1982; Lindenmayer *et al*, 1984). A recent study of Pogue-Geile & Harrow (1984) indicated that "these patterns may better characterise male than female schizophrenic patients", and an independent meta-analysis by Aylward *et al* (1984) revealed that premorbid intellectual deficits are also more prevalent among males and forebode poorer heterosexual adjustment, worse clinical functioning, and sustained deficits. These observations give meaning to our finding that relate negative syndrome to lower IQ and, after adjusting for age and EPS, to male gender.

Cumulatively, these data underscore the importance of a biodevelopmental perspective on the aetiology of schizophrenia. For a subgroup characterised by a predominantly negative syndrome, it appears that pre-existing developmental failures foreshadow lifelong multimodal deficits (cf. Aylward *et al*, 1984; Pogue-Geile & Harrow, 1984). From a different perspective, high-risk studies (Marcus *et al*, 1981; Parnas *et al*, 1982; Walker & Emory, 1983)

have implicated early developmental dysfunctions as harbingers of schizophrenia and, in keeping with family and cross-fostering studies (Gottesman & Shields, 1976), have suggested a genetically mediated process. The current work, congruent with this model, has identified family loading and gender as important determinants of how the schizophrenic condition will be manifested in the chronic phase.

The proposition that negative features are associated with structural organic deficits (Crow, 1980b; Andreasen *et al.*, 1982; Seidman, 1983; Weinberger & Wyatt, 1983) was not supported in the present study. Although some of the correlations approached significance at the $P < 0.10$ level, the negative syndrome showed no clear-cut relationship to tests of perceptual-motor development (PFDT), neurological soft signs (MFD), or disturbed attention (SOA). Indirect evidence from hand dominance (Andreasen & Olsen, 1982) and season of birth (Opler *et al.*, 1984; Opler & Kay, 1985) was also not substantiated by this larger scale study.

Further investigation is needed to confirm and extend our findings. Of special importance is assessment of the stability of the positive-negative dimension across time and in relation to neuroleptic drugs. For example, the question of whether the syndromes

represent a temporary state or enduring trait has not yet been resolved. Equally compelling is the possibility of their being iatrogenic transitional or end states. Thus, it is conceivable that 'positive' schizophrenic patients are potential drug responders still in a preliminary stage of chemotherapy or not under the ideally suited neuroleptic, whereas those with a predominantly negative syndrome are the residual types who have remained deficit-ridden after the florid presentation has been controlled by medication. Since the significance of positive and negative syndromes under drug-free conditions has still to be explored, the generality of our findings remains in question. Finally, we must point out that the inclusion of 65 variables, even in a large sample study, increases the possibility of some spurious results. Although many more significant coefficients were obtained than would be expected by chance, and a distinctive pattern emerged which differentiated the positive and negative syndromes, caution must be exercised in interpreting the importance of any individual variable.

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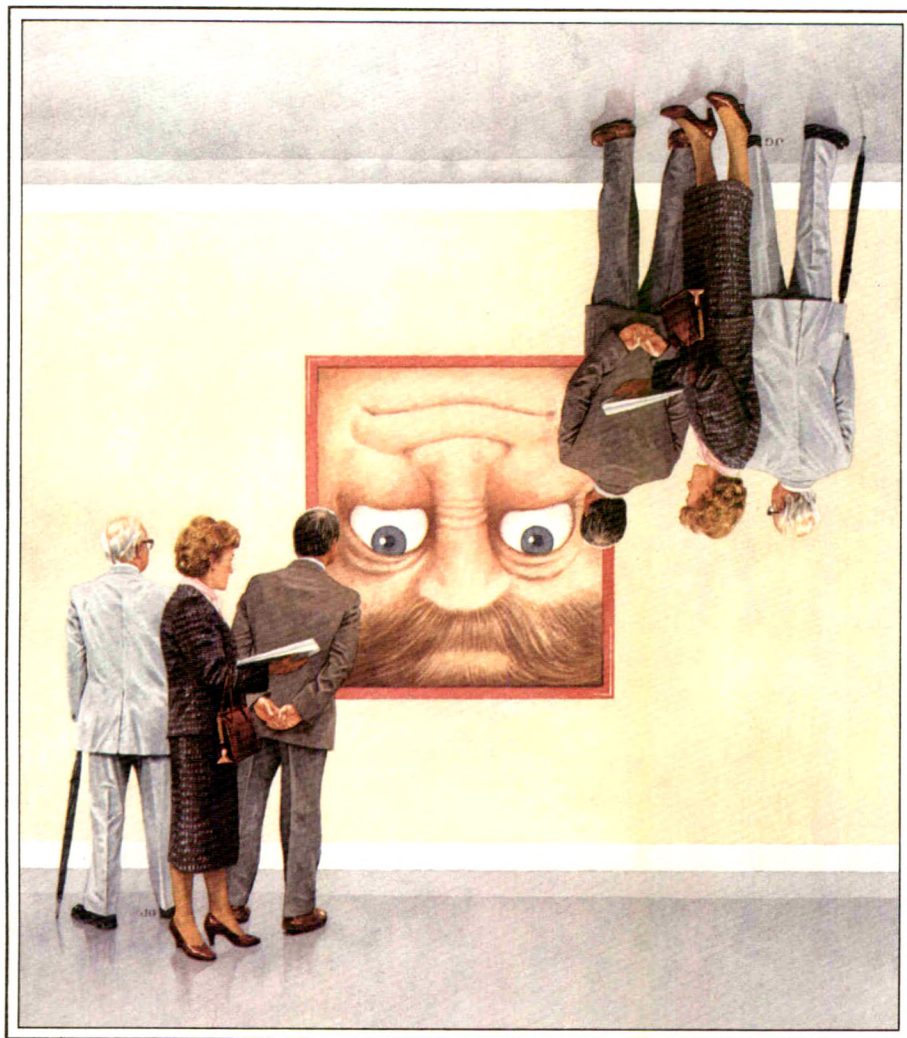
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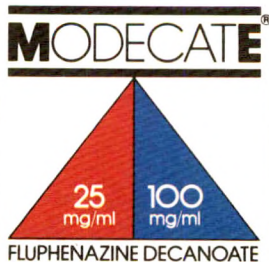
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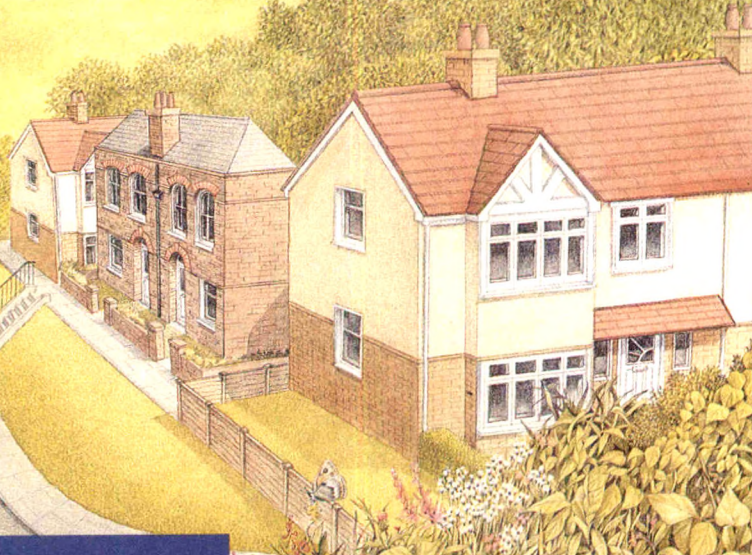
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A Haven for the Severely Disabled Within the Context of a Comprehensive Psychiatric Community Service

J. K. WING and R. FURLONG

The priorities accorded severely disabled or disturbed long-stay patients in Regional and District planning strategies vary enormously. A scheme to cater for the needs of this group is described. The scheme includes: non-stigmatising housing; a domestic regime, daytime occupation and leisure activities offering forms of enabling and caring that foster the highest possible levels of functioning; a secure home; private and peaceful outdoor space; and graduated steps towards independence that allow for the possibility of relapse. The central concept is the establishment of a Community for people with severe difficulties in making social contacts. The importance of integrating the scheme into a comprehensive District psychiatric service is emphasised.

The term 'asylum' has both benevolent and pejorative connotations, and motives for establishing sheltered institutions have usually been mixed. Lunatic asylums were established both for humanitarian reasons and because the community at large was frightened by manifestation of insanity and understood that protection worked both ways. The extraordinary strength of this combined motivation is evident from the volume of resources put into building and maintaining the county asylums. They still stand: a visible index of the capital and revenue needed to create a new system of care for mentally afflicted minorities, and a challenge for our own time. In this paper we discuss, in the context of evidence that the build-up of 'community' alternatives for the long-term mentally ill is at best half-hearted compared with the speed of rundown of the hospitals (Social Services Committee, 1985), what is to be the fate of people who need to be offered a haven.

Planning for the needs of the disabled

The concept of 'need' is based on that of 'social disablement', which in turn is derived from a concept of normal social functioning and expected quality of life. 'Disablement' is defined in terms of the degree to which an individual falls short of the level of performance that is generally expected within a given society, as interpreted by the afflicted individual or those closely connected with him or her. The more severe and general the departure from 'normal' social functioning, the more likely is agreement to be reached that social disablement is present and, potentially, that help is needed.

A full analysis of the concept of need requires consideration at three levels:

- (a) the causes of social disablement, which can be summarised under the headings of intrinsic impairment, social disadvantage and personal distress or demoralisation.
- (b) specific, effective and acceptable forms of care (including treatment, training, rehabilitation, shelter, security, and welfare) for problems defined under (a)
- (c) the service that will prevent, ameliorate or contain these problems, through the 'delivery' of specific, acceptable and economic forms of care (Wing, 1972).

The application of this kind of analysis across a district allows comprehensive 'bottom-up' planning, based on the aggregated needs of individuals. Within a geographically based national health service, planning and provision should be based on three principles—identification of individuals in need through the application of epidemiological knowledge, provision of a wide variety of service components to deliver packages of treatment or care in accordance with fluctuating need; and organisation and management that ensures integration and continuity in a system that could otherwise become fragmented (Wing, 1978).

Systematic surveys provide data that demonstrate needs for a wide variety of methods of help, and therefore a wide variety of services. The latter can be illustrated by the analogy of three stairways with landings (see Table I), each representing a major functional area of everyday life: occupational, residential and recreational. Disability in one of these areas does not necessarily (or even usually) imply disability in all. This analogy provides a reminder of the interaction between the functions of services

TABLE I
The stairway analogy. Service and needs can be likened to three stairways (columns) with landings (horizontal lines)

<i>Dependency</i>	<i>Occupation</i>	<i>Residence</i>	<i>Recreation</i>
Low	Can occupy self	Maintains own home	Own and shared activities
	Sheltered paid work	Supervised flat or lodging	Accompanied to public amenities
Moderate	Industrial therapy unit	Group home	Reserved hours e.g. at swimming pool
	Occupational therapy unit	Staffed hostel	Restaurant and social club
	High dependency day unit	High dependency hostel	Private grounds for exercise
		Sheltered Community	
High		Secure Unit	

N.B. An individual can be highly dependent in all three areas but living at home with relatives.

(movement up and down, respite on the landings, and the use of artificial aids to help people climb) and their structures, which should be subordinate to function. Towards the top of each of the stairways, individuals are able to make use of the options available and to help chart their own paths through the system. We are concerned in this paper with those with high dependency needs, who remain on the lower steps in spite of prolonged efforts to decrease the severity of their social disablement. Unless the rest of the service is in place, however, the chances of their moving towards greater independence, or of moving down according to need (rather than precipitously), will be substantially fewer.

The high-dependency group

The term 'dependency' is non-specific and provides no guidance about requirements for particular forms of service; it simply indicates a high all-round level of need for care which has hitherto usually been equated with a need for long-term residence in hospital. There are many causes of high dependency, and systematic surveys have always revealed complex causal patterns of multiple impairments, social disadvantages and adverse personal reactions. Because each individual's pattern is unique (and

often changes over time), each needs an individually oriented programme of care.

The characteristics of patients (other than those with severe mental retardation or dementia) who have recently had hospital stays of over one year, in spite of current trends to avoid such long stays, can be used to describe the wider group of people with which we are concerned (Mann & Cree, 1976). Chief among the common factors is a record of contact that stretches back long before admission. Very few have put down roots in an outside community, have recently been employed, are married and in touch with their husbands or wives, or have a home that can accept them (although most have relatives somewhere). Most are middle-aged to elderly.

The most common diagnosis is schizophrenia, but up to half have other conditions such as short-cycle manic-depressive psychoses, chronic depression, or personality disorders. Many patients have multiple disabilities such as epilepsy, mental retardation, brain damage, sensory deficits, and neurological disorders. Physical diseases of all kinds are common, and have a high frequency in the elderly. In many cases, social disablement has remained severe and intractable in spite of the fact that the quality of assessment and care has been as good as possible. This is mainly due to five factors which complicate treatment and set limits to the degree of independence that can be achieved:

- (a) risk of harm to self or others
- (b) unpredictability of behaviour and liability to relapse
- (c) poor motivation and capacity for self-management or performance of social roles
- (d) lack of insight
- (e) low public acceptability.

These factors interact with each other. Each may be assessed differently, in any particular case, by a stranger, a caring relative, a member of staff or an afflicted individual. Staff who act as gatekeepers to services have to try to assess all of these factors and allocate a degree of priority to each. The less experienced and skilled the observer, and the shorter the acquaintance with a chronically afflicted individual, the less visible many of the problems appear to be.

Those people with high dependency needs tend to present problems that are complicated by several of the five factors, acting either together or within short intervals of one another. Dangerousness is complicated by unpredictability and by lack of insight, or by a degree of insight that is sufficient to control behaviour in one setting (e.g. an out-patient clinic or at a domiciliary visit) where compulsory action could be taken, but not in another (e.g. at home with relatives) where no such power exists.

Self-harm not only includes immediate self-injury or suicide but may arise from poor self-management by reason of neglect of physical health (nutrition, personal hygiene, exercise, smoking, alcohol, exposure to cold or other dangers), and from lack of awareness of the value of medication or of attendance at a day centre. A complicating factor is public hostility due to socially embarrassing behaviour or to deterioration in the environment; for example, when a flat falls into disrepair, begins to smell and constitutes a fire hazard. A lack of motivation makes it impossible for some disabled people to imagine how problems can be overcome and leads to an apathetic acceptance of a drift into destitution (Lamb, 1980; Leach & Wing, 1980). A less obvious consequence is that the individual becomes easily exploitable.

These extremes of behaviour occur much less frequently in a well regulated and protected setting. Hence, characteristics of the environment are an important part of treatment. The question is one of how far caring environments can be prescribed for people whose lifestyle is gradually deteriorating through apathy, in a way that is unacceptable to their relatives and to the public in general. This lack of concern for one's own welfare is similar to that of long-term residents in old-fashioned mental hospitals, who were either indifferent to discharge or actively wished to stay, even when their intrinsic

impairments were not severe. Such gradually acquired negative attitudes to discharge from hospital ('institutionalism') were hypothesized to be part of a process of development of adverse self-attitudes and decreased motivation that can occur in any setting characterised by 'social poverty', and is potentiated by the cognitive deficits of several psychiatric disorders (Wing, 1964; Wing & Brown, 1970). The process is still occurring in socially impoverished 'community' settings, now that most of those at risk no longer become long-stay hospital inmates. The most readily influenced of the environmental factors, both for good and harm, is the quality of social environment provided.

The size of the high-dependency group

There is still a sizeable group of people who need security, protection or shelter in two or more of the three major areas of life, whether or not that protection is currently being offered or taken up. Calculating the size of need by extrapolating from hospital bed occupancy is problematic, because some long-term in-patients could be discharged if lower dependency facilities existed elsewhere, and some of those 'in the community' would have a better prospect of a reasonable quality of life if they were in a sheltered community.

The statistics of rundown of the 'old' long-stay group and build-up of the 'new' are best summarised in Robertson's note of guidance to regional statisticians (1981). His higher estimate (which most closely fits the trends) suggests that, if there are no radical changes in service practice, an average health district will be using 140 in-patient beds per 100 000 inhabitants by the end of 1991, of whom 80 would have stayed for a year or more. For those without a diagnosis of dementia, the long-stay rate would be 53 per 100 000 (Robertson, 1981; Wing, 1986).

The survey by Mann & Cree (1976) suggested that 34% of long-stay (1-3 years) in-patients under 65 needed further hospital care, 25% needed 'hospital-hostels', and 32% needed other types of supervised residence. One hospital in each health region in England and Wales was visited, but in nearly all cases the alternative accommodation did not exist. Dependency becomes substantially higher the longer people stay and the older they get.

Two factors, acting in opposite directions, could upset any estimate of numbers. On the one hand, the discharge of long-stay patients might accelerate; on the other, a recognition that adequate non-hospital facilities were not being provided might lead to a greater use of long-term hospital care. Neither contingency is predictable with any confidence, but

such calculations are in any case irrelevant to the questions that interest us, which are concerned with meeting need wherever it occurs.

Surveys of day attenders, hostel and group home residents in Camberwell have not demonstrated any substantial unmet need for long-term high dependency care, but the local services are generally well above average and the results cannot be regarded as representative (Wykes *et al.*, 1982). On the other hand, surveys of families known to organisations such as the National Schizophrenia Fellowship (Creer & Wing, 1974) and of single homeless people (Leach & Wing, 1980) have shown that an unmet need for a good quality of sheltered environment is not confined to hospital in-patients. Register data suggest that some Health Districts, particularly those with a declining population near the centre of conurbations, are likely to have high rates, while Districts in attractive areas with increasing populations will have low rates (Gibbons *et al.*, 1984).

Sociodemographic indices such as age, social isolation, poverty and ethnicity have important implications, as have geographical factors and local traditions in the provision of services. There can be no such thing as a national or even a regional norm. Our best estimate of 50 places per 100 000 population for an 'average' District is not intended to be taken as more than a starting point for better-informed local calculations. In particular, we urge that the fact that some (usually privileged) Districts appear to have a low order of need should not uncritically be used to set a standard for the whole country.

Appropriate caring environments

Large-C and small-c communities

The term 'community' is often used loosely, but two particular uses are common. The word is often used to create an image of a well-integrated village or neighbourhood whose residents know and care for each other, where few inhabitants feel isolated or remote from help should they need it. This would be a 'large-C' Community. 'Small-c' communities lack this cohesion. Examples are inner city areas, which are notoriously deficient in these respects. Even the residents of affluent suburbs may be preoccupied with material standards that demand a conventional normality. Moreover, rural villages may fall short of romantic expectation. A recent study of attitudes towards mentally retarded children showed that urban mothers were more likely to be favourable than village mothers (Sinson, 1985). Neighbours do not always help or even sympathise with the problems of families with a mentally disabled member. Formal systems of service delivery developed

"because the informal networks of mutual aid in local communities were manifestly incapable of meeting the kinds of personal need which arise in complex industrial societies" (Pincus, 1982). Public attitudes, as the last White Paper on services for the mentally ill pointed out, need to become more favourable before it becomes feasible to give sufficient spending priority to them (DHSS, 1975).

We share the view commonly held throughout the caring professions that severely disabled and disturbed people should, as far as possible, be helped to use public amenities and facilities, to live in ordinary houses in ordinary streets, to undertake the same activities, and to receive the same personal support from friends, relatives and neighbours as those who are physically and mentally fit. However, implicit in some influential formulations of this position is the assumption that virtually everyone, no matter how severely afflicted, can achieve an acceptable integration while dispersed in 'the community'. 'The institution' (i.e. any structure that implies a degree of segregation) is thus seen as inherently harmful.

The term 'normalisation' is often used to express the underlying thrust of this argument. From this point of view, to begin with an analysis of the causes of social disablement, particularly if this includes the concept of physical or mental impairment, is regarded as counter-productive. The term has most frequently been used in discussions of services for the mentally handicapped, and is now appearing in plans for long-term residents of mental illness hospitals. The danger in both instances is that the underlying good intention may be thwarted by the fact that becoming 'as normal as possible' (a more modest but more realistic formulation) depends on particular kinds of help from people and agencies familiar with the varied causes of social disablement, and therefore with specific needs. Trying to apply a general standard of 'normality' without starting from the concept of impairment can lead, in spite of the best intentions, to a drop in the quality of life of disabled people.

A different use of the term 'Community', explicitly with a large C, is to denote a group of people coming together because of a shared interest, in order to pursue through personal relationships and the exercise of special skills some common purpose—moral, artistic, political, or therapeutic. The Steiner Communities, for example, represent the first and last of these purposes. Few people who have visited them would deny the sense of real Community, even for those residents who are so disabled as to be incapable of understanding the full meaning of the word, and for whom it must be partially artificial. This sense is created by the beauty and simplicity of the buildings and surroundings, the shared

motivation of the staff, the significance given to each activity of daily living, and the evident quality of life of the residents. Nevertheless, each Community is an institution. There is also an inherent element of segregation. Moreover, such charitable organisations do not accept what has hitherto been the final responsibility of the National Health Service—not to select. (It must be said that the NHS does not always accept this responsibility nowadays.) People who are disruptive or socially impaired are no more welcome than they are in most local authority hostels.

However, the success of the Steiner Communities demonstrates that a semi-detached house in an urban street is not the only alternative to a hospital ward, particularly for those people who cannot (and do not) use community amenities and who are unable to create any sense of Community around themselves because their social skills (in spite of adequate efforts at training) are lacking.

Open versus custodial care

Erving Goffman (1961) introduced the concept of the 'total institution'. He argued that American state mental hospitals in the 1950s resembled other segregated communities in which inmates were isolated from the everyday life of the community such as prisons, orphanages, concentration camps, and even battleships at sea. The staff and inmates had fundamentally different points of view and perceived each other in terms of narrow, hostile stereotypes. Inmates slept, worked and played in one place, and an overall rational plan guided all behaviour. Even the smallest details, such as when an inmate should bath or go to bed, were decided by authority. Inmates were no longer looked upon as fathers, or employees, or customers, and their abilities to play social roles atrophied from misuse.

Although British mental hospitals, even then, did not much resemble this picture (for a comparison

with an American county hospital see Wing & Brown, 1970), an image of hospitals as globally and inherently custodial has stuck and the term 'institution' is now, in this context, almost exclusively pejorative. Table II illustrates the two extremes of custodial and open systems of care. Attempts to measure the degree of restrictiveness indicate substantial variation and overlap within and between care settings. Custodial care cannot be equated with hospital wards, nor open care with non-hospital alternatives (Rawlings, 1985a,b; Ryan, 1979; Wykes *et al.*, (1982). Moreover, the term 'restrictiveness', whatever the setting, carries a connotation of arbitrary authority that may be unjustified (Hewett, 1979). The proper question, in respect of particular individuals, is whether help is being given that will reduce the causes of social disablement to the minimum possible level and maintain them there. If so, needs for asylum functions will be met with the minimum segregation and dependence, always taking into account that some people need solitude more than others.

The haven concept

The idea of Haven Communities arose from two kinds of experience. One was with a hostel-ward ('hospital-hostel' or 'ward in a house') set up at the Maudsley hospital for people from Camberwell under the age of 65 who had been living in hospital wards for 1–7 years (Garety & Morris, 1984; Wing & Hailey, 1972; Wykes, 1982). The results suggested that virtually all such people could benefit from the greater personal attention and gradual introduction to responsibility that such an environment made possible. A subsequent, better controlled, evaluation of a hostel-ward in Manchester, and an uncontrolled study of one in Southampton, confirmed most of these findings (Gibbons, 1986; Goldberg *et al.*, 1985). These two hostel-wards were off-campus, and hence had to be selective; some other unit would still be

TABLE II
Custodial and open care

<i>Characteristic</i>	<i>Custodial settings</i>	<i>Open settings</i>
Power and staff attitudes	Oligarchic, authoritarian and arbitrary	Democratic; team working to clear guidelines
Boundaries	Impermeable, whether visible or not	Human scale; flexible security
Stigma	Concentrated	Diluted
Outside influences	Easily evaded; occasional scandals	Open access; elected committee, inspectorate
Milieu	Social poverty; pauperism; inactivity; unnecessary dependence	Socially rich; local standards; choice for growth

required for the most disturbed. The second experience was that of a mental hospital that (even in the late 1950s) provided a social environment that was markedly above average in quality, and where the greatest priority and prestige was accorded the long-stay wards (Freudenberg, 1970). From the statistics given earlier, it is clear that several such houses would be needed in many Health Districts.

Though there are a few exceptions, non-NHS alternatives currently tend to reject the most severely disabled and disturbed people, and therefore do not come into consideration. We rule out the use of District General Hospitals as sites for communal living; they are busy, crowded, and lacking in space and opportunities for occupation and recreation. The orientation of the wards (which are not, in any case, satisfactory living environments) is not conducive to gradual healing over years; and often there is not a tree or blade of grass to be seen.

This leaves three possibilities: use of part of a mental hospital site, if conveniently situated near the District it serves and managerially independent; conversion of some other equally convenient site within the District; and dispersal of the component elements throughout the District. The first two of these are in most respects similar.

The haven project

An opportunity to set up a first Haven Community has arisen in the NE Thames Health Region as part of plans to close the large Friern Hospital and to use part of the site to serve the borough and Health District of Haringey. The underlying principles described below could be adapted to other situations.

Candidates for the Haven will be adult Haringey residents of all ages (excluding those with severe mental retardation, dementia or primary addictions) who have spent more than one but less than fifteen years in hospital, are chronically mentally disabled or disturbed in behaviour, and are seriously impaired in ability to care for themselves. Initially, 50 places will be provided to serve West Haringey (population 110 000).

Range and style of housing

Based on a survey of Friern patients eligible for the Haven Community, we estimate that a core group of four houses will be needed for those needing most care and shelter. One house (or hostel) with twelve residents will serve as the heart and resource centre of the Community. It will provide for patients who need frequent short readmissions and younger persistently disturbed people. The three other core houses will

each have six residents: one will cater for the long-term severely disabled with physical problems such as severe epilepsy or Huntington's Chorea, another will provide treatment and containment for those who tend, if unsupervised, to wander without regard to common danger, and the third will provide for frail elderly people with long-standing functional psychiatric disorders. The remaining 20 residents will live in a range of houses and flatlets with a lower degree of supervision. They will provide a gently graduated range of facilities, so that maximum independence can be developed and maintained in those whose disorders necessitate lengthy care, e.g. those with unpredictable behaviour, those detained on Home Office regulations and those with major dependency problems.

Staff offices, an occupational therapy store, a visitors' room, and recreation rooms with a beverage bar for the whole Community will be provided adjacent to the central house. Staff will use the same accommodation as residents. Each resident will have a personal bed-sitting room, and each house will have the usual domestic offices and living arrangements. Residents will participate in running their own houses, including cooking, cleaning and repairs. Each house will be allocated a weekly or monthly budget, so that the responsibilities of domestic management can gradually be acquired.

The choice of site for this group of houses is important. It should be clearly separated from hospital buildings, and have its own identity and access from a public road. Fortunately, the Halliwick part of the Friern estate provides a most attractive area for this development, and recently built local housing is pleasant enough to serve as model so that the Haven can merge into the background of the community. All houses could readily be converted for other uses if, in due course, they became redundant.

In addition, linked to the Haven but scattered among the local housing estates would be peripheral group homes and supervised apartments, set up in association with the local authority or charitable organisations, in order to provide for graduates who still need various degrees of Haven support and also for people who are a rung or two up the ladder but highly vulnerable to relapse. This arrangement has proved successful in connection with the first hostel-ward at the Maudsley Hospital (Wykes, 1982).

Administration

The Community would be administered from the central hostel and offices, with a single manager who would be part of the multi-disciplinary team

(including nursing, medical, occupational therapists, psychology and social work staff) responsible for running the Haven. The central hostel would be staffed day and night by trained and experienced staff, who would also be available to help when problems arose elsewhere in the Community. The other three core houses would also need some cover at night. Day-to-day running would be left to the houses themselves. The lower dependency houses would function with the help of a housekeeper during daytime hours and call buttons for emergencies at night or weekends. Staff would be aware of situations where problems could arise, and organise their rounds accordingly.

Since the Haven Community will be serving a designated population it is essential that it has clearly established links with other parts of the district services. In particular, it must provide an integrated service with those units that form steps, on the three 'stairways' described earlier, that are within reach of Haven members. Rehabilitation for this group consists of numerous small advances separated by pauses for consolidation. Continuity of care is therefore vital. Ease of movement into and out of the Community is essential. Haven staff would all take part in off-site activities both in following up and supporting ex-members and in developing links with people who might need their help in future. Close contact would therefore be kept with families, relatives' organisations and other community groups, so that early intervention can be achieved when necessary. Co-ordination will also be required with acute hospital services, psychogeriatric facilities, and primary and secondary medical services. Staff will be selected because of their interest in and aptitude for creating a living Community in partnership with residents and families. Because of its innovative aspects, the Haven Community would be an ideal setting for staff training.

Occupation and day care

In part, Haven members will be occupied within the Community, and domestic experience is part of the function of the houses. Those who have reached retiring age may not wish to engage in many formal activities. However, the exercise of all one's faculties is a necessary condition of physical and mental health, and Community members will be given a choice of opportunities. The Health District will be served by industrial and occupational day units, both in and away from the Friern site, which will facilitate continuity and flexibility of occupation for those members who can make use of these opportunities. A market garden is also planned. At the other end of

the spectrum, an intensive day-care facility will be required for those people who need a degree of security, usually during a prolonged day-care period from breakfast to supper time, including weekends. Disturbed behaviour is usually less evident in the later evening and at night. A small unit will be attached to the hostel to cater for this need, and to provide more organised domestic re-training to enable members to retain their daily living skills.

A further type of provision is also being considered. As part of the outreach links to the wider community, it is proposed to set up a charitable trust in association with local church and citizens groups to establish and administer a community centre that will serve a membership drawn from the local neighbourhood, Haven residents, relatives' organisations and staff. The centre would include meeting, recreation and dining rooms, a kitchen and bar, and sheltered workshops, such as printing and upholstery, that could provide some revenue and be of general use. In due course, an arts centre would be an appropriate addition.

Opportunities for recreation will also be needed by people who cannot make use of public amenities or who, because of their slow or odd or indecisive behaviour, may not be welcomed. There will be space where people can wander in private and where oddities of gesture or demeanour will not incur arrest or public ridicule. Ideally, some amenities would be created that were shared with the locality.

The Friern estate

The fate of the large Friern estate has yet to be decided, although approximately one third (known as present as the Halliwick site) is likely to be retained for Haringey District Health Authority and 'supra-District' functions. The rest will probably be sold and, in view of the Secretary of State's assurance (*The Times*, 17 February, 1986), the money thus raised will be earmarked for the further development of services for the mentally ill. The main hospital building is in poor repair, and it is difficult to see what purpose most of it could serve once better alternatives had been provided. The Regional Health Authority has given assurance that priority will be given to the development of services for the most severely and chronically disabled—including the Haven Community for which funds have already been allocated. We strongly support this point of view, and would like to see it adopted by other Health Regions.

The opportunities offered for improving the quality of life of mentally disabled people by the redevelopment of the Friern estate are tremendous, and it would be a tragedy if they were missed. The

overriding principle of planning should be to regard the site as a precious public amenity and to open its parkland to wider community use. Any commercial development should be limited to the provision of facilities that would attract the public onto the site. Possibilities include a centre for indoor and outdoor sports and leisure activities, a shopping complex, an arts centre, and a restaurant. Some industrial development might be considered if this included provision for sheltered work. The scale of any new building should not take up much more space than the present main hospital complex. The central administrative block, which has a certain Victorian charm and is listed Grade II, should be preserved and converted for other uses, including a museum and archive. However, there would be room for a small housing estate, which could incorporate some houses for staff and ex-patients. Local new housing is already of a good standard and appearance. Imagination and a coherent architectural plan is required to combine visual attractiveness with convenience of access and use.

The area set aside for health services would include substantial private areas, but there is no reason why the public should not use paths that crossed it. Similarly, members of the Haven Community and other users of the Halliwick site would be able to benefit from the facilities in 'Friern Barnet Park' as members of the public in their own right.

Hospitals such as Friern Barnet, which are in the middle of busy urban areas, have achieved over the years a status in the neighbourhood that is not entirely unfavourable to their residents. The sight of patients using local shops and pubs and strolling round local streets is familiar, generally accepted and, in certain respects, welcomed. Such stigma as is due to the inappropriate attribution of characteristics such as unpredictability and violence is likely to be diluted by bringing 'Community' and 'community' together in the way we suggest. The sympathies of those who are able to appreciate something of the problems caused by mental disablement could be engaged. On the other hand, geographical dispersal in small units is most unlikely to dilute the stigma that is aroused by truly socially embarrassing or unacceptable behaviour and could actually provide a series of nuclei around which stigma became concentrated.

A different kind of solution to the one proposed here has been suggested for the Claybury Hospital site, which is also in the NE Thames Region (*Architectural Review*, January 1986; Burrell, 1985). The proposal is to transform "the existing hospitals into urban quarters" that are fully integrated into the surrounding area. Ward blocks will be converted

into shared flats and houses. We welcome this idea, and hope that there will be competition throughout the country to develop imaginative and practical solutions to the varied planning problems presented in different localities.

The context of district planning

The three principles of Health District planning described earlier can be applied to the case of any particular group of people in need. The first principle is identification of individuals living within a defined geographical area of responsibility who are in need of various forms of care. This includes people who have only transient geographical links, or who drift from one place to another, never establishing 'residential rights' in any one location. Resources should be allocated to Health Districts and to local authorities on the basis of local requirement, including the needs of those who are homeless. We argue that responsibility for the most severely disabled and disturbed group (of which those we have discussed form a substantial part) should be given high priority rather than left as a residual group to be provided for only when all other services are in place. This means, as the Social Services Committee pointed out (1985), that the budget currently available to this group should be identified and should not be used for any other purpose unless there is clearly a surplus. This is most unlikely to be the case during the next decade or two.

The second principle—flexible, comprehensive and varied service provision—means that members of a Haven Community will have the opportunity to move towards independence by easy steps and stages as and when improvement occurs. By the same token, access down the 'stairways' to haven membership will also be simplified.

The third principle—sound management and organisation—ensures the maximum degree of autonomy for individual components (such as the Haven houses) of a District service, while maintaining an overall operating policy that ensures continuity of care and smooth working relationships between all units. Good administration will also ensure that staff numbers and staff training are appropriate to cope with the levels of difficulty likely to be encountered. Ideally, one executive body, with an identified budget, should eventually be made responsible for co-ordinating all care for the mentally disabled. For the moment, the District Health Authority should be regarded as the lead authority and given the duty of initiating and maintaining a co-ordinated service that will meet the needs of the population it serves.

Evaluation

The NE Thames Regional Health Authority has had the foresight to set up the Mental Health Services Evaluation Committee and to support a small research team for assessment of psychiatric services based at Friern, under the honorary directorship or Dr Julian Leff. The value of the Haven Community

will be independently assessed in comparison with that of other, more dispersed, core and cluster schemes and in the context of more general evaluation of the rundown of Friern and Claybury hospitals and the build-up of various patterns of new community services, particularly in the boroughs of Islington and Haringey. It is greatly to be hoped that other Health Regions will follow this example.

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Providing Information to Relatives About Schizophrenia: Some Comments

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There has recently been an increased interest in providing relatives of schizophrenic patients with information about the illness and how to manage the patient at home, due partly to the development of psychosocial intervention programmes (which frequently include an educational component) and a consumer demand for such information in an era of community care. Studies which describe the effects of such educational programmes are reviewed, and two different models of information acquisition are contrasted. These models have different implications, in terms of outcome measures, on how educational programmes should be evaluated. Guidelines are offered for practitioners wishing to give information to relatives.

A general rise in public interest in health and in desire for knowledge about illness (Fry, 1984) has coincided with increased recognition of the importance of social factors in the course of schizophrenia, and with the beginning of prophylactic psychosocial interventions to try to reduce relapse rates. The development of expressed emotion (EE) ratings (Vaughn & Leff, 1976a) has allowed a dimension of social stress to be operationalised and quantified, and its relationship with post-discharge relapse rates to be investigated. The finding that patients returning to a relative rated as showing high EE have higher relapse rates than those discharged to low-EE homes has been consistently reported (Brown *et al*, 1972; Vaughn & Leff, 1976b; Vaughn *et al*, 1984). These results gave impetus to attempts to reduce the risk of relapse by psychological intervention (see Barrowclough & Tarrier (1984) for a review), but a lengthy training period is required for the administration of the Camberwell Family Interview (CFI) and for rating the audiotaped interviews on which the EE concept is based. Moreover, the amount of time required to administer and rate the CFI may restrict its clinical use.

An integral part of the intervention process has been a knowledge or information component, by which patients are provided with information about the diagnosis, symptomatology, aetiology, treatment, and course of schizophrenia (e.g. Leff *et al*, 1982; Falloon *et al*, 1982; Anderson *et al*, 1981), and this might be integrated more easily into clinical practice. However, this necessitates further investigation into how information about schizophrenia might best be presented to relatives and patients, and what effect this information has on them.

The burden on relatives caring for a person with schizophrenia is well known (Creer & Wing, 1974), yet many patients with significant disability have little contact with psychiatric services (Johnstone *et al*, 1984), and in many cases families and relatives are the real primary care rehabilitative agents for the chronic patient. With the increased impetus towards community-based services, families are more likely to be taking on this role, often by default, and at a time when other environmental factors (e.g. economic trends and unemployment) probably make this role increasingly difficult (Barrowclough *et al*, 1984). However, relatives are often not actively supported in this work (Lamb & Oliphant, 1978), and may even be blamed for the illness itself (Appleton, 1974). How information about the nature, course, treatment, and management of schizophrenia could assist relatives to help to rehabilitate patients needs to be examined.

Presentation of content of the information

In different studies, the method of delivery of such information has varied from lecture-style presentations (Leff *et al*, 1982) to combining individual presentations with written material (Falloon *et al*, 1982) and video presentations (Smith, 1984). Information has been presented to individual families (Leff *et al*, 1982; Falloon *et al*, 1982), to groups of families (Smith, 1984), in a workshop format (Anderson *et al*, 1981), and by post (Smith, 1984). However, the general lack of evaluation of different modes of presentation led Smith to comment that "the means of delivery has tended to be fairly arbitrary, seemingly based on the unwritten assumption that information is information irrespective of how it is given".

While the effectiveness of different modes of presentation

needs further investigation, the accessibility of the information given to the recipient must be assured, and not assumed. For example, when a reading analysis (Flesch, 1948) was performed on the booklet used in the Falloon *et al.* (1982) study, it was found that only the upper 24% of the population would be able to comprehend it, functioning at their highest reading level. This is not a criticism of the latter study; Falloon *et al.* recognised that some patients and relatives were unable to read the handouts, and the chief communication modes used were oral presentation and discussion (McGill *et al.*, 1983). Moreover, the educational sessions were part of an intensive family intervention. The issue of readability of information becomes more pertinent in cases where brief educational interventions are utilised, or where there is heavy reliance on relatives utilising written material (e.g. Smith, 1984).

Since research into doctor-patient communication suggests that only a few items of information can be retained by recipients (Ley, 1979), it may be necessary to be more selective in the information put over. It is therefore important to identify the relevant aspects of schizophrenia for this purpose.

Models of information-giving

The rationale for providing information can be argued from two points of view. Firstly, from a practical standpoint: that such information from health care professionals will either alleviate an undesirable state (e.g. anxiety, confusion, or distress) or encourage positive and desirable behaviour (e.g. health-promoting behaviour or compliance with medical treatment) on the part of the recipient. Secondly, from a moral basis: that the consumer or recipient should, as a basic right, have access to information if he desires it, and in a form that he will understand. Both of these points are open to debate, but it is the first that concerns us here.

To investigate the optimal means of providing information, two models of the practical aspects of giving information can be applied. The basic premise of the simple deficit model is that lack of information plays a causative role in producing behaviour detrimental to health; providing relevant information, and so eliminating the deficit, would also eliminate (at least partially) the undesirable behaviour and consequent health risk. Such a deficit model can be applied to the EE concept and data: Vaughn & Leff (1981) found that relatives who were rated as high-EE tended to view the bizarre and difficult behaviour of the patient as being deliberate or at least controllable; in contrast, low-EE relatives believed that this behaviour was due to a legitimate illness which the patient was not able to control. Hence the prevalence of criticism and hostility found in high-EE relatives could be explained by the attribution of personal control and causation to the patients themselves rather than to the illness, but high ratings of emotional over-involvement found in some high-EE relatives are less easily explained by this model. The deficit model would predict that providing adequate information about schizophrenia and its consequences would alter the attributions about the causality of the patient's behaviour which are made by the high-EE relative, and thus result in a

reduction in criticism and hostility. Hence, Berkowitz *et al.* (1984) commented: "the reasoning was that if high-EE relatives could learn more about schizophrenia, they might begin to understand that some of the patient's bizarre or difficult behaviour could be attributed to the schizophrenic condition. Such understanding could be one of the apparently important changes necessary for the change from high to low EE". These authors consider information to be only one aspect of the necessary process for change, but it is an important goal of their intervention, which aims to change relatives from high to low-EE status, and hence reduce the patient's risk of relapse. Similarly, McGill *et al.* (1983) hypothesised that "family members can effectively enhance their knowledge of this illness by participating in a brief educational intervention . . . and that such knowledge results in better understanding and care from the person with schizophrenia". They believed that high levels of EE can be due to lack of knowledge about the illness, and that this may sometimes be due to an unwillingness of mental health professionals to educate relatives.

A second, more complex model—interaction—can also be applied to giving information; it draws on concepts used by medical sociologists and anthropologists to discriminate between the objective and subjective aspects of disease. The distinction between 'disease' and 'illness' has often been made; both are conceptual models to explain certain phenomena. The former, a paradigm of medicine, refers to an 'objective' pathology in structure or function of body organs or systems (Eisenberg, 1977). 'Illness' refers to the subjective effects on the patient having the disease; it is therefore the patient's perception of the disease and its consequences, and will be influenced by individual, environmental, social, and cultural variables. Helman (1981) points out that each patient has his own lay model of sickness, and that "such models, even if based on scientifically false premises, can have an internal logic and coherence and should be taken seriously by the clinician as they are the patient's way of trying to make sense of, and deal with, their ill-health in terms of their own reality". It would seem that the consumer may develop a cognitive set which influences his behaviour and the assimilation of new information.

The interaction model therefore postulates that during exchange of information, the health care professional may assume a disease model of the pathology, whereas the patient will assume an individualised illness model. Information given to the patient will be assimilated, organised, and possibly rejected on the basis of the illness model held by the patient, and not on that of the disease model, even if the patient adopts pseudo-scientific terminology. The use of lay models of sickness by the patient and his relatives is more likely in psychiatric illness, where symptoms are often manifest as changes in behaviour (verbal and overt), as opposed to physical signs of pathology.

The adoption of either a deficit or interaction model will affect the way information is communicated, as well as the goals of such provision, and probably the satisfaction and compliance of the recipient.

Applying the models to the studies

Although a number of recent studies concerning relatives

and schizophrenia incorporated an educational and informational component in their intervention, there are few published reports on the effectiveness of the education itself. However, it would seem pertinent for future work to fit what data are available to these two models.

Several studies (Berkowitz *et al*, 1984; McGill *et al*, 1983; Smith, 1984) have demonstrated that relatives receiving educational packages or components about schizophrenia do acquire information; how much is acquired, relative to the amount of information given, is of interest to the deficit/interaction debate. Berkowitz *et al*, (1984) report that their criteria for acquisition had to be set at a very low level, and that "the absence of any knowledge of aetiology is quite striking, and the tendency to retain their own version of causes of the illness is consistent across all groups". This calls into question the appropriateness of the deficit model, and suggests that it is unable to account for all the data. Further support comes from Falloon *et al*, (1981), in an early study looking at behavioural family therapy with three young men at high risk of relapse and their families. After 25 family sessions, of which the initial ten were education seminars, one mother "had not understood the rationale for continued maintenance medication and was encouraging her son to take a proprietary tonic instead". The interaction model would predict that some relatives might be very resistant to changing their existing lay conceptualisation of the illness.

Are relatives critical, hostile, or over-involved due to a lack of information, as the deficit model suggests? The fact that Berkowitz *et al* found little difference between information known about schizophrenia at baseline between relatives who were rated high- or low-EE does not support this hypothesis. Does information eliminate 'undesirable' behaviour? In the case of high-EE relatives, this would involve the study of whether those relatives who acquired information about the illness and its management consequently changed their behaviour in relation to the patient. If this were so, how much did the information-giving contribute to the success of reducing relapse rates in the psychosocial intervention? If a direct relationship could be demonstrated between acquisition of information, relative's behaviour, and the patient's symptomatology or relapse, there would be strong support for a deficit model. However, the relatives' resistance to changing their views suggests that the relationships between the variables is more complex, an investigation of these relationships would require an analysis of a wider range of variables than information acquisition *per se*.

In Smith's (1984) study, attention was paid to a wider number of variables. Patients who were living with their families in the community were randomly allocated to one of two groups; families in one group met on four weekly occasions with others, and were provided with information about schizophrenia through verbal presentation, discussion, written handouts, and video. A second group received information in the form of written material through the post. Both groups had to complete homework exercises. Besides attempting to assess the acquisition of information, Smith also attempted to look at the consequences of giving information on various patient and relative variables. Like Berkowitz *et al* (1984) and McGill *et al* (1983), she found

that both groups significantly increased their knowledge concerning schizophrenia, and both showed a decrease in measures of fear and worry. Relatives in both groups demonstrated a decrease in scores of the symptom rating test (Kellner & Sheffield, 1973) after education, but no change in perceived burden of the patient, and there were no changes in patients' symptomatology or disturbance. Interestingly, and contrary to a deficit model, education did not result in a reduction in criticism (although this measure is not the same as critical comments assessed in EE), and the reduction in fear and symptom levels in relatives was independent of information acquired about schizophrenia. Thus, although the provision of information may result in benefits to the relatives, even when provided with such minimal professional contact as can be given through the post, it does not appear that this benefit results directly from information acquisition. However, the method of recruitment of this sample of patients was different from other studies, so they may not be an equivalent population.

Implications

It has been demonstrated that the simple deficit model cannot account for the available data in the studies reported, and that the interaction model might be more profitably adopted. This will affect the way in which information is communicated: most obviously, there will be a need to tailor the information to the individual family case, rather than focusing on psychopathology in general. Even more so than in general medicine, there will be the need to simplify and reduce the amount of information presented.

The goals of the informational input will also need to be examined. The acquisition of information may have little impact on the individualised illness model that the relative holds, and consequently little effect on their behaviour. Evaluation of the amount of information recalled may not be the most important variable: much closer attention will need to be paid to the illness models of relatives, behaviour of information recipients (family members in this case) in relation to the patient, and patient outcome variables.

Assessing the outcomes of information-giving

The behavioural literature of staff training is useful in looking at the effects of education, because there are similarities in using families as rehabilitative agents and in teaching behavioural techniques to front line staff working with client groups who have severe chronic problems. Ziarnik & Bernstein (1982) have outlined five different outcome measures which have been used to evaluate staff training: subjective (what trainees report); cognitive (what knowledge is gained); behavioural (how training is applied); client-centred (the effects on the client); and organisational (the effects on the organisation). A cautionary parallel is that although staff training often implicitly adopts a deficit model, there is little unequivocal evidence that such training procedures produce stable changes in staff behaviour (Ziarnik & Bernstein, 1982; Woods & Cullen, 1983). Most evaluations of staff training have only examined the first two

outcomes (subjective and cognitive), and these measures may have little effect on the more meaningful outcomes of behaviour change in staff and any resultant client improvement or progress. This classification might usefully be applied to outcome measures evaluating the effect of giving relatives information about schizophrenia.

Subjective. i.e. relatives' reported feelings about the provision of information. Measures could include self-reports of satisfaction about the content, form, or presentation of material, as well as measures of any non-specific effects that relatives attributed to the service, such as reassurance or support; the latter might include measures of mood or anxiety.

Cognitive: i.e. measures of knowledge of information about schizophrenia acquired by relatives as a result of the information-giving exercise. Measures would include the amount of technical or academic information gained, e.g. the symptomatology and causes of schizophrenia. According to the interaction theory, more important measures would include those tapping the individualised illness model of the relatives: their attitudes, beliefs, and attributions about the cause and consequences of the illness as it affects their family member

Behavioural: i.e. any changes in the behaviour of the individual family members in relation to the patient as a result of the information received. The aspects of behaviour to be measured would be determined by the theoretical orientation of the researchers or practitioners, although the relationship between information, acquisition, and compliance with treatments may be of general interest. The latter might include measures of relatives' participation in medication and therapy compliance. Those seeking to reduce EE might attempt to measure aspects of behaviour directly related to its concept, such as behaviour related to criticism or hostility (arguments, punishments) or to emotional over-involvement (intrusiveness, self-sacrifice, dependency-maintaining behaviour). Conversely, increases in positive behaviour such as strategies to avoid criticism (reduction in contact time, problem-solving discussions) or independence-encouraging behaviour (prompting of self-care and independent social behaviour) should also be measured, to avoid concentrating exclusively on negative aspects of the situation. Direct observational measures of such behaviour in 'real' life would be full of practical and methodological problems. Reliance on the reported behaviour of the relatives might be necessary, increasing the reliability of self-reports with those from other observers (possibly the patient or other family members). Alternatively, behaviour observed and measured in more artificial laboratory conditions and correlated with EE measures might be used (Kuipers *et al.*, 1983).

Client-centred i.e. the measurement of patient variables, including the frequency of relapse and hospital admission, along with reduction in symptomatology and the real and

perceived reduction in 'problem' behaviours. More positive measures might also be included, such as increases in independent functioning.

Organisational. In the case of more than one relative or significant household member, this might refer to changes in significant family interaction variables. As with measures of relatives' behaviour, what was identified as 'significant' would be determined by both the theoretical and empirical findings. The use of applied behavioural analysis as an assessment tool might be useful in identifying functional relationships between family members' behaviour which resulted in outcomes detrimental to the patient (see Goldiamond (1974) and Owens & Ashcroft (1982) for a discussion of functional analysis). Alternatively, interaction between family members which were correlated with patient prognosis or EE might be used (e.g. problem-solving, Falloon *et al.*, 1982).

The use of the multiple outcomes framework in association with the interaction model may be helpful in evaluating attempts to give relatives information; for example, the acquisition of purely academic knowledge about schizophrenia in general may have little relationship to any of the other outcome measures. Several reasons could account for this. A relative might acquire all the facts presented, but reject the idea that they appertain to his family member; or a relative might believe that the patient has some other condition (in our experience including depression, drug addiction, or no problems at all). Alternatively, the academic knowledge presented or acquired may have no relevance to desired change in behaviour or consequent progress of the patient. In the cases of both rejection and irrelevance, positive changes in the relative's behaviour or in client-centred or family variables would not be expected, but with irrelevance subjective gains from the non-specific effects of service contact might be found. In the case of rejection some relatives might express dissatisfaction, since the disease model presented was at odds with the illness model (or non-illness model) held.

Attitudes, beliefs, and attributions about the illness may bear more relationship to relatives' behaviour than may academic or technical knowledge. The evidence from the EE studies would support this hypothesis: for instance, a relative may be unaware of the name of the disease or its diagnostic symptoms, but may believe that the patient has a legitimate illness, and attribute whatever the person does when ill to reasons beyond their control, and consequently may act in a supportive manner. Conversely, a relative may believe that the patient hears voices, but think that this phenomenon is within the person's control; although 'knowing' one symptom of the disease model, this may not influence the relative's consequent behaviour. Alternatively, understanding that the patient is suffering from an illness may contribute to higher levels of over-involvement, for example over-protective or self-sacrificing behaviour.

Guidelines

The study of how best to present information to relatives about schizophrenia has a relatively short

history, but the interaction model and the use of multiple outcome variables may provide fruitful areas for future work. Meanwhile, on the basis of existing studies and the theoretical discussion presented here, the following guidelines are suggested for practitioners wishing to embark on giving information to relatives.

1. There is a need to assess and take into account both patients' and relatives' perceptions of the illness, prior to embarking on the task of giving information. If we adopt the interaction model, then we must take the view that the information is not given in a vacuum. This will involve probing patients and relatives about their ideas as to the cause and consequences of the illness, as well as their views on the current treatments received and opinions as to how its consequences are best managed in the home environment.

2. There is a need to accept the individualised illness model, which involves taking the views of patients and relatives seriously, while presenting alternative explanations and ways of coping. It will also involve presenting information about the particular patient's symptomatology, rather than focusing too broadly on psychopathology in general.

3. There is a need to be selective about the information which is to be conveyed. Since previous studies of doctor-patient communication showed that only a small number of items of information are remembered by patients, we should concentrate on those which are hypothesised to be most useful in assisting patients and relatives to cope with the problems caused by the illness and to comply with medical treatments. Providing simple, easy-to-read written information about these facts may facilitate learning.

4. Given that we are dealing with individualised illness models, shaped by many complex personal, environmental, and social variables, it should not be

assumed that everyone will assimilate the information at the same rate. Consequently, perceptions of the illness must be reassessed routinely, following the exchange of information. In this way, patients and relatives whose views about the illness are most resistant to change and whose consequent behaviour may be most potentially detrimental to the patient might be identified, and offered further help, information, and alternative coping strategies.

5. The interaction model would predict that the longer the duration of the illness, the more time and opportunity there has been for a personalised lay view of that illness to have become established. This will particularly be the case where no 'professional' view has been offered as an alternative. Consequently, the sooner information is given after the illness begins, the more likely are professionals to influence individualised illness models.

6. The consistent results of the measure of EE in research on relapse rates has often led to requests for some type of clinical application of this measure. However, because of the practical problems of using this lengthy interview and rating method, it may be more appropriate to look for a clinical methodology based around an assessment of the relative's knowledge of schizophrenia, using broad outcome variables. By eliciting the relative's attitudes, beliefs, and attributions concerning the illness and their consequent behaviours in interactions with the patients, an assessment of the relatives' existing coping strategies could be made. Development of this type of approach might be an appropriate and clinically useful application of the concept of expressed emotion.

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Delineating Social Phobia

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The natural history—including psychiatric symptoms, precipitating factors, onset and course of illness, and personality characteristics—of 47 social phobics, 80 agoraphobics, and 72 simple phobics was examined. The social phobia group differed from the agoraphobia group by having a lower mean age, fewer females and married members, and a higher educational and occupational status. They were less fearful generally, less obsessive, and less likely to follow a fluctuating or phasic course. There was overlap between the two groups with regard to main phobias, and they were similar with regard to adjacent symptomatology. Both the social and agoraphobia groups differed in similar and significant ways from simple phobics.

DSM-III (American Psychiatric Association, 1980) lists three criteria for the diagnosis of social phobia:

- (a) a persistent, irrational fear of, and compelling desire to avoid, a situation in which the individual is exposed to possible scrutiny by others, and fears that he or she may act in a way that will be humiliating or embarrassing
- (b) significant distress because of the disturbance and recognition by the individual that his or her fear is excessive or unreasonable
- (c) not due to another mental disorder, such as major depression or avoidant personality disorder.

In both DSM-III and ICD-9 (Commission on Professional and Hospital Activities, 1978), social phobia and agoraphobia (with and without panic attacks) are the only two phobic conditions accorded diagnostic categories separate from the general diagnosis of simple phobia. Before the third revision, the DSM included only one phobic disorder: phobic neurosis.

Theoretically one can develop a phobia to any object or situation (Mowrer, 1947), but the observed range of phobic objects is distinctly limited. The practice, until recently, of classifying each phobia on the basis of the feared situation and cataloguing each by adding the suffix *-phobia* to a Latin or Greek word denoting the thing feared, prevented the recognition that certain fears cluster together. However, if it can be shown that a certain cluster of phobias systematically and consistently differs from others in terms of aetiology, other symptomatology, age of onset, course and response to treatment, then a separate diagnostic category can be justified. Such is the case with the DSM-III sub-category of agoraphobia.

That agoraphobia differs fundamentally from other phobias is well documented. Most agoraphobic patients are women, and may also suffer from other phobias, have spontaneous panic attacks, depression, depersonalisation and mild obsessions. Snaith (1968) compared 27 agoraphobic patients with 21 patients suffering from either social or specific miscellaneous phobias: those suffering from agoraphobia were more anxious, their illness more often ran a remitting course, and their distribution of phobias was markedly different from the patients with discrete phobias. Factor analyses of the self-report data of phobic patients have consistently yielded a distinct factor of agoraphobia (Marks, 1967; Hallam & Hafner, 1978; Arrindell, 1980).

The evidence for a category of social phobia separate from both agoraphobia (with mild social phobias as part of the syndrome) and other simple phobias is less substantial. Marks (1969) assigned a separate category to social phobia 11 years before DSM-III, but the data he describes to define the category fail to distinguish social phobia from agoraphobia. Drawing on the results of six published and two unpublished studies (Gelder *et al*, 1967; Kelly, 1966; Lader, 1966; Lader & Wing, 1967; Marks & Gelder, 1966; Martin *et al*, 1969), Marks compared agoraphobics, social phobics, animal phobics, miscellaneous specific phobics, and illness phobics on 11 variables (percentage of women, onset age, treatment age, overt anxiety, modified Cornell Index, neuroticism, extroversion, galvanic skin response (GSR) spontaneous fluctuations, GSR habituation rate, resting forearm blood flow, and eye-blink conditioned response acquisition). The social phobics differed significantly from the animal phobics on six of these variables (onset age, overt anxiety,

modified Cornell Index, neuroticism, GSR spontaneous fluctuations, and GSR habituation rate), but did not differ significantly from the agoraphobics on any of the variables. Marks (1969) concluded cautiously: "We need to know more about social phobics before definitely classifying them on their own". Amies *et al* (1983), on the other hand, compared a group of social phobic patients with a group of agoraphobics on demographic, clinical, and questionnaire data, and concluded that significant differences on important variables (age, sex, social class, age of onset, main phobic complaint, and type of autonomic response) justified the recognition of two distinct syndromes. They conceded, however, that 50% of the agoraphobic patients experienced anxiety in social situations and that 25% of social phobics experienced anxiety in agoraphobic situations, in both cases at significantly lower intensities; the two groups did not differ with respect to their symptoms (e.g. anxiety, depression, obsessional symptoms).

Method

The 199 patients in the present study were consecutive referrals, primarily from other psychiatrists, over a 16-year period (1965–1980) for behavioural treatment of phobia. Following the establishment of our behaviour therapy unit in 1965, patients who had not responded to other treatment were referred to the senior author. Diagnosis was confirmed or clarified during a semi-structured clinical interview. 80 patients were diagnosed as agoraphobic, defined as 'fear of leaving home, of being alone at home or on the street, in crowds, of travelling by car, bus or train' (Solyom *et al*, 1972); 47 were diagnosed as suffering from social phobia, defined as a fear of performing (e.g. speaking, writing, eating, using a public washroom) in the presence of others. By emphasising fear of performing, we distinguish social phobia from fear of crowds, where the patient is anonymous—a classic symptom of agoraphobia. Social phobics may have avoided social situation for fear of blushing, speaking, trembling, or panic attacks. However, the 72 simple phobics suffered from a single but life-disrupting fear—animals, heights, etc. (e.g. an estate agent, afraid of dogs, could not function adequately at work; a snake phobic could not watch TV for fear a snake might unexpectedly appear on the screen), and did not fall into the agoraphobia or social phobia categories. The most common simple phobias treated were fear of dogs or cats ($n=10$), cancer and other diseases ($n=9$), airplanes ($n=6$), snakes ($n=5$), and bees or spiders ($n=5$). Patients whose avoidance was secondary to some other major disorder (e.g. social phobia due to paranoid fear or withdrawal, resulting from clinically significant depression) were not included in the sample. Despite being assessed during a 15-year period prior to its publication, these diagnostic criteria are identical to those of DSM-III.

After the diagnosis had been made, the following data were collected before treatment was administered.

Psychiatric Questionnaire (Solyom *et al*, 1971). This was administered in the form of a semi-structured interview by the senior author, and in some cases by a second psychiatrist as well. It included description and ratings of the phobic and obsessive symptoms, depression and other clinical features, course of disorder, and personal history. Symptoms were rated on a five-point scale (0–4), with a rating of 0 indicating no symptoms and 4 indicating incapacity. Only ratings of 2 (indicating definite uneasiness and frequent avoidance) or more were considered clinically meaningful.

Self-rating of symptoms. The patient was asked to rate his symptoms on a scale of 0–4 by choosing one of the five statements that best described how he had been feeling with respect to each symptom during the previous week. The five points roughly corresponded to absent, mild, moderate, severe, and extremely severe. Symptoms rated by the patient were his main phobia (his worst fear), two measures of anxiety (cognitive apprehensiveness and somatic symptoms of anxiety), depression, depersonalisation, obsessive symptoms, and change of habits.

Self-rating of social maladjustment. The patient was asked to rate on a similar five-point scale the extent to which his symptoms interfered with adjustment at work (either outside the home or as a housewife), adjustment with regard to leisure activities, sexual adjustment, social adjustment within and outside the immediate family, and expressed self-satisfaction.

The three rating scales listed above were adapted from Gelder & Marks (1966), who measured inter-rater reliability as well as the reliability of the psychiatrists' ratings compared with the patient's own ratings of the same symptoms.

IPAT Anxiety Scale Questionnaire (Cattell & Scheier, 1963). The patient completed the IPAT. Half of its 40 questions refer to overt, symptomatic, conscious anxiety; the remainder consist of covert, hidden-purpose probes.

Fear Survey Schedule (Wolpe & Lang, 1964). This is a patient self-rating, on a five-point scale (from 'not at all' to 'very much') of the amount of fear elicited by 72 common phobic stimuli. These stimuli fall into six clusters of fears: death and tissue damage (18 items); social (17 items); other, e.g. large open spaces, crossing streets, journeys by train, bus, car (16 items); animals (9 items); miscellaneous (8 items); and noise (4 items).

Maudsley Personality Inventory (MPI) (Eysenck, 1958). Eysenck's 48-question inventory yields scores on two orthogonal dimensions of personality: neuroticism or emotionality, and extroversion.

Leyton Obsessive Inventory (LOI). (Cooper, 1970). This consists of 69 items, 46 of which relate to symptoms

of obsessive—compulsive neurosis and 23 to obsessive personality traits. In addition, the LOI has two intensity scales—resistance and interference—which measure the degree to which the patient resists his or her obsessive symptoms, and the extent to which the symptoms interfere with other activities. Because the questionnaire was published five years after our data collection began, scores are available on only 24 patients.

General Information Sheet. This single-page questionnaire elicited demographic information about the patient, his parents and siblings.

The three groups of social, agoraphobic and simple phobics were compared with respect to symptomatology and other aspects of natural history and demographic and personality variables. On those variables that were ordinal or better with respect to level of measurement, the statistical analyses consisted of analyses of variance followed, when the overall difference among the three groups was significant at the one in twenty level, by *t*-test comparisons of the groups two at a time, using the Tukey B procedure (Wine, 1964) to test for differences. On nominal variables, the groups were compared using the chi-squared method. Missing data were not estimated; analyses of variance, *t*-tests and chi-squares were computed using available data only.

Results

Social phobics were found to differ from both agoraphobics, as Amies *et al* (1983) reported, and from simple phobics, as Marks (1969) demonstrated.

Demographic data

Demographic data for all groups are given in Table I. Compared with the agoraphobia group, the social phobia group was younger, had fewer women and married persons, was better educated and had more members in the professions. Compared with the simple phobics, the social phobics were younger and fewer were female or married, but they did not differ significantly with respect to education or occupation.

Symptomatology

The three groups differed in the content of their phobias, but not in the intensity of their main phobia (worst single fear, as described by the patient; for a social phobic this might for example be 'parties'; for an agoraphobic it might be 'supermarkets'). Among the three groups, the agoraphobic patients scored significantly higher on ratings of agoraphobia, the social phobics on social phobia, and the simple phobics on simple phobia (full details of results are obtainable from the authors on request).

On the total FSS score—a measure of general fearfulness—the agoraphobics obtained a score of 133, which was significantly higher than the 99 and 91 returned by the social phobics and simple phobics respectively.

The social fears score on the FSS was identical in the agoraphobia and social phobia groups. In addition, 55% of the agoraphobic patients suffered from clinically significant social phobias (≥ 2 on a 0–4 scale) as rated by a psychiatrist, and 30% of the social phobics suffered from clinically significant agoraphobic symptoms.

On measures of associated symptomatology, the social phobics and agoraphobics did not differ on the four measures of anxiety, on the two measures of depression, or on the self-rating of maladjustment. On each of these measures, the social phobics and agoraphobics had significantly higher scores than the simple phobics, with the exception of self-rated depression. The agoraphobics were rated higher on rituals, ruminations, and horrific temptations than the social and simple phobics, but the three groups did not differ on self-rated scores of rituals, pervasive doubts, and horrific temptations.

The mean symptom scores for all three groups on the LOI (social phobia, 18.6; agoraphobia, 19.1; simple phobia, 13.2) fell between the norm for obsessive patients (33) and the norm for normal men (9) and normal women (11) (Cooper, 1970). The three groups did not differ significantly on this measure.

History of illness

Data on the history of illness for all three groups are presented in Table II. The social phobics experienced their first phobic symptom significantly earlier than the agoraphobics, but significantly later than the simple

TABLE I
Demographic variables

Variable	Agoraphobic	Social	Simple	P (overall)
Age: years	36.0	30.5	34.2	0.01
Female: %	86	47	78	0.0001
Married: %	78	58	71	0.05
Professional: %	12	41 ¹	30 ¹	0.0001
Educational: years	10.2	13.1 ²	12.4 ²	0.0001

1, 2 Do not differ significantly

TABLE II
History of illness variables

Variable	Agoraphobic	Social	Simple	P (overall)
Age of onset				
Age of 1st symptoms	24.5	16.6	12.8	0.001
Age of onset of illness	26.0 ¹	23.5 ¹	16.0	0.0001
Precipitating factors: % endorsing a precipitant	79 ²	79 ²	61	0.05
Course of disorder				
Constant static: %	30.0	43.6	45.5	NS
Constant worsening: %	6.7	10.3	12.7	
Fluctuating: %	35.0	25.6	30.9	
Phasic: %	28.3	20.5	9.0	
Getting better: %	0	0	1.8	
Delay in seeking help: years	1.0 ³	3.7 ³	21.0	0.0001
Mental disorder in parents				
Fathers: %	23.2	21.6	25.5	NS
Mothers: %	30.4	16.7	30.9	NS

1, 2, 3 Do not differ significantly

phobics. With respect to age of onset of the illness (i.e. when the symptoms for which the patient ultimately sought help became continuous), the social phobics and the agoraphobics did not differ significantly; both occurred significantly later than the simple phobics.

The Psychiatric Questionnaire listed 14 life events occurring during the six months preceding the onset of the present illness: fright, acute danger, serious illness, death of a relative or friend, domestic crisis, unavoidable conflict, sexual problem, bethrothal, occupational problem, school problem, childbirth, pregnancy, menopause, and other crises. A larger proportion of the social and agoraphobic groups, compared with the simple phobia group, cited one or more of the precipitating factors. The three groups differed significantly with respect to four precipitants: domestic crises, occupational or school difficulties, and fright. Compared with the agoraphobics, the social phobics more often cited occupational (7% vs 0%) or school (10% vs 0%) difficulties and less often domestic crises (19% vs 33%). Compared with the simple phobics, the social phobics more often cited occupational (7% vs 0%) or school (10% vs 2%) difficulties and less often mentioned fright (12% vs 23%). Fright among the simple phobics usually involved the phobic object (e.g. being stung by a bee).

The patients were categorised according to Ingram's (1961) four types of course of disorder (with the additional category of 'getting better', which characterised one of our patients). The modal course for agoraphobics was 'fluctuating and phasic' (63.3%). For social phobics the figure was 46.1%, and for simple phobics, 39.9%; these differences did not reach statistical significance.

Delay in seeking help was defined as the interim between the time the phobic symptomatology became continuous and when the patient first sought psychiatric help.

There were no differences among the three groups in the incidence of neurosis, personality disorder, psychosis, and organic brain syndrome among fathers, mothers, siblings, or other relatives regardless of the relative group examined.

Personality traits

The obsessional personality trait scores from the LOI of the social phobics (9.9) and the agoraphobics (10.5) were much higher than those of the simple phobics (6.2), but the overall difference among the three groups did not reach statistical significance ($F = 2.6$).

On the MPI, the neuroticism scores of the social phobics (29.4) and the agoraphobics (30.2) did not differ from each other, but both were significantly higher than the scores of the simple phobics (21.4), who scored in the same range as normals: 20.9 and 19.9 for American and English students respectively (Eysenck, 1958). A similar pattern emerged with respect to extroversion, with the social phobics (19.2) and the agoraphobics (20.6) differing significantly from the simple phobics (25.3), whose mean extroversion score was closer to the normal score: 28.5 and 24.9 for American and English students.

Discussion

The results of the present study support the recognition of social phobia as a distinct diagnostic category. Although sharing features with both agoraphobics and simple phobics, the social phobics do not fit satisfactorily into either category.

Our comparison of social phobics with agoraphobics confirmed many of the differences reported by Amies *et al* (1983), but produced a few

discrepancies. Like them, we found more males, more single persons, better education, and higher occupational status among the social phobics. Like the British samples, our socially phobic patients were as anxious and depressed as were the agoraphobics, and had equally high neuroticism scores. We also found, like Amies *et al*, that the first symptoms appeared earlier in life in the social phobics than in the agoraphobics. Discrepancies between the two studies involve obsessive-compulsive symptoms, delay in seeking help, and extroversion scores: whereas Amies *et al* reported no differences between the two groups on obsessional symptoms (undifferentiated), we found higher ratings for rituals, ruminations, and horrific temptations among the agoraphobics. As was the case in Britain, however, few patients in either group had clinically significant obsessive symptoms (Amies *et al* reported obsessive symptoms in 20% of each group). Our data on delay in seeking help (social phobia 3.7 years, agoraphobia 1.0 year) are apparently substantially different from those of Amies *et al* (social 12 years, agoraphobia 10 years), but if delay is calculated from the time of first symptom, as was done in that study, rather than from the time when the illness became continuous, the delay for social phobics is increased to 11.2 years, which is closer to Amies' figure of 12 years. The revised definition of delay for the agoraphobia group, however, yields a figure of 2.5 years, which is much shorter than the 10 years that Amies reports. One possible explanation is that agoraphobic subjects came to therapy following considerable publicity about the diagnosis and its favourable response to behaviour therapy. Shortly thereafter, the Medicare system was introduced in the USA, enabling patients to come for treatment without direct cost.

We found no differences between the social phobics and agoraphobics on either of Eysenck's orthogonal dimensions of personality, whereas the social phobics of Amies *et al* scored lower than their agoraphobic patients. This discrepancy may in part be accounted for by the fact that we used the MPI and Amies used the later version—the Eysenck Personality Inventory (Eysenck & Eysenck, 1964), which may support different spectra of the two dimensions.

The overlap between social phobia and agoraphobia, first reported by Amies *et al* (1983), also occurred in our groups. Among the social phobics, 30% had clinically significant agoraphobia (though rated at lower intensity than their social phobia), among the agoraphobics, 55% had clinically significant social phobia (again, rated at lower intensity than their agoraphobia). Thus, one can conceive of

three groups—social phobia, agoraphobia, and a group with combined social and agoraphobia. The diagnosis was based, however, on the presenting complaint (the 'main phobia') of the patient. Had we assessed 'pure' social phobics and 'pure' agoraphobics, the differences between them may have been enhanced, most notably perhaps in terms of situations avoided and types of autonomic and cognitive responses. For instance, a social phobic woman also avoided supermarkets; the stated basis of her fear: 'What if I meet someone I know?'. Many agoraphobics, if not all of them, are also fearful of making fools of themselves because of their phobic responses.

The problem of overlap may also arise because both agoraphobia and social phobia may be based on fear of having a panic attack; i.e. both may suffer from panic disorder (DSM-III).

We have attempted to summarise the differences between social phobics and agoraphobics in Table III. The separations are derived by definition and from clinical observations, and would perhaps be supported by research findings on 'pure' social phobics and agoraphobics.

The data presented here support the view (Snaith, 1968; Solyom *et al*, 1974; Klein, 1981) that agoraphobia and also perhaps social phobia are produced by essentially different mechanisms from those which seem to produce simple phobia. The typically tangible onset (fright involving the phobic object is the most common precipitant), and their modal 'constant static' course are consistent with the view that simple phobias are conditioned classically and maintained instrumentally by avoidance (Mowrer, 1947). Mowrer's two-factor theory, however, should be amended to account for the non-random variation in the content of phobias by including the notion that humans are more 'prepared' to develop certain conditioned fears because of their importance to the survival of the species (Seligman, 1971; Seligman & Hager, 1972).

The significantly higher level of anxiety among the agoraphobics and social phobics, the less discrete nature of the phobic object, and the lack of correspondence in content between precipitating factors and avoidance of these phobic situations and adjacent symptomatology are inconsistent with the conditioning theory of aetiology.

How then might they develop? One possibility is that separation anxiety (agoraphobia) and fear of strangers (social phobia)—common fears in childhood that usually disappear by the age of four (Jersild & Holmes, 1935)—may be incompletely unlearned, so that later, when the person's general anxiety reaches a critical level because of calamitous

TABLE III
Differences in features of man phobia, as described by social phobics and by agoraphobics

Feature	Social phobics	Agoraphobics
Situations avoided	Parties and social gatherings Meeting friends or acquaintances	Crowds in public places Public transport Being alone
Response	Blushing Tremor Stammering	Panic—dyspnea, tachycardia, faintness, etc.
Activity avoided	Speaking, eating, writing in public	Travel, shopping
Anticipatory thoughts	What if I stutter, blush?	What if I faint, die, lose control
Fear expressed	Embarrassment Loss of control	Panic, loss of control Embarrassment
Constancy of response	Constant	Fluctuates
Modifiers of response	A spouse (for some)	Presence of dependable other
Course	More frequently static (44%)	More frequently fluctuating and phasic (63%)

events, a domestic crisis, or difficulties at work or school, the fears re-emerge. The failure to unlearn may result both from maternal over-protection and modelling of maternal fear behaviour. There is evidence that mothers of agoraphobics are more anxious and more over-protective than mothers of normal subjects (Solyom, *et al*, 1976), and that both parents are perceived as less caring and more over-protective by social phobics (Parker, 1979). Whether maternal over-protection elicits fear behaviour in the child or the fearful child evokes over-protective behaviour in the mother, the general effect of over-

protection seems to be the preservation of separation anxiety or attachment behaviour (Bowlby, 1969) and fear of strangers (Freedman, 1965; Schaefer, 1966)—the basis of 'preparedness' for agoraphobia or social phobia.

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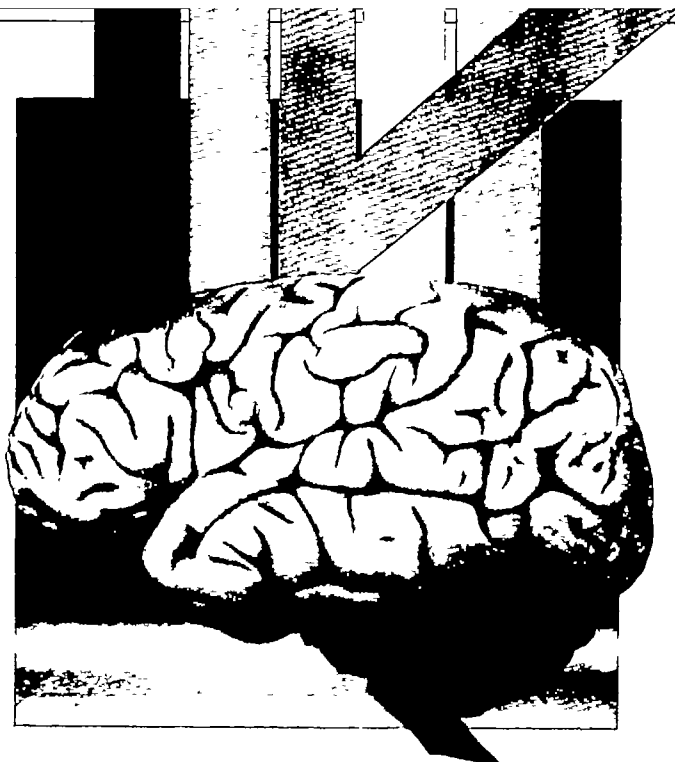
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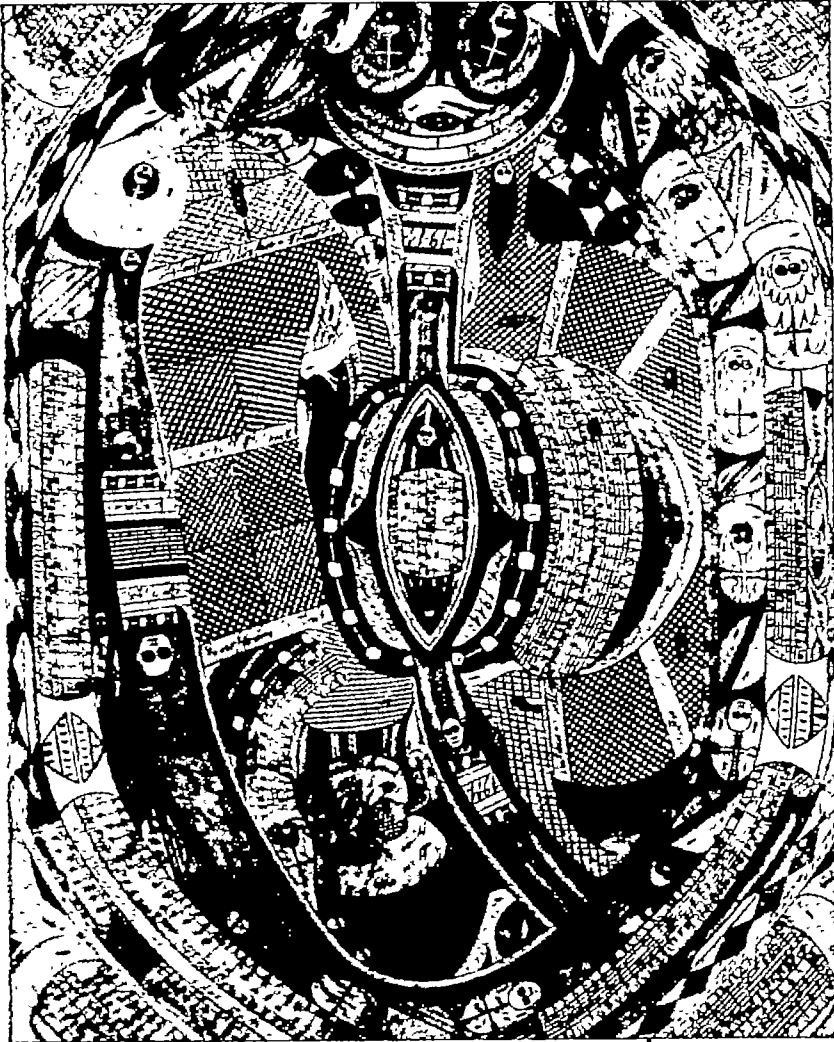


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A SCHIZOPHRENIC.

Adolf Wölfli spent 35 years in the Waldau Mental Hospital, Berne, suffering from schizophrenia. During that time he produced many drawings that have since gained widespread acclaim.

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MA/2415

The Use of Seclusion in Psychiatric Hospitals in the Newcastle Area

P. THOMPSON

Between 1981 and 1984, the frequency with which seclusion was used decreased in Newcastle. Secluded patients were younger, more often male, unmarried, unemployed, and admitted formally, and there was an association with certain diagnoses. Most seclusions occurred early in the hospital stay, mainly in the daytime, especially at mealtimes, and first seclusions were the longest. Violent incidents were usually associated with non-psychotic patients, and staff were the commonest victims. Medication used was major tranquillisers or paraldehyde.

Although seclusion has been in use for many centuries, early asylums used mechanical restraint to control disturbed patients. By the early nineteenth century the emergence of 'moral treatment' for the mentally ill, developed by Pinel, Conolly, Hill and others, led to the use of seclusion in preference to mechanical restraint.

With the development of new drugs, in particular chloral hydrate and bromides, workers such as Hill, Charlesworth, and Smith felt that seclusion "as a means of control, might be as successfully and usefully dispensed with as instrumental restraint had already been" (Walk, 1970). The availability of paraldehyde and barbiturates led to further changes in the management of the mentally ill in the early 1900s, and thereafter ECT and other modern treatments made their impact.

By the 1950s seclusion seemed largely restricted to the 'disturbed wards', many workers believing that it promoted neglect and abuse, besides provoking fear and resentment in the patient. The 'open-door' movement, encouraged by the Mental Health Act of 1959, led to staff in psychiatric units becoming less experienced in coping with violence, less willing to accept it, and happy to give up seclusion rooms.

Seclusion is, however, still in use, but information about its use is currently limited and comes largely from the USA, with the exception of a small study from the UK (Campbell *et al.*, 1982).

'Restraint' is the enforced control of patients' ability to behave in ways that threaten or cause harm to themselves, to others or to property. It may be manual, mechanical, situational, or chemical. 'Situational restraint' includes the use of special hospitals, secure units, locked wards and seclusion.

Thus 'seclusion' is a form of situational restraint where the patient is forcibly isolated from other patients in a room, the door of which is locked from the outside. It is used most often following manual

restraint, makes mechanical restraint unnecessary, and allows chemical restraint to come into effect (Wells, 1972). It may be that the environment of the seclusion room itself has a calming effect, but there are essential differences between seclusion and the behavioural technique of 'time-out': a reinforcing stimulus need not be identified for seclusion, which is always an emergency procedure, whereas 'time-out' is a planned conditioning method not necessarily involving the use of a locked room.

Method

The study looked retrospectively at the use of seclusion from 1981 to 1984 in all the psychiatric units of the Newcastle area, serving an initial population of 426 000. It did not cover admissions or seclusions in units for mental subnormality, psychogeriatrics and child psychiatry. Seclusion does not occur in the latter two, and it is rarely used in long-stay wards.

For the year 1981, all general adult psychiatric admissions were studied in more detail, to obtain appropriate control groups. Information was obtained from case-notes, admission documents, nursing notes, and seclusion records. Statistical tests used were Student's *t* test, chi-squared analysis with Yates' correction factor, Fisher's exact test, and the Wilcoxon rank-sum test.

Results

Over the period studied, there was a decline both in the number and rate of seclusions in Newcastle (Table 1). Most patients were secluded once only, and changes were most pronounced in the group of patients most frequently secluded.

Of patients admitted in 1981, 10.4% (23) with bipolar affective disorder were secluded ($P < 0.001$), 6.9% (22) with schizophrenia were secluded ($P < 0.01$), and 37.5% (3) of mentally handicapped men were secluded ($P < 0.001$). Other diagnoses were not significantly associated with seclusion. Overall, 77.3% (51) of secluded patients had diagnoses of psychotic illnesses, compared with 49.2%

TABLE I
Number and rates of seclusions in 1981-1984

	Year			
	1981	1982	1983	1984
Number of admissions	2017	1636	1867	1884
Number of patients secluded	66	56	42	49
Patients secluded/1000 admissions	33	34	23	26
Number of patients with 1 seclusion	39	35	19	31
Number of patients with 2-3 seclusions	15	12	12	11
Number of patients with 4-9 seclusions	8	6	11	1
Number of patients with >9 seclusions	4	3	0	0

TABLE II
Nature of incidents resulting in seclusion in 1981, and frequency of repetition

Diagnosis	Seclusion frequency ¹	n	Self	Type of behaviour: 1% of incidents			
				Violence to: Staff	Other	Threat	General
Schizophrenia and paranoid psychosis	S	25	0	48	0	44	8
	R	14	14	21	21	43	0
Bipolar affective (hypomania)	S	32	0	16	9	16	59
	R	27	0	15	19	15	52
Other psychoses	S	8	12	25	25	0	37
	R	68	26	25	19	16	13
All psychoses	S	65	2	29	8	22	40
	R	109	18	22	19	19	21
All non-psychotic disorders	S	23	13	48	13	22	4
	R	37	24	57	16	3	0
All patients		234	14	32	15	17	22

¹ S = seldom (<3), R = repeated (>4)

(991) of controls ($P < 0.001$), and 61% (41) of secluded patients were men ($P < 0.05$).

The secluded group had a mean age of 36.1 ± 13.6 years, compared with 44.4 ± 16.0 years for the controls ($P < 0.05$). Secluded patients were admitted for a median of 29 days, compared with 20 days for controls ($P < 0.01$). Only 36.4% (24) of secluded patients were married, while 52.9% (898) of controls were ($P < 0.001$), and only 17.1% (7) of secluded men were employed compared with 52.6% (430) of controls ($P < 0.001$).

While previous admission was unrelated to seclusion, compulsory admission under the Mental Health Act (1959) was associated with subsequent seclusion; 51% (39) of secluded patients had been admitted formally, compared with only 8.4% (162) of all admission. Of all formal

admissions, 19.4% were later secluded, as against only 1.9% of informal admissions ($P < 0.001$).

The type of incidents resulting in seclusion are shown in Table II. Of the 12 patients who were repeatedly secluded (i.e. four or more times), eight were male and ten had psychotic illnesses. There was a median gap of two days between seclusions, and in this group of patients incidents tended to be more often acts of violence, to self or others.

First seclusions were longer (median 9.1 h) than subsequent seclusions (median 3.3 h), $P < 0.01$, and the overall duration of seclusions ranged from 10 min to 25.5 h (median 4.3 h). Most seclusions occurred shortly after admission, 25% (54) within 24 hours. Of these 'early' seclusions, 67% (36) occurred between 6 p.m. and 6 a.m. Overall, however, most of them occurred in the daytime,

57% (123) happening between 6 a.m. and 6 p.m. The three mealtime hours accounted for 54% (23) of seclusions of non-psychotic patients, but only 14% (23) of those involving psychotic patients ($P < 0.001$).

There were 12 patients who were suffering from other illnesses (e.g. cardiac failure, faecal impaction) at the time of seclusion, and 29% (19) of cases were noted to have a current alcohol or drug problem in addition to their main psychiatric diagnosis. None of the secluded females had been in prison, but 30% of psychotic and 63% of non-psychotic male patients had been.

Only ten patients were secluded without 'as required' medication. Major tranquillisers were used alone in 40% (86) of incidents, tranquillisers with paraldehyde in 19% (40), and paraldehyde alone in 8% (17). A benzodiazepine was used once (< 1%).

Discussion

The percentage of patients found to have been secluded in this study was low compared with the US figures, but diagnoses were similar - except that bipolar affective disorder was a larger and schizophrenia a smaller group in Newcastle, probably reflecting different diagnostic criteria (Phillips & Nasr, 1983; Soloff, 1983). The decrease in the use of seclusion between 1981 and 1984 may have been related to the introduction of the Mental Health Act in 1983.

The findings in relation to age and marital and employment status probably reflect the more disturbed characteristics of this group of patients, and the onset, duration, severity, and social effects of their illnesses.

Staff intolerance of threatened violence in males resulted in 36 seclusions of males, compared with only two of females ($P < 0.001$). The most frequent type of incident was violence to others, staff being the commonest victims. Non-psychotic patients were most often violent, and patients suffering from bipolar affective disorder (hypomania) most often generally disturbed. Schizophrenic patients were often secluded for threatened, as well as actual harm. Incidents involving other psychoses were not of any specific type but, as Fottrell (1980) also found, a small number of patients were repeatedly involved in violent incidents.

Both Campbell *et al* (1982) and Mattson & Sacks (1978) suggested that self-harm and assault can occur during seclusion. In the present study this was noted on only one occasion, when a male patient demolished the seclusion room.

The timing of incidents is probably a reflection of

the response of non-psychotic patients to external stimuli; psychotic patients may be responding to internal stimuli, with a more random timing of their outbursts.

If the experience, morale, and number of staff on a ward are all low, this may lead to patients becoming disturbed and thus to seclusion rather than other responses, particularly at mealtimes. New patients to the ward are usually at their most disturbed, and of unknown potential; they may be secluded 'to be on the safe side', especially if they are male and their appearance is intimidating. What is perhaps most important is the attitude of nurses, doctors and other ward staff towards the patient, which may partly explain why staff are the commonest victims or why attacks on staff may lead not to reprimands but to seclusion.

From the author's personal experience, it seems that special observation of selected patients, i.e. allocating a nurse to be with one patient only at all times (usually on admission or after a disturbed period), helps to reduce violence and thus the incidence of seclusion.

Guidelines have been published on the management of violent patients (Department of Health & Social Security, 1976; Confederation of Health Service Employees, 1977; Royal College of Psychiatrists, 1980) and recommendations on the practice of seclusion have been formulated, the most widely used probably being those of the Royal College of Nursing (Society of Psychiatric Nursing 1979; Royal College of Psychiatrists, 1982; Jones, 1982; Wilkinson, 1983). Further guidelines from the Mental Health Act Commission are expected.

While there may remain a place for seclusion in general psychiatry, most incidents can and should be managed in other ways, although the prevention of such incidents is even more important. Once seclusion has occurred, it should be for short periods, reviewed regularly, and only rarely without medication.

It is difficult to generalise about seclusion as its use is so relatively rare, the individuals involved so varied, and the published data from this country so lacking. Seclusion is such an embarrassing topic that reports have been scanty and anecdotal, or else simple statements of hospital policy or person pronouncements. The subject deserves further study.

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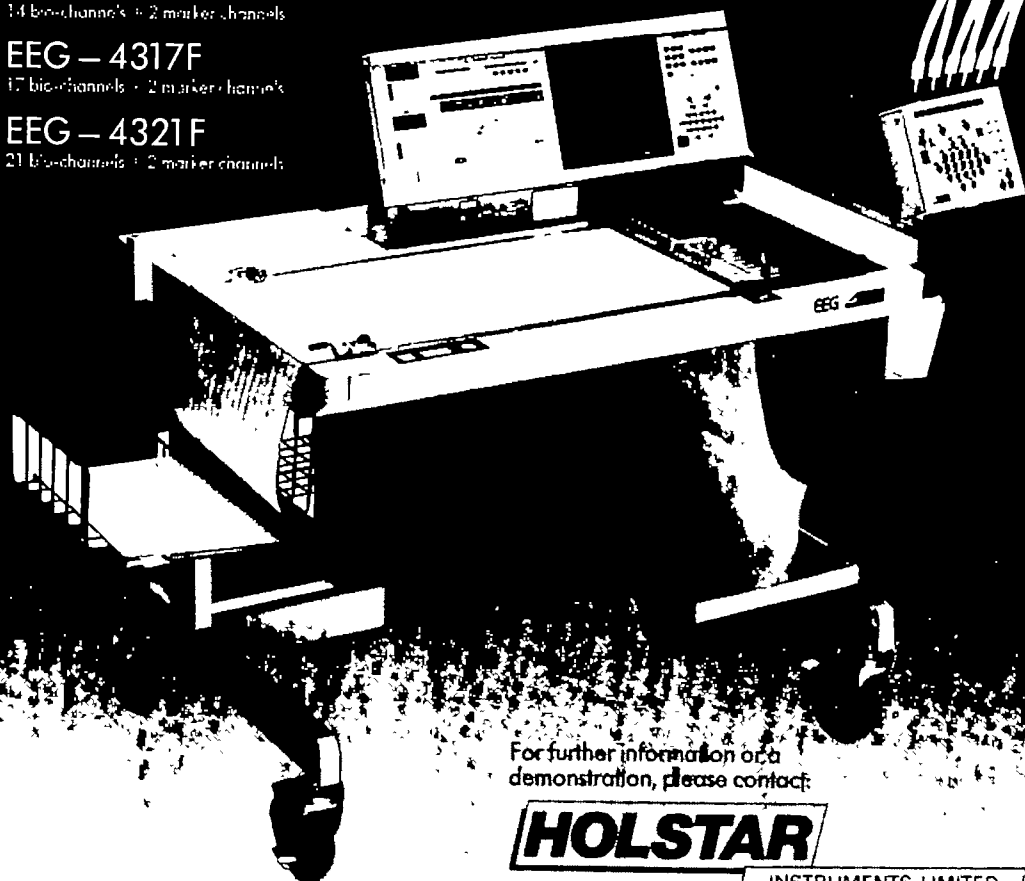
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Suicide in Young Doctors

J. C. RICHINGS, G. S. KHARA and M. McDOWELL

Fifty-five cases of suicide by doctors under the age of 40 were studied because of concern about deaths among anaesthetists in training. The excess mortality of doctors from suicide was confirmed, suggesting that risk factors associated with being a member of the medical profession continue to operate, although mortality for young male doctors from accidental poisoning is falling. Analysis of the branches of medicine in which the doctors were working showed no definite specialty predominance. There was a somewhat greater risk for women doctors born overseas.

Suicides account for a substantial number of deaths in England and Wales; evidence shows that they are on the increase and that, although the risk is greatest among middle-aged men, the greatest rise recently has been in young people (McClure, 1984; Bulusu & Alderson, 1984). Successive *Decennial Supplements on Occupational Mortality* (General Register Office (GRO), 1958, 1971; Office of Population Censuses and Surveys (OPCS), 1978) for England and Wales have shown relatively high rates of suicide among male members of the medical profession; comparable figures are not available for women doctors because of limitations in the data and methods used for calculating occupational death rates (McDowell, 1983). However, an increased rate of suicide has been reported in women doctors in America (Steppacher & Mausner, 1974). A number of studies have suggested that a doctor's specialty may increase the likelihood of suicide: anaesthetists (Helliwell, 1983), psychiatrists (A'Brook *et al.*, 1967), ophthalmologists (Waterson, 1976), and pathologists (Harrington & Shannon, 1975) have at various times been thought to be particularly vulnerable.

The standardised mortality ratios (SMRs) for male doctors from the post-war Decennial Supplements (GRO, 1958, 1971; & OPCS, 1978) are shown in Table I. The supplement based on the year 1951,

1958) notes that the excess was greatest among the younger male doctors, with SMRs of 400 for the 20-30 age group, 283 for 35-44, 211 for 45-54, and 113 for 55-64 (the SMR for all males in England and Wales is set as 100 for each age group). Similar breakdowns by age were not published for the two subsequent studies (GRO, 1971; OPCS, 1978), but a special tabulation of mortality ratios for suicide in male doctors for 1970-1972 shows excess mortality at all age-groups (273 for age 25-34, 388 for 35-44, 316 for 45-54, and 388 for 55-64). Recent concern over the number of suicides in one particular group of young doctors (Helliwell, 1983) prompted us to undertake a study using more up-to-date information.

Method

The under-reporting of suicides in official statistics has been well recorded (Sainsbury & Jenkins, 1982; Adelstein & Marden, 1975), many will be recorded as open verdicts or accidental deaths, due to the natural reluctance of coroners to enter a verdict of suicide where some possible measure of doubt remains. A wider view of suicide, which includes deaths recorded as 'suicide' (ICD-9 categories E950-E959), 'undetermined' (ICD-9 categories - E980-E989) and 'accidental poisoning by drugs etc' (ICD-9 categories E850-E859) has been proposed by Adelstein & Marden. Although it has been argued that the wider definition adds little to most analyses (Sainsbury & Jenkins, 1982), the presentation and recording of suicide in doctors may vary from that of the general public. In order to avoid any possible bias, both the narrow and wide definitions of suicide have therefore been used in this study.

From death registration records at the OPCS we extracted draft death entry forms for deaths from the three groups of causes above, for medical practitioners under the age of 40 in England and Wales. We covered five years: 1972, 1975, 1979, 1980, and 1982. Suitable data on populations at risk were only available from 1972, and the years chosen were those for which occupation at death had been

TABLE I

Standardised mortality ratios for suicide in male doctors in England and Wales (1949-53, 1959-63, 1970-72) (GRO, 1958, 1971, OPCS, 1978)

Years	Age-group	SMR
1949-1953	20-64	226
1959-1963	15-64	176
1970-1972	15-64	335

TABLE II
Mortality ratios in England and Wales by the wide definition for suicide and its components in male and female doctors aged 25-39 for 1972, 1975, 1979, 1980 and 1982 combined

	SMR	Male Deaths·n	SMR	Female Deaths·n
A. Suicide (narrow definition)	165*	29	310*	7
B. Undetermined deaths	146	8	—	0
C. Accidental poisoning, drugs etc.	246*	8	550*	3
Wide definition (A + B + C)	170*	45	274*	10

* $P < 0.05$

TABLE III
Mortality ratios and number of suicides by narrow and wide definitions in doctors aged 25-39 in England and Wales

Year	Suicide (narrow definition)				Suicide (wide definitions)			
	Males		Females		Males		Females	
	SMR	n	SMR	n	SMR	n	SMR	n
1972	204	5	592	2	345*	12	421	2
1975	221	6	264	1	274*	11	476	3
1979	152	6	194	1	116	7	120	1
1980	73	3	198	1	81	5	125	1
1982	196	9	375	2	143	10	331	3

* $P < 0.05$

coded by OPCS. Each case was checked against detailed data available in the Civilian Doctors' Career Index (CDCI) maintained by the Department of Health and Social Security. The study is based on 55 cases - 36 suicides, eight 'undetermined', and 11 cases of accidental poisoning. Estimates of population at risk were based on data from the CDCI, and both mortality and population figures were cross-classified by sex and country of birth. For most cases we were also able to determine the main medical occupation and hospital specialty, if any.

Mortality ratios were calculated by comparing observed deaths of doctors under 40 with those expected. The expected deaths were derived from England and Wales mortality rate (age 25-39) for the respective causes (and for each of the study years) applied to the population of medical practitioners aged under 40, derived from the CDCI.

Results

The mortality ratios for the wide definition of suicide and its component parts for both male and female practitioners under the age of 40, for all the study years combined, are shown in Table II. Although the numbers of deaths are small, the highest ratios for both male and female doctors

are from 'accidental poisoning with drugs etc'. Ratios for both sexes and by both definitions of suicide are significantly greater than 100. Table III shows mortality ratios for each year of the study, for both the narrow and the wide definitions of suicide. The number of cases in each year is small, leading to quite marked variation between individual years. The mortality ratios for young female doctors are based on a small number of cases, and there is no discernable pattern of a time-trend, but the pattern for young male doctors varies according to the definition of suicide employed.

Mortality under the narrow definition of suicide shows no clear pattern over the years, but there is a marked decline in mortality ratios under the wider definition. Part of this decline is due to a fall in the crude rate of suicides (wider definition) of young male doctors - from 48 per 100 000 in 1972 to 32 in 1982 - but in the same period there has been a steady rise in the national rates for men under 40 (from 14 per 100 000 to 22), and this too has contributed to the decline in mortality ratios. This difference in mortality patterns for young male doctors between the two definitions is almost entirely due to a fall in deaths from accidental poisoning from drugs and other substances, of which there were five in 1972, three in 1975, and none in the subsequent years. Young female doctors, on the other hand, have consistently

TABLE IV
Mortality ratios in England and Wales for the wide definition of suicide in male and female doctors aged 25-39, for the years 1972, 75, 79, 80 and 82 combined, categorised by place of birth

	<i>British Isles</i>		<i>Elsewhere</i>		<i>All place of birth</i>	
	<i>SMR</i>	<i>n</i>	<i>SMR</i>	<i>n</i>	<i>SMR</i>	<i>n</i>
Males	188*	29	172	6	170*	75
Females	186	5	523*	5	274*	10

* $P < 0.05$

higher mortality ratios than their male colleagues (Tables II and III). The crude rate of suicide (wider definition) in young female doctors was in fact less, at 23 per 100 000, than that for young male doctors (31 per 100 000). However, the national suicide rates for women are considerably lower than those for men, thus giving a higher mortality ratio.

The England and Wales SMR for the wider definition of suicide for Social Class I, (Registrar-General's classification), which includes doctors, for men aged 15-44 in 1970-72 (the latest figure available) was 90 (OPCS, 1978). Young persons born outside the British Isles do have a higher risk of death from suicide: SMRs for ages 25-39 for the wider definition of suicide in 1969-73 for these men and women were 169 and 165 respectively (unpublished OPCS data). This suggests that the increased SMR for suicide in doctors is not related to social class.

Table IV cross-classifies the combined years' mortality ratios (wider definition) for each sex with place of birth (British Isles or elsewhere); the higher ratio in young female doctors compared with male doctors is almost entirely due to a very high ratio for the female doctors born overseas. Four of the five cases of young women doctors who were born overseas were registered as suicides, the other one as accidental poisoning. The similarity of the ratio for young male doctors, born in the British Isles or elsewhere, and the young female doctors born in the British Isles suggests a comparable degree of excess risk for the three groups.

Poisonous substances, including drugs, were the cause of death in 39 out of 55 doctors in our study (55% of the male and 90% of the female doctors). Members of the medical profession usually have ready access to drugs, and this probably accounts for the preponderance of this method of suicide. At the time of death, ten doctors were working as general practitioners, twenty-seven were employed in the hospital services, three worked in universities, and one worked in the community health service, the employment of the remaining fourteen was unknown.

By using the wider definition of the population at risk, the crude rates per 100 000 for general practitioners and all hospital specialties were both 23. For the individual hospital specialties, the rates were: anaesthetics 67, general medical 20, surgical 19, psychiatry 39, and pathology 36; these differences should be treated with caution, as the numbers are very small.

Discussion

There is an excess mortality from suicide in both young male and female doctors, using both the wide and narrow definitions; this had been shown previously for male doctors. The mortality ratios based on the narrow definition of suicides do not show a significant trend. The figures are based on a small number of cases, but do suggest that there might have been an underlying trend for the rates to decline since the early 1970s; if this is true, doctors would have decreased their suicide rate against the general tide. Mortality ratios based on the wider definition of suicide show a definite decline in the mortality ratios for young male doctors over the years studied, but there is no such clear evidence for young female doctors.

The overall excess mortality from suicide is not explicable in terms of social class, at least for young male doctors (female social class rates are not available) (Bulusu & Alderson, 1984), which suggests that there are risk factors associated specifically with being a member of the medical profession. Doctors usually have ready access to drugs, and this was a common cause of death for both men and women doctors. This contrasts with the general population, where access to drugs is restricted and where self-destruction by physical means such as hanging and poisoning by vehicle exhaust fumes assumes a more prominent role (Farmer & Rhode, 1980). In 1980, for men in England and Wales, death by poisoning accounted for 30% of total suicides (wider definition), in contrast with hanging, which accounted for 19% of cases. However, poisoning remains the commonest method for women: 56% in England and Wales for 1980 (OPCS, 1982).

The figures given for the individual medical specialties of these doctors must be treated with caution, as the majority of those in the hospital service were still in the training grades, and it is

difficult to be certain that the specialty in which they were working at the time of death was their main working experience or was intended to be their final career choice. The rate for anaesthetists is significantly higher than those in the other specialties. In our study, the suicides of anaesthetists were recorded in one year, but this may have been an atypical year. A study of suicide in anaesthetists in training for the years 1979–1982 (Helliwell, 1983) showed five deaths from suicide in that period: using the target population for anaesthetists for these years, the rate for that study is 39 per 100 000. It also examined each case in depth, showing that the individuals were not under-achieving or showing stress due to the work situation, but that each had a considerable emotional problem which was thought to lead to the suicidal act. We were not able to identify the medical specialty of 14 doctors; it is likely that most of them worked for short periods in either the hospital or general medical services. These doctors could make a considerable difference to the crude rates shown for the two main groups and the individual specialties, so these figures should be viewed with caution.

A doctor's place of birth is relevant to mortality

rates from suicide. Unpublished data from OPCS established that young people from overseas have a significantly higher mortality rate from suicide than those born in the British Isles. For male doctors there appears to be little direct influence of the country of birth on suicide mortality, compared with the experience of all men and women born overseas, but the higher risk of death from suicide of young persons born outside the British Isles does not appear to explain why the ratio for young female doctors born overseas is significantly greater ($P < 0.05$) than the ratio for young female immigrants. This gives cause for concern, and since this ratio exceeded that in all other groups of doctors studied it cannot simply be explained by gender or by ready access to lethal means. It is possible that the feelings of isolation, including separation from the extended family and failure to adjust to a new social environment, coupled with difficulty in obtaining medical posts in this country (Smith, 1980) have contributed to this high ratio. The absolute numbers are small, so the position should be monitored in the future to see if this trend continues.

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Relationship of Mood Alterations to Bingeing Behaviour in Bulimia

W. H. KAYE, H. E. GWIRTSMAN, D. T. GEORGE, S. R. WEISS and D. C. JIMERSON

Twelve women with bulimia participated in a study in which they binged and vomited on the day after hospital admission. Caloric intake, time spent bingeing and vomiting, and self-reported mood ratings demonstrated much variation from subject to subject. Both subjective and objective ratings of mood indicated that anxiety decreased more frequently and to a greater extent than depression, both during and after bingeing and vomiting. The present data, obtained in a controlled setting, tend to confirm previous information on binge episodes obtained by self-report from bulimic patients. Bingeing and vomiting episodes may provide bulimic patients with a physiological mechanism for temporarily relieving a dysphoric mood state.

The bulimic symptom complex in women has received much attention in the past decade and has been found to be surprisingly common (Stangler & Printz, 1980; Halmi *et al*, 1981; Pyle *et al*, 1983; Johnson *et al*, 1984). Bulimia has been hypothesised to be related to affective disorders. Patients with bulimia (Pyle *et al*, 1981; Gwirtzman *et al*, 1983; Hudson *et al*, 1983a) and their first-degree relatives (Hudson *et al*, 1983b) often have disturbances of mood and/or a history of substance abuse. Bulimia and affective disorders share certain neuro-endocrine dysfunctions (Gwirtzman *et al*, 1983), and the bulimia symptom complex often responds to anti-depressants (Pope *et al*, 1983; Walsh *et al*, 1984).

Only a few studies, using patient self-report, have examined the relationship between bingeing-purging and mood. The initiation of bingeing appears to be associated with weight control (Chiodo & Latimer, 1983) or stress (Strober, 1984). Several studies suggest that bingeing may serve to relieve tension (Johnson & Larson, 1982; Abraham & Beumont, 1982). The relation between bingeing-purging behaviour and the global depression often associated with bulimia is not clear.

Because patient self-report is not always reliable, we designed this preliminary study to allow observation and documentation of mood during and after bingeing and vomiting episodes. Bulimics rated their mood subjectively while bingeing and vomiting, and for two hours afterwards. Objective mood ratings were performed twice, once before and once after the sequence of bingeing and vomiting. Global mood was measured by standardised subjective and objective ratings some time after the bingeing and vomiting were over.

The mechanisms, biological or otherwise, whereby food intake alters mood are not known. Studies of

animals suggest that carbohydrate foods can produce alterations in certain brain neurotransmitters such as serotonin (Wurtman, 1983), a neurotransmitter also implicated in mood regulation; therefore it is important to determine whether different components of the food consumed, such as carbohydrate, influence mood directly.

Caloric intake and time spent in bingeing and vomiting were documented and compared with mood changes, because we were particularly curious as to whether improvement in mood was the factor responsible for terminating bingeing and vomiting. We therefore instructed patients to binge and vomit until they achieved whatever effect was important to them.

Method

Twelve women who satisfied DSM-III criteria for bulimia were admitted to an NIMH intramural research ward: clinical data for these subjects are given in Table I. All subjects gave informed consent for the study. All patients had a history of bingeing and vomiting at least once per day for three months prior to admission. Bulimics who frequently abused laxatives were excluded. We included one subject with current anorexia nervosa (patient No. 7 in Table I). All other subjects were between 82% and 118% average body weight. (Metropolitan Life Insurance Company, 1959). Three of the subjects had met DSM-III criteria for anorexia nervosa in the past but were currently over 82% average body weight.

Subjects were informed of the study prior to admission and were contacted when possible by the dietitian to determine their choice of binge foods. On the day of admission a physical examination, electrocardiogram and laboratory studies were performed. Many subjects had some degree of electrolyte abnormality, but none so severe as to exclude them from this study. The study was performed on the second day of admission, after medical clearance.

TABLE I
Clinical data for bulimic patients

Patient	Age	Height	Body weight (% of average)			Duration of bulimia (months)	Number of binge-vomit cycles	Depression scores	
			Current	Past min.	Past max.			Beck	Hamilton
1	20 yrs	163 cm	100%	75%	104%	46	1	26	5
2	23 yrs	165 cm	94%	86%	118%	63	1	80	30
3	21 yrs	161 cm	101%	71%	105%	54	2	29	23
4	23 yrs	167 cm	87%	78%	94%	66	2	53	46
5	29 yrs	165 cm	94%	90%	139%	144	2	4	2
6	27 yrs	162 cm	100%	91%	112%	102	3	17	16
7	31 yrs	168 cm	62%	61%	131%	85	3	65	14
8	23 yrs	152 cm	95%	84%	115%	85	3	6	5
9	20 yrs	166 cm	93%	88%	123%	36	4	39	13
10	20 yrs	165 cm	118%	93%	156%	36	4	1	1
11	25 yrs	159 cm	108%	88%	112%	144	4	41	13
12	26 yrs	168 cm	83%	68%	133%	42	5	48	28
Mean	24 yrs	163 cm	95%	81%	120%	75		34	16
S.E.M.	1	1	4%	3%	5%	11		7	4

Because bulimics often prefer isolation during bingeing, we decided to allow them to binge and vomit unobserved in a ward room, but with a physician immediately outside the door. On the day after admission, subjects fasted from midnight. They were placed alone in a ward bedroom with adjoining toilet facilities. At 9.00 a.m. an intravenous line was established. At 10:00 a.m. base-line mood ratings were obtained and the first blood sample drawn. Subjects were then given their first tray of food. A physician entered the room after each binge and vomit to supervise the self-report ratings and draw blood. (Our biological findings will be reported elsewhere.)

In an attempt to replicate the home environment, patients were permitted to take as much time as they desired to binge and vomit. We tried to supply as many as possible of the foods that subjects requested. Each subject was offered a tray of 5000–6000 kcal of food. The amount of food consumed was estimated by the dietitian by methods described elsewhere (Petersen *et al*, 1986). Patients were instructed to continue to binge and vomit until they had achieved whatever 'desired effect' determined the point at which they stopped at home, but with the exception of one subject who binged and vomited five times, we terminated the study after four binge-vomit episodes even if the 'desired effect' had not occurred.

Our comparison group comprised seven healthy female controls without personal or family history of psychiatric illness. Each was given a large (900–1700 kcal) test meal.

The controls did not vomit, and consumed their entire test meal at one sitting over one hour.

Mood was quantified by several methods. All 12 bulimics and all 7 controls self-rated changes in mood and other factors on a 7-item visual analogue scale before eating. This subjective rating was repeated by the bulimics after each binge-and-vomit, and at hourly intervals after termination of the last binge-vomit cycle (see Figure 2). The same scale was given to the controls immediately after their test meal and twice more at hourly intervals. Two psychiatrists rated seven of the bulimics on the Brief Psychiatric Rating Scale (BPRS) (objective rating) before they were given any food and again about 20 minutes after the last binge-vomit cycle. BPRS ratings for the two raters were averaged for each bulimic. The Beck Depression Inventory (Beck *et al*, 1961), the Hamilton Depression Scale (Hamilton, 1960) and the Montgomery-Asberg depression rating scale (Montgomery & Asberg, 1979) were administered by one rater within two days of subjects completing the bingeing and vomiting study.

Results

Caloric intake and binge-vomit patterns

The caloric intake per binge and the time spent bingeing, vomiting, or resting between cycles varied between subjects

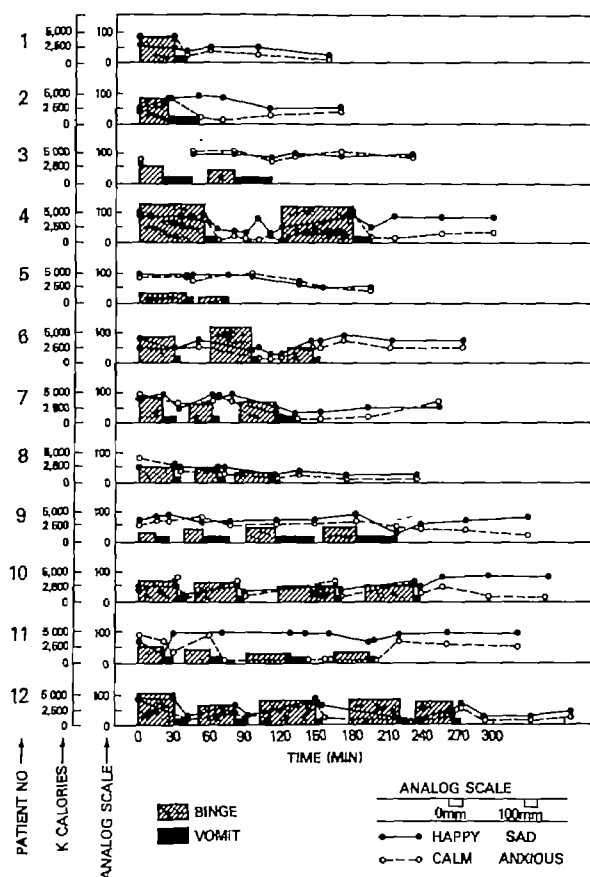


FIG. 1. Time course, caloric intake, and changes in subjective mood ratings during and after bingeing and vomiting. The horizontal dimension of the striped box indicates time spent bingeing, the vertical dimension indicates caloric intake per binge (0–5000 kcal). The horizontal dimension of the black box indicates time spent vomiting. Visual analogue mood ratings for anxiety (open circles) and depression (black dots) are indicated for each patient (100 = anxious, depressed; 0 = calm, happy). Patient 3 missed one rating.

(Fig. 1). However, every subject who engaged in multiple bingeing and vomiting cycles appeared to have a consistent pattern of caloric intake from binge to binge, and of time spent bingeing and vomiting. The mean duration of each binge was 30 minutes (s.e. 10) and the mean vomit duration was 13 minutes (s.e. 9). The mean individual binge consisted of 3500 ± 1338 kcal, and total caloric intake over the whole cycle of binges had a mean of 9360 ± 5275 kcal. Macronutrient distribution was $52\% \pm 10\%$ carbohydrate, $37\% \pm 9\%$ fat, and $11\% \pm 2\%$ protein.

Subjective mood ratings

Before eating, bulimic subjects rated themselves as significantly more depressed, more anxious, less hungry, and less confused than controls (Fig. 2). After bingeing, bulimic subjects showed a significantly greater decrease in anxiety,

and a smaller decrease in hunger, than the controls did after their meal. Neither group showed an overall change in depression ratings.

Each bulimic appeared to have a unique pattern of changes in mood on the self-rating scales during and after binge–vomit cycles (Fig. 1). The great variation between individuals in patterns of depression and anxiety are apparent when graphed with caloric intake and time spent bingeing and vomiting. For example, subjects 6, 7, and 8 each had three cycles of bingeing and vomiting. Subject 6 had her greatest improvement in anxiety after her second binge and vomit, but less change in depression; and her anxiety and depression increased after the third binge and vomit. Subject 7 had a substantial reduction of both depression and anxiety after her third binge and vomit. Subject 8 had a step-wise reduction in anxiety and depression after each binge and vomit.

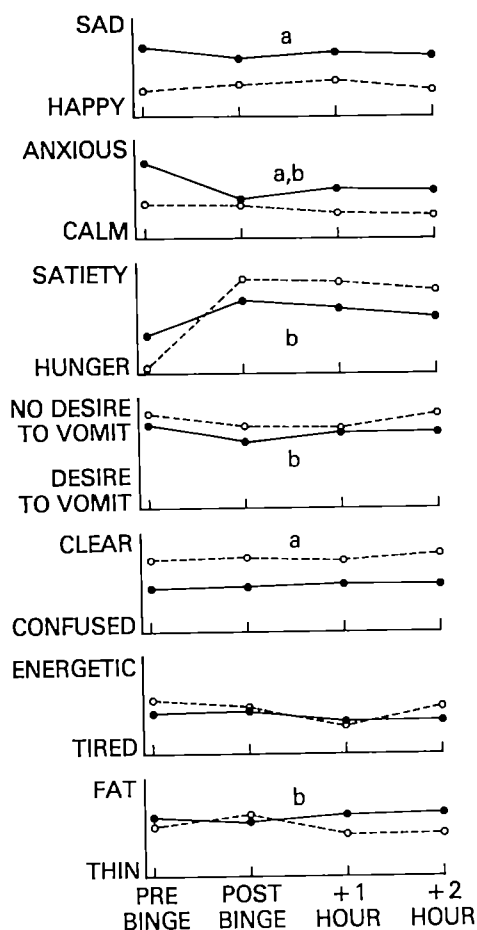


FIG. 2. Comparison of bulimic patients ($n=12$) and healthy control women ($n=7$) on visual analogue self-ratings. Seven factors were self-rated at base-line ('pre-binge'), ratings were repeated 'post-binge' (immediately after the last binge and vomit for bulimics, after the test meal for controls), and again at 1 and 2 hours 'post-binge'. Bulimics are indicated by black circles, healthy controls by open circles. 'a' indicates a significant difference ($P<0.05$) between bulimics and controls by repeated-measures ANOVA, 'b' a significant difference in group $\times$ time interaction (difference in pattern of response between groups).

On the basis of our clinical impressions during the study, we considered a subjective score meaningful only if there was more than 50% change from base-line. Because of heterogeneous response on the self-rating scale, we categorised changes in mood into three patterns:

1. Of twelve subjects, six (numbers 4, 6, 8, 10, 11, 12) had a reduction of 50% or more in depression and/or anxiety between cycles of bingeing and vomiting: even

though their mood improved these subjects continued to binge and vomit. After all the cycles of bingeing and vomiting were over, most of these subjects had greater residual depression than anxiety.

2. Four subjects (numbers 1, 2, 7, 9) had an improvement in anxiety and/or depression of at least 50% at the time they stopped bingeing and vomiting.
3. Two of the twelve subjects (numbers 3 and 5) did not achieve any improvement in mood either during or after bingeing and vomiting

A substantial decrease in anxiety occurred at some point during or after the binge-vomit cycles in eight of the twelve bulimics, whereas only four had a substantial reduction in depression. Base-line self-rating analogue scores for anxiety and depression were positively correlated ($r=0.643$, $P=0.02$, $n=12$), but there was no significant correlation ($r=0.480$, $n=12$) between subjective anxiety and depression after the end of the last binge-vomit cycle. Base-line subjective and objective anxiety scores were significantly positively correlated ($r=0.772$, $P<0.05$, $n=7$), but base-line depression scores were not.

BPRS ratings

The last seven bulimic subjects to participate in this study were assessed with the BPRS at 10:00 a.m. (base-line) and again 20 minutes after the end of bingeing and vomiting. Paired t -tests revealed a significant reduction in anxiety ($t=3.74$, $P<0.01$) but not in depression (Fig. 3). Two other measures on the 24-item BPRS were significantly reduced: guilt ($t=3.07$, $P<0.05$) and tension ($t=4.8$, $P<0.01$).

Global depression ratings

Bulimics had a wide range of depression scores on the Beck Depression Inventory and the Hamilton Depression Scale (Table I). Montgomery-Asberg depression scores were of a similar magnitude. There were no correlations between base-line or subsequent scores on the self-rating or BPRS depression scales (completed on the day of the binge study) when compared with the Beck, Hamilton, or Montgomery-Asberg depression rating scales (completed later). Hamilton, Beck, and Montgomery-Asberg ratings were significantly correlated with each other at the $P<0.01$ level.

Relation of caloric intake to mood

There was a weak positive correlation between rate of food consumption (calories per minute) during the binge-vomit period and both initial Beck scores ($r=0.653$, $P<0.05$, $n=12$) and Hamilton scores ($r=0.546$, $P=0.07$, $n=12$). Rate of caloric consumption showed a negative correlation with the percentage of carbohydrate in the chosen meal ($r=-0.591$, $P<0.05$) and a positive correlation with the percentage of fat ($r=0.554$, $P=0.06$). There was no relationship between alterations in depression and anxiety during the binge study and the subject's total caloric consumption, or her relative preference for carbohydrate, protein, or fat.

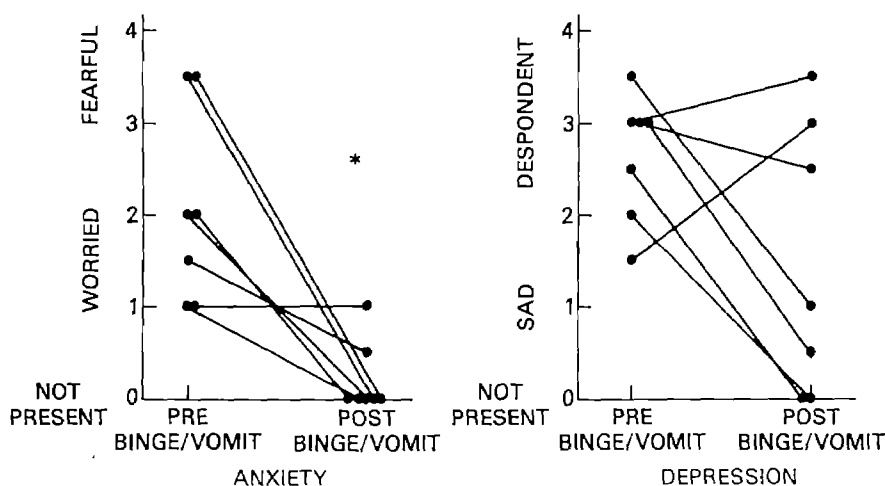


FIG. 3. Brief psychiatric rating scale (BPRS) scores for anxiety and depression in 7 bulimic subjects. Scores just before patients began bingeing and vomiting ('pre-binge/vomit') are compared to scores 20 minutes after they finished the last cycle of bingeing and vomiting ('post-binge/vomit').

Discussion

The results of this study demonstrate heterogeneous patterns of mood changes during and after bingeing and purging in bulimic patients. Abraham and Beumont (1982) found that 34% of patients got relief from anxiety *during* the cycles of bingeing and 66% reported freedom from anxious feelings *after* the cycles of bingeing had concluded. The corresponding subjective reports of decreased anxiety in our study (50% during bingeing, 67% after bingeing) were similar and were confirmed by objective ratings. Johnson & Larson (1982) did not measure anxiety but reported a general worsening of affective state during a binge in terms of greater guilt, shame and anger: after the binge-purge episodes were over, the patients in the Johnson & Larson study were sadder, drowsier, weaker, and more bored than usual. Similarly, many of our subjects remained depressed during and after bingeing and vomiting. The DSM-III states that although eating binges may be pleasurable, and vomiting often reduces post-binge anguish, depressed mood and self-deprecating thoughts follow eating binges. While our results generally support this statement, we would add that a reduction in anxiety or tension often occurs during or after bingeing and vomiting.

Episodes of bingeing and vomiting have been described as being associated with, or precipitated by, dysphoria or stress (Abraham & Beumont, 1982; Johnson & Larson, 1982; Strober, 1984). Our study was performed irrespective of pre-binge mood state. While it may be assumed that the bulimics were

stressed since this study was done on the day after hospital admission, the BPRS ratings only indicated mild-to-moderate anxiety and tension. In-patient studies, by their very nature, may produce artifacts because they take place in an environment different from that in which bingeing and vomiting usually occur. It is difficult to know how much environmental effects may have altered base-line mood and responses in our study. The bulimics did volunteer the information that they might not have begun a binge in these circumstances if on their own, but once in the midst of bingeing and vomiting they described their responses as fairly typical. In spite of these factors, many subjects had a decrease in dysphoria with bingeing and vomiting. The self-report literature suggests that many bulimics have a similar response in their more usual settings.

It is not clear what stimuli cause the subject to terminate an episode of bingeing and vomiting. Only 4 of our 12 subjects had an improvement in anxiety and/or depression (compared with base-line) at exactly the time they ended bingeing and vomiting. It is possible that we may not have rated the most significant change in mood: some subjects appeared to be giddy, silly, or somewhat intoxicated at the time they volunteered to stop bingeing and vomiting. It is also possible, as Johnson & Larson (1982) have suggested, that patients entering a treatment programme after years of bulimia may no longer be getting relief of symptoms by bingeing and purging. As with substance abuse, tolerance may develop over time: bingeing is then needed to maintain a dependency rather than to get high. Our bulimics had been

symptomatic for three or more years. We did not find relationships between duration of bulimia and observed response, but perhaps studying bulimics much earlier in the course of the disease would reveal more consistent mood changes.

This study confirms patient self-reports as to the number of calories and proportions of carbohydrate, fat and protein consumed during binges, as well as confirming that bulimics do not necessarily consume high-carbohydrate foods but may also consume high-protein or high-fat foods (Mitchell *et al*, 1981; Abraham & Beumont, 1982). Our data tend to suggest that the rate of consumption (45–166 kcal per minute) bears some relationship to the type of food eaten and to mood. The bulimics who ate the fewest calories appeared to preferentially select carbohydrate and to avoid fat-containing foods, whereas those that consumed the most calories per minute had the opposite relationship. Whether the most rapid eaters preferred fat-containing foods or just ate any available food is not clear since we did not give the bulimic subjects an unlimited supply of food to choose from. Food preferences during bingeing might reflect differences in hypothalamic neurochemical control of eating behaviour, since the choices of fat and carbohydrate may be regulated by separate systems (Leibowitz, 1980). Those bulimics that consumed the most calories per minute had the greatest depression ratings on global mood scale. If such a finding can be replicated it might suggest that endogenous neuromodulators that influence the rate of eating may differ between depressed and non-depressed bulimic subjects.

While bingeing and purging may often produce an immediate but brief reduction of anxiety and hunger, the relationship between bingeing and depressed mood is less clear. In this study many subjects were not substantially depressed (according to their global mood ratings) and only some of those that were depressed had a reduction of depression during bingeing. Thus bingeing does not appear to be used to 'self-medicate' an underlying depression. Alternatively, depression could be a secondary manifestation related to chronic bingeing and vomiting (Johnson-Sabine *et al*, 1984). An increase in negative mood states has been reported on days when patients engaged in bingeing and/or purging (Johnson-Sabine *et al*, 1984). Johnson & Larson (1982) have suggested that bingeing and purging might be a form of substance abuse, with extremes of food intake and/or vomiting used to improve mood briefly. The immediate brief rewards associated with bingeing and vomiting may be followed, at other times of the day, by periods of withdrawal or rebound that account for the increased dysphoria on global mood

scales. Thus bulimics may become trapped in vicious cycles of binge-induced reductions of dysphoria alternating with rebound, i.e. withdrawal-induced increase in dysphoria.

It is possible that some bulimics, particularly those without depression or any anxiety disorder, adopt binge-vomit cycles to counter a psychological or biological predisposition towards obesity. A few studies have suggested that bulimics might easily become obese (Pyle *et al*, 1981; Fairburn & Cooper, 1984). Similarly, it has been suggested that bingeing and purging might be a short-term solution to counter a drive to eat and gain weight (Chiodo & Latimer, 1983). Several studies have suggested that genetic vulnerability to obesity might be inherent in relatives of those with normal-weight bulimia (Fairburn & Cooper, 1984; Mitchell *et al*, 1981). In the present study, all three (25%) of the bulimics who had low global depression scores on admission (numbers 5, 8, and 10) had a past history of weight equal to or greater than 115% average body weight, suggesting that they might have a tendency towards obesity.

This study is, to our knowledge, the first naturalistic investigation using behavioural ratings during bingeing. Several methodological problems have appeared that should be borne in mind in future investigations. Several authors (Walsh *et al*, 1984; Johnson-Sabine *et al*, 1984) have suggested that many bulimics do not have a major depressive disorder, but rather have a fluctuating anxiety or tension disturbance: such behaviour would not be accurately measured by the standardised ratings that we used. We have frequently noticed alexythemia in bulimics; thus they may be poor witnesses to their own behaviour. Lastly, 'a reduction in anxiety' may not accurately describe the effects of bingeing, as some bulimics appeared to be intoxicated or 'spaced out'. Future studies should employ a broader range of objective behavioural ratings.

In summary, we have demonstrated in this study that patients with chronic symptoms of bulimia generally experience more marked reduction in anxiety than in depression during the course of bingeing and vomiting episodes. While patterns of bingeing and vomiting appeared to remain stable for each individual, different subjects demonstrated a wide range of mood responses. Further study of the mechanisms by which bingeing and vomiting alter mood states may be expected to contribute to the treatment of bulimic symptoms.

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Follow-up of Agoraphobic Patients Treated with Exposure In Vivo or Applied Relaxation

L. JANSSON, A. JERREMALM and L. G. ÖST

The present study describes the results of a 7-month and a 15-month follow-up of 32 agoraphobic patients treated with exposure *in vivo* or applied relaxation. During the follow-up period, all patients were given self-exposure instructions. Assessments were made in three response systems—subjective-cognitive, behavioural, and physiological—at the follow-up points. The study showed overall maintenance of treatment results in all three response systems for exposure-treated patients. For applied relaxation/self-exposure, there was a relapse on heart-rate at 7 months for physiologically reactive patients, but the improvement was regained at the 15 month follow-up. Furthermore, a large proportion of the total improvement occurred during the follow-up period: 36% and 22% for exposure and applied relaxation/self-exposure respectively. The proportion of patients reaching a clinically significant improvement was 50% at the end of treatment and 66% at the 15 month follow-up.

Different behavioural treatments have been recorded as having a powerful immediate effect on agoraphobia, with a mean of 63% of the patients being much improved after treatment by the most widely used method, exposure *in vivo* (Jansson & Öst, 1982). However, the long-term effects of behaviour therapy on agoraphobia are more uncertain, since few studies report on the long-term results (see Table I).

Marks (1971) reported the follow-up results on 36 agoraphobic patients treated with systematic desensitisation and other forms of psychotherapy. Four years after treatment the results were maintained on phobia ratings made by the patients and their therapists. Emmelkamp & Kuipers (1979) assessed 70 agoraphobic out-patients with self-report forms at four years after treatment with variations of exposure therapy. They found that the improvements were maintained during the follow-up period.

Four studies have used independent assessor ratings of agoraphobic symptoms, giving more credence to the follow-up results. Hafner (1976) administered self-report assessment forms to 39 patients treated with *in vivo* exposure one year after treatment, and found that one third of them were unimproved or worse at follow-up. However, on independent ratings of agoraphobic symptoms the improvements were maintained at one year. Munby & Johnson (1980) assessed 66 patients at 5-9 years after treatment, and they also used both self-report and independent assessor ratings. Both these

measures showed that the improvements were maintained, and no evidence of symptom substitution was found. Another investigation also used self-report and independent assessor ratings for 40 agoraphobic patients treated with imipramine or exposure (Cohen *et al*, 1984). Two years after treatment, two thirds of the patients remained improved or much improved in their phobias.

It seems that the effects of behaviour therapy for agoraphobia are maintained for several years after treatment, as evidenced by both subjective measures and independent ratings. However, little is known about the long-term stability of the behavioural changes in agoraphobic patients, since only two unpublished studies have included a behavioural test at follow-up. Burns *et al* (1983) reported the follow-up results at eight years of 18 out of 32 patients treated with different behavioural methods. At the follow-up point the patients were assessed by self-report, independent assessor ratings and a behavioural test. In general, the improvements were maintained on all three measures.

The primary purpose of the present investigation was to study the long-term effects of behavioural treatment on self-report, behavioural and physiological measures of agoraphobia. A secondary purpose was to compare the amount of change immediately after treatment with that after the follow-up period, and also to determine the clinical significance of the improvements at the different points of assessment.

TABLE I
Follow-up results of controlled studies on behavioural treatments of agoraphobia

Authors	n at follow-up	% of n at treatment	Measure ¹	Results			Total improvement ² %	Proportion of improvement during follow-up ³ %	% receiving further treatment
				Pre-treatment	Post-treatment	One year follow-up			
Marks (1971)	36	92	P+T	6.0	4.2	4.0	40	25	15
Hafner (1976)	39	95	I	5.8	1.97	1.77	69	5	?
Emmelkamp & Kuipers (1979)	70	86	P	6.05	3.45	—	49	12	19
Munby & Johnston (1980)	63	95	I	6.60	4.11	—	38	—	49
Burns <i>et al.</i> (1983)	18	56	B	5.1%	79%	83%	71	28	20
Cohen <i>et al.</i> (1984)	40	89	I	6.65	2.55	2.9	62	14	23

1 P=patient, T=therapist, I= independent assessor, B=behaviour test (distance walked)

2 Figure in brackets is time of later follow-up in years after treatment

3 On a scale of 0-8, either as originally reported or converted from a scale of 0-5

4 Difference between pre-treatment and lowest value as a percentage of pre-treatment value.

5 As a percentage of total improvement

Method

Patients and design

Details of the original design and treatment have been described by Öst *et al.* (1984). The subjects were out-patients at Ulleraker Mental Hospital, and fulfilled the following criteria.

- They were aged 20-60.
- The major presenting complaint was fear of, and avoidance of, being alone in public places from which escape might be difficult or help not available in case of sudden incapacitation.
- They have no other psychiatric problem in need of immediate treatment.
- If any medication was used the intake was to be held constant during the study
- They indicated their willingness to participate in the study for a period of three months.
- They received no other kind of psychiatric or psychological treatment except for the ongoing medication during the treatment.

Of the 40 patients that were included in the study, 38 were women and 2 were men. The sample's mean age was 36.2 years (range 25-52) and the average duration of the phobia was 8.3 years (range 1-28). Twenty-nine of the patients (73%) experienced panic attacks outside their phobic situations. Only 7 were drug-free at the start of treatment, while 20 were taking benzodiazepines, 11 some combination of benzodiazepines and other psychoactive drugs, and 2 were taking neuroleptics

Each patient was first classified as a behavioural or a physiological reactor depending upon their reactions on a behavioural test (Öst *et al.*, 1984). The patients were then randomly assigned to receive 12 sessions of either exposure *in vivo* (E) or applied relaxation (AR). The applied relaxation included practical training in how to use relaxation as a coping technique in difficult situations. The treatment was carried out by two experienced therapists with relevant training and five years of clinical experience in behaviour therapy.

A maintenance programme was delivered at the final session consisting of the following strategies: a list of high-risk situations; an individualised strategy in case of a set-back; a monthly self-monitoring schedule for self-exposure; and monthly telephone contacts with the therapist (Jansson *et al.*, 1984). All patients received this self-exposure component during the first five months of follow-up.

Assessment

All of the following assessments were made before and after treatment and (on average) 7 months and 15 months after the end of treatment.

Self-report. The patients completed a Swedish version of the Wolpe & Lang (1964) Fear Survey Schedule (FSS-III). To assess the specific agoraphobic reaction they filled out the Agoraphobia Scale (constructed by the authors) with twenty common agoraphobic situations, estimating both anxiety in entering the situation and degree of avoidance of the situation. Also, the patients filled out the Agoraphobia Questionnaire (Zitrin *et al.*, 1980) and panic attacks were

assessed with the Acute Panic Inventory (Zitrin *et al.*, 1980). The physiological reactions in anxiety-provoking situations were measured with the Autonomic Perception Questionnaire (Mandler *et al.*, 1958). To tap negative thoughts during the behavioural test the patients rated on a scale from 0 to 4 the frequency of ten negative self-statements. To measure the patients' degree of depression and marital satisfaction the Beck Depression Inventory (Beck, 1967) and Maudsley Marital Questionnaire (Crowe, 1978) were administered. Lastly, the patients also rated an individual hierarchy of the 15 agoraphobic situations used in the behavioural test, on a scale from 0–100 in anticipated anxiety.

Behavioural measure. A behavioural test was conducted: each patient was asked to enter real agoraphobic situations unaccompanied. From an individual hierarchy of 15 situations, the patients entered frightening situations until they failed or refused to enter two consecutive situations. The request to enter these situations was presented with emphasis on doing their utmost to provide a realistic picture of their performance and their physiological reactions in the phobic situation. At the post-treatment test the patients were asked to enter both the most difficult situation they had performed at the pre-treatment test and then to try the most difficult situation that they would dare to try after treatment. The test continued in the same way as before: until the patient failed or refused to enter two consecutive situations, or performed the most difficult situation of the hierarchy. The dependent behavioural measure was the percentage of situations completed.

Self-rating of anxiety. During the behavioural test the patients carried a portable ECG-cassette recorder (Medilog 4-2, Oxford Instruments) which also recorded their verbal ratings of anxiety every 2–5 minutes depending on the total estimated time in the phobic situation. The cue for the anxiety rating was a signal from a portable timer, set for the time intervals chosen for the situation. A microphone was attached to the patient's collar to record what was said in the situation as well as sounds from the surroundings.

Physiological measure. The patient's heart-rate was continuously recorded during the behavioural test through one of the four channels on the portable tape recorder. The patient's resting pulse was recorded while the patient was both seated and walking around for five minutes during the screening interview. In this way, the patient's Δ heart-rate (difference between resting pulse and heart-rate in the most difficult phobic situation) could be calculated.

Clinically significant improvement

In order to assess if the degrees of improvement shown by the patient's were clinically significant (Hugdahl & Öst, 1981) the criteria developed by Jacobson *et al.* (1984) were used. According to these, a patient's score on the outcome measure must fall outside the range of the agoraphobic population, where the range is defined as mean ± 2 s.d. As there are no norms for agoraphobic patients on the measures used in this study the sample's pre-treatment values were used. The criteria which had to be fulfilled were as follows: on the behavioural measure the patient had to complete 73% of the situations; the Δ heart-rate must not

exceed $+0.5$ beats/min, and the self-rating of anxiety had to be 0. An overall criterion was also constructed, according to which a patient had to be improved on any two of the three criteria above.

Results

Four patients dropped out from the study and did not complete the original treatment. Another three patients were not available for the 7 month follow-up, leaving 33 patients, 32 of these were available for the 15 month follow-up. The division of subjects into behavioural and physiological reactors did not yield significant differences in treatment effects for exposure *in vivo* and applied relaxation/self-exposure (Öst *et al.*, 1984), consequently the two response patterns were combined and data presented for the two treatment methods only.

Within-group changes

Student's *t*-tests for independent samples were computed to analyse the differences between pre-treatment and post-treatment testing, post-treatment and 7 month, and between 7 month and 15 month follow-ups.

Self-report measures. Both groups improved significantly after treatment on most of the measures. E on seven and AR on nine of the ten measures (see Öst *et al.*, 1984). A table of the complete data can be obtained from the authors.

At the 7 month follow-up, all self-report measures showed that the improvements were maintained for the exposure group. There were no significant differences between post-treatment test and the 7 month follow-up on any of the self-report inventories for those patients receiving exposure *in vivo*. For the applied relaxation/self-exposure group, all but two of the self-report inventories showed maintained improvements. On the Fear Survey Schedule there was a further significant deterioration ($P < 0.001$), and on the Acute Panic Inventory there was a significant deterioration ($P < 0.05$).

At the 15 month follow-up there was a significant further improvement on several of the self-report measures for the E group: Agoraphobia Scale (Anxiety) ($P < 0.05$), Individual Hierarchy ($P < 0.05$), Acute Panic Inventory ($P < 0.05$), APQ (Specific) ($P < 0.05$), and the Negative Thought Index ($P < 0.01$), on all other inventories the improvements were maintained but without further improvement. For the AR group there was a significant further improvement from 7 to 15 month follow-up on the Individual Hierarchy ($P < 0.01$), APQ (Specific) ($P < 0.01$) and on the Negative Thought Index ($P < 0.05$), while the other scales showed a maintained improvement.

Behavioural measure. The results on the behavioural test at 7 month and 15 month follow-ups showed a maintained improvement for both groups: there were no significant differences for either group between the results at post-treatment testing compared with 7 month follow-up, or between situations performed at 7 month and 15 month follow-up.

Physiological measure. There was a steady and continuing improvement for the exposure-treated patients in heart-

rate, responding from pre-testing, across post-testing and 7 month follow-up to the 15 month follow-up. No significant differences, however, were found between post-treatment testing and 7 month follow-up, or between 7 month and 15 month follow-up. However, for the AR group there was a significant increase in Δ heart-rate at the 7 month follow-up compared with post-testing ($P < 0.001$). This increase in physiological reactivity occurred mainly among the physiologically reactive patients. There was a significant difference between physiological (mean = 27.5) and behavioural reactors (mean = 0.8) on Δ heart-rate at 7 month follow-up ($P < 0.01$). At 15 month follow-up, however, there was again a significant improvement in Δ heart-rate compared with the results at 7 month follow-up ($P < 0.01$).

Self-rating of anxiety. Both groups improved significantly after treatment regarding the degree of anxiety experienced during the behavioural test. The E group continued to improve somewhat at the 7 and 15 month follow-ups. The AR group, however, displayed a significant deterioration ($P < 0.05$) at 7 month follow-up, paralleling that seen in the physiological measure. At the 15 month assessment the group's mean was again down at the post-treatment level.

Between-group comparisons. Analyses of variance (split-plot design) were computed to investigate between-group differences. These showed that the two treatment groups were equal on 10 of the 13 measures. Significant F values for the group factor were obtained on the Anxiety ($P < 0.05$) and Avoidance ($P < 0.05$) sections of the Agoraphobia Scale, and on the Agoraphobia Questionnaire ($P < 0.05$). Subsequent Tukey tests on the Agoraphobia Scale (Anxiety) showed that the AR group was better than the E group at the 7 month follow-up. On the Avoidance part, the AR group was better than the E group at the 15 month follow-up. Finally, on the Agoraphobia Questionnaire, the difference between the groups was due to a significant pre-treatment difference, which disappeared when analysis of covariance was used.

Improvement during treatment and follow-up. There was a large variation between the different measures, expressed as percentage change from pre-treatment values, with 13% for the E group on the Maudsley Marital Questionnaire (items 1–20) and 98% for the AR group on Δ heart-rate as the extremes. The means across all measures were 56% for the E group and 68% for the AR group.

If the improvement is divided into that occurring during treatment and that occurring during follow-up, the E group shows a continued improvement on all measures. The AR group, however, improved further on 11 of the 13 measures, the two exceptions being Δ heart-rate and Acute Panic Inventory. The proportion of each group's total improvement that occurred during follow-up was 36% for the E group and 22% for the AR group.

Clinical significance of the improvements

The proportion of patients who had improved to such an extent that it could be considered clinically significant is higher on the behavioural measure than on the subjective and the physiological measures. For exposure-treated patients, 59%, 65% and 71% were clinically improved on this measure directly after, at 7 month and at 15 month

follow-up respectively. For relaxation/self-exposure treated patients, the corresponding figures were 58%, 67% and 83%. For the three forms of measure taken overall there is a tendency for AR to yield a higher proportion of improved patients (AR: 58%, 50%, 72%; E: 41%, 47%, 59%). When the two groups are taken together, 50% are clinically improved at the end of treatment; this figure is increased to 66% at the 15 month follow-up. A comparison of the 15 month with the post-treatment outcome showed that only one patient had relapsed, while 17 had maintained their improvement and 6 had changed from not improved to clinically improved.

During the follow-up period eight patients (25%) received further treatment, five from the original exposure group and three from the applied relaxation/self-exposure group. All patients received exposure *in vivo* for 3–10 sessions (mean = 5). However, four of these patients showed no improvement at any assessment point and were clearly treatment 'failures' in spite of further treatment. Another changed from clinically improved to not improved at the 7 month follow-up, and requested further treatment, which led to his regaining the status of clinically improved at the 15 month assessment. The remaining three patients, who were not improved at post-treatment, changed to clinically improved at follow-up as a result of obtaining further treatment.

Discussion

This is, to our knowledge, the first published study which reports follow-up results for subjective, behavioural and physiological measures. The study showed that the improvements on subjective and behavioural measures were maintained up to 15 month follow-up for exposure-treated patients, and that there was a non-significant trend of continuing improvement in anxiety-ratings and Δ heart-rate among physiologically reactive patients at 7 month follow-up. However, at 15 month follow-up the improvements in all the three response systems were maintained. One possible explanation for the deterioration at 7 months for the AR group could be that they had difficulties with continuing to practice and with applying relaxation techniques during the first follow-up period. However, because of the maintenance programme, which involved self-exposure practice, the behavioural gains were maintained and eventually resulted in a satisfying long-term result at 15 month follow-up. It could be that physiologically reactive patients treated with applied relaxation need an individually tailored maintenance programme containing prompts to continue to practice and to apply relaxation techniques. These specific elements could be incorporated into a standard maintenance programme which is more oriented towards self-exposure (Jansson *et al.*, 1984). This investigation shows a change on the behavioural measure (E group 61%; AR group 72%) which is comparable with the

71% found by Burns *et al* (1983). Also, the changes in ratings of anxiety are in the expected range (E group 57%; AR group 66%).

However, when looking at all the outcome measures taken together, it was found that 36% and 22% of the improvements for exposure and applied relaxation/self-exposure treated patients respectively occurred during the follow-up period. These are more promising results than usually found in follow-up studies (see Table I, where the figures range from -14% to +25%). The findings of a continuing improvement at follow-up is encouraging; one possible explanation for it could be the maintenance programme employed in this study. Self-exposure alone has been found to be as effective as therapist-directed exposure (Gosh *et al*, 1984), and it therefore seems reasonable to assume that the self-exposure component of our maintenance programme was responsible for the continuing improvement found in this study. Therapy with agoraphobic patients

should contain a maintenance programme of self-exposure for at least the first half-year after treatment to ascertain continuing improvement. However, further research is needed on how to administer an effective maintenance programme.

The proportion of patients having a clinically significant improvement was surprisingly high (66%) in the present study. These results give further credence to the clinical usefulness of the treatments used. It is suggested that future studies in the treatment of phobias should use the same way of determining clinical significance (Jacobson *et al*, 1984), so that a more universal way of comparing results will be possible.

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Depression and Anxiety in Patients Undergoing Diagnostic Investigations for Head and Neck Cancers

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Patients attending a regional head and neck Oncology Unit were assessed for depression and anxiety (using the Leeds self-assessment scales) before diagnostic biopsy investigations. At the time of the assessments, neither patients nor investigators knew the biopsy results. Patients whose biopsies subsequently proved positive (showing the presence of a tumour) had significantly higher depression scores than those whose biopsies were negative. Differences in mean anxiety scores did not differ between groups. It is suggested that depression is part of the clinical picture of head and neck cancer.

Interest in the psychological concomitants of cancer has increased in recent years (Greer & Silberfarb, 1982; Feinstein, 1983; Wellisch *et al*, 1983; Manos & Christakis, 1985). Five areas of psychological research have been identified: the psychological morbidity of cancer and its treatments; precursors of the disease; the relation of psychological characteristics to outcome; psychobiological studies; and intervention studies (Greer & Silberfarb, 1982). In head and neck cancer psychological research has focused on the first area, especially on the quality of life of patients following treatment (Olson & Shedd, 1978; Shedd *et al*, 1980; Morton *et al*, 1984). Morton *et al* report that 40% of patients treated for buccopharyngeal cancers satisfied the DSM-III criteria (American Psychiatric Association, 1980) for depression six months after active treatment ended; i.e. depression is part of a pattern of residual disability in these patients. Depression was not related to the type of cancer treatment received, and was itself largely untreated. Massie & Holland (1984) point out that depression in cancer patients is under-diagnosed and undertreated partly because it is believed to be reasonable for cancer patients to be depressed and partly because it is believed that all cancer patients are depressed from the start of their illness. It is not known what proportion of patients are depressed at the beginning of their illness, nor at what stage (if any) depression deepens.

In order to investigate mood disorder from the start of the patient's hospital attendance, mood levels were studied in a group of patients undergoing biopsy as part of a procedure to investigate possible buccopharyngeal tumours. The study was carried out in the same unit as was the work of Morton *et al* cited above, but deals with the early contact of patients with the unit.

Method

Sample

Seventy-five patients (46 men and 29 women) admitted to a regional head and neck oncology unit for diagnostic investigations were given the Leeds scales for self-assessment of anxiety (SAA) and depression (SAD) (Snaith *et al*, 1976); data from the General Health Questionnaire (Goldberg, 1978) were available for 44 patients. Patients were referred from a variety of sources, including general medical and dental practitioners and other hospital departments, and were examined in an out-patients clinic. If there was any suspicion that the patient had a neoplasm, arrangements were made for a 48 hour in-patients stay, during which time a biopsy was carried out. The psychological interview was carried out during this period, on the day before the biopsy investigation.

Data were discarded for three patients (one was discharged before biopsy, one did not reattend, and one had a pre-existing psychiatric disorder); analysis of data was based on the remaining 72 patients. The sample was collected in two stages, each part of separate larger studies (DelPo, 1985), but using the same procedures. There were 18 men and 10 women at stage 1 (mean age 51.9, s.d. 16.0), of whom 17 had a negative biopsy result (i.e. no evidence of a malignant neoplasm was found); the other 11 patients examined had evidence of a malignant buccopharyngeal tumour. Six of the 11 patients were suffering recurrence of a previously treated neoplasm. At stage 2 there were 44 patients, 26 men and 18 women (mean age 54.0, s.d. 8.5), 17 of whom had no evidence of malignancy and 27 of whom were found to be suffering from a buccopharyngeal cancer.

Mood measures

The Leeds scales are self rating scales with four alternatives provided for each of 15 statements, e.g. "I feel scared or frightened". Possible replies are "Yes, definitely"; "Yes, sometimes"; "No, not much" and "No, not at all". Since some of the patients were elderly or of low educational level, the paper and pencil format was modified; all items and

response categories were retained, but the items were read out by an interviewer. The interviewer, a psychologist, went through each item with the patient and ascertained the response that the patient said best fitted what he or she felt.

The scales provide a six item anxiety scale and six item depression scale, with a 'general' and a 'specific' measure for each. The general measures are designed to be used as indicants of severity of symptoms in patients who have not received a primary diagnosis of affective illness, this is the measure appropriate for the present study (The 'specific' measure is concerned with severity of diagnosed affective illness.) The maximum score possible for depression (SAD) and for anxiety (SAA) is 18. The recommended cut-off is between 6 and 7, and scores on each subscale above 6 are regarded as indicating anxiety or depression (Snaith *et al*, 1976). (In the present study the Leeds scales were used to investigate mood, not as a diagnostic tool.) The General Health Questionnaire (30 item version) was scored according to the bimodal method recommended by Goldberg, (1978); patients scoring over 4 were considered to have a mood disturbance.

Results

Data from patients in stages 1 and 2 were examined using *t*-tests for independent groups. There were no significant differences for age, or anxiety and depression scores. Further analysis were therefore carried out on the combined group of 72 patients.

Table I shows the means and standard deviations of self-assessed depression and anxiety and the number above the recommended cut-off points both for the whole sample and broken down by biopsy outcome. Separate analysis of variance of SAD and SAA scores (with age and sex as covariates) were carried out with biopsy outcome as an independent variable. Biopsy outcome was a significant

factor for depression scores ($F=5.59$, d.f. 1,68; $P<0.02$), but not for anxiety scores ($F=1.64$; d.f. 1,68; NS). The covariates age and sex did not exert a significant effect. Raw score correlations of the SAD and SAA with age were not significant (confirming the results of Snaith *et al*, 1976). The analyses showed that patients with positive (malignant) outcome had higher depression scores than those with negative (non-malignant) outcome. The direction of result was the same for anxiety. Analysis of variance of GHQ scores (with age and sex as covariates) showed no relationship between mood disturbance and biopsy outcome, however ($F=0.7$, NS). GHQ raw scores showed a weak significant negative correlation with age ($r=-0.25$, $P<0.05$) and, in the 44 subjects for whom scores were available, moderately high positive correlations with SAD ($r=+0.64$, $P<0.001$) and SAA ($r=+0.47$, $P<0.001$).

The number of patients with positive biopsy scoring above the cut-off for SAD was double that of those with non-malignant outcome. Rates of SAA and GHQ mood disturbance were comparable over the two groups. In no case, however, did differences reach statistical significance.

In the procedure section of this paper it was mentioned that the two stages in which the present sample was collected were each part of larger studies. Although examination of the distribution of scores suggested that data could be pooled, nevertheless the data were collected 12–18 months apart. It was observed that the proportion of positive biopsies in the clinic changed over that interval, from 39% to 61%. The criteria for inclusion in the study did not change; eligible patients were those admitted for biopsy. However, introduction of out-patient diagnostic facilities in the interval between the two stages made it possible for patients with easily accessible tumours to be examined at their first visit to the clinic. Comparison of mean SAD and SAA scores of patients in stages 1 and 2 showed non-significant differences for those with negative biopsy outcome; however, differences were larger for those with positive outcome, and in each case scale scores were higher for patients in stage 1. One inference is that the out-patient facilities filtered out patients with easily accessible (e.g. mouth) tumours who nonetheless had substantial psychiatric distress. The higher rate of malignancies in stage 2 coupled with lower (although not significantly so) levels of mood-related symptoms is consistent with this.

Discussion

The finding that patients who are found subsequently to have buccopharyngeal tumours have higher depression scores prior to biopsy than those with negative biopsy results was not expected, and must be regarded as tentative, but it accords with some previous reports. Brown & Paraskevas (1982) reviewed the literature and suggested that depression may present as an early symptom of cancer, antedating discovery of the malignancy. This would have implications for early diagnosis and the prevention of avoidable delay in investigation. In the case of head and neck cancers, however, the effect may also

TABLE I
Means and standard deviations of mood measures and patients scoring above the test cut-off points

	<i>n</i>	Mean	<i>s d.</i>	Above cut-off	
				<i>n</i>	%
All patients					
Depression (SAD)	72	4.5	2.9	16	22
Anxiety (SAA)	72	6.0	3.2	29	40
GHQ	44	4.4	2.3	19	43
Biopsy positive					
Depression (SAD)	38	5.1	3.0	11	29
Anxiety (SAA)	38	6.3	3.1	15	40
GHQ	27	4.6	2.6	13	48
Biopsy negative					
Depression (SAD)	34	3.8	2.5	5	15
Anxiety (SAA)	34	5.6	3.3	14	41
GHQ	17	4.2	1.9	6	35

be due to the patient's subjective appraisal of the seriousness of the physical symptom: patients with hoarseness or difficulty in swallowing attributable to a tumour may have more profound discomfort or interference with functioning than patients whose symptoms have other causes. Additionally, the non-verbal and verbal interaction of medical staff with patients (e.g. reassurance or its lack) are likely to be different even before biopsy. However, these factors do not explain why the psychological effect is more marked for depression than for anxiety. Brown & Paraskevas's preferred hypothesis is in terms of a specific immunological interference with the activity of serotonin. We have no relevant data on this, but dissociation between depression and anxiety (measured by the Leeds scales) is suggestive. No significant differences were found using a less specific measure of psychiatric disturbance (GHQ).

Maguire (1984) has drawn attention to the lack of information concerning prevalence of affective disorders in head and neck cancers. He advocates screening patients with GHQ as one strategy to increase physicians' sensitivity to the presence of mood disturbance. In the present study 48% of those with

positive biopsy fell above the GHQ cut-off, almost 40% were above the Leeds SAA (anxiety) cut-off and almost 30% were above the SAD (depression) cut-off. These findings, in conjunction with those of Morton *et al's* (1984) study of patients at a later stage in the treatment process, suggest that symptoms of depression are part of the clinical picture of head and neck cancer both early and late in its history. Clinic staff also need to be aware of the high anxiety of those undergoing diagnostic investigations for buccopharyngeal cancers, even among those patients who are subsequently found not to have a malignant neoplasm.

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Editorial note

The Leeds Self-Assessment Scale has undergone further revision in order to improve suitability for assessment of mood disorder in the setting of physical illness. The revised instrument is the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983).

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Post-ECT Blood Pressure Rise and its Relationship to Cognitive and Affective Change

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Post-ECT blood pressure rise and its relationship to cognitive and affective change were studied in 29 depressed patients, using the Benton Visual Retention Test, the Hamilton Rating Scale for Depression, and the Leeds Self-Assessment Scale for Depression and Anxiety. No real difference was established in the Benton Test error scores before and after ECT, no correlation between rise in blood pressure and change in Benton Test error score with ECT was found, and no correlation was found between blood pressure rise and affective improvement following ECT. Previous studies which reported such correlations are not supported.

Perrin (1961), in his comprehensive review of the cardiovascular aspects of electroconvulsive therapy (ECT), noted that marked increases in systolic blood pressure (BP) immediately following ECT are the rule, whether or not muscle relaxants are used in the procedure. This elevation of BP has been associated with research into other aspects of the effects of ECT, including its effect on cognitive functioning (Hamilton *et al*, 1979; Stoker *et al* 1981). Using the Benton Visual Retention Test (BVRT) as a measure of cognitive function, Hamilton *et al*, (1979) studied 27 depressed patients before and after ECT, the second cognitive assessment being made three to four hours after the treatment. Only six patients had improved performance on the BVRT after ECT, the remainder showing impairment. It was concluded that impairment of performance on the BVRT with ECT was highly correlated with the maximum rise in systolic BP immediately following the convulsion. If this finding were confirmed, it would raise the possibility that cognitive impairment associated with ECT might be reduced or eliminated through devising means of avoiding the rise in BP (Kendell, 1981).

A further study by the same group (Stoker *et al*, 1981) re-examined these issues using an electronic sphygmomanometer. Three patterns of BP response to ECT were found, each showing different levels of cognitive performance, as measured by the BVRT four to five hours after ECT. However, in contrast to the previous study, the mean BVRT difference score for the total patient group was not significantly different from zero. Within the total group, though, an 'early rise' group was isolated with a peak BP rise occurring within one minute of shock, and with decline to near baseline levels within three minutes.

The mean Benton difference score for this group was in the direction of impairment. The two other groups isolated (a 'late rise' group with peak systolic BP reading occurring from one to three minutes after the shock, with subsequent slow recovery ($n=8$), and a 'no response' group ($n=7$), with BP rises of less than 20 mmHg) actually demonstrated improved Benton scores.

Taylor *et al* (1985) studied 20 psychiatric in-patients before and after five bilateral ECTs for major depression. Elevation of BP after ECT, using a standard sphygmomanometer, was studied using the following variables: mean peak elevation over pre-treatment levels, mean peak blood pressure during five treatments, mean percentage rise over the pre-treatment level, peak systolic BP, and peak diastolic BP. Patients showed a mean rise of 45 mmHg over pre-ECT levels; this rise was compared with any significant memory or neuropsychological changes after treatment, and also with affective change. The neuropsychological screening battery included a test of visual memory (not the BVRT). Patients were assessed before ECT and 24-48 hours after their fifth ECT. Significant deficits occurred in backward spelling (Mini Mental State Examination), verbal fluency, temporal memory, and visual memory, but none of these changes correlated with any measure of blood pressure elevation. The authors speculated, however, that the BVRT might have been more sensitive to such vascular changes after ECT.

Our study, using the BVRT, re-examined these issues. The test is one of visual memory, the performance of which requires a combination of spatial perception, short-term retention and recall and visuo-constructive abilities (Anastasi, 1968). It has

been used in several studies on post-ECT memory changes (Strain *et al*, 1968; Bidder *et al*, 1970; Hamilton *et al*, 1979; Stoker *et al*, 1981).

We also examined the suggested relationship between post-ECT rise in BP and affective change (Hamilton *et al*, 1979; Taylor *et al*, 1985). Hamilton's group speculated that the therapeutic efficacy of ECT might be associated with the rise in BP through an unknown mechanism – perhaps changes in cerebrovascular permeability. Taylor *et al* (1985), however, were unable to find any correlation between reductions in rating scores of depression and various measures of BP elevation during a course of treatment. We sought evidence of such an association, for a positive correlation between post-ECT rise in BP and reduction in depression would be an important finding to simulate new work into the mechanism of the antidepressant action of ECT.

Method

Patients were studied before and after they had received ECT, each patient being sampled on only one day. The experimental group consisted of 20 females and 9 males, with an age range of 23–70 years (mean 44.6 years). Patients were referred by their psychiatrist for treatment of unipolar depression (23 patients), bipolar depression (three patients), or depression occurring during a schizoaffective illness (3 patients). Eleven patients were studied at the time of the first ECT of their current admission; for six of these it was their first ECT ever. Seventeen patients were tested on the day of their second to eighth treatment, and one patient on her twelfth treatment. Exclusion criteria were: clinical suspicion of organic mental change, history of brain damage, neurological disease, malignancy, or recent history of epilepsy.

ECT was administered using a Mark III or Mark IV Ectron apparatus, the waveform being sinusoidal in 28 patients and brief pulse in one patient. Electrode placement was bilateral fronto-temporal in all patients. The anaesthetic used was thiopentone (200–300 mg), the muscle relaxant was suxamethonium (25–40 mg), and atropine (0.6 mg) was administered with the anaesthetic. Cognitive assess-

ment was completed in 26 patients, using the BVRT forms C and D. Assessment of affective state was carried out using the Hamilton Rating Scale for Depression (HRSD), the Leeds Self-Assessment of Depression Scale (LSADS), and the Leeds Self-Assessment of Anxiety Scale (LSAAS). Pre-treatment cognitive and mood assessments were performed on the afternoon before treatment, and post-treatment assessments made five to six hours after ECT. Systolic BP was measured by the same doctor in all cases, using a standard sphygmomanometer, before and after administration of the anaesthetic and after that of the muscle relaxant. A series of 12 readings were taken over a period of five minutes after convulsion. BP rise during ECT was taken as the difference between the post-anaesthetic level of systolic BP and the maximum level attained after the administration of ECT.

Previous studies indicated that on average, systolic BP rose, BVRT error scores increased, and mood ratings improved with ECT. For this reason the one-tailed *t*-test was used to analyse BP rises, and the one-tailed Wilcoxon Matched Pairs Signed Rank Test used for the changes in the BVRT error scores and in depression and anxiety ratings.

Results

The results showed that BP does increase on average after ECT for the total patient group, for those receiving their first ECT, and for those on their subsequent treatments (Table I). All patients showed peak rise in systolic BP within the first minute following the convulsion. However, five of our sample showed BP elevations of less than 20 mmHg, falling into the 'no response' group described by Stoker *et al* (1981).

Analysis of changes in the BVRT error scores with ECT revealed no real difference in the number of errors made before and after ECT for the total patient group or for each of the sub-groups (Table II). Analysis of changes in affective ratings showed significant improvements in affective state in all three groups of patients after ECT, although improvement was at the borderline of significance in case of the LSADS in the subsequent ECT group.

A plot of rise in systolic BP with ECT against changes in BVRT error score (Fig. 1), in HRSD scores (Fig. 2), in LSADS scores and in LSAAS scores demonstrated no significant relationships.

TABLE I
Systolic blood pressure rise following ECT

	First ECT group	Subsequent ECT group	Total group
<i>n</i>	11	18	29
Mean rise, mmHg	40.0	47.2	44.5
s.d., mmHg	30.8	31.3	30.7
Range, mmHg	0–100	5–110	0–110
<i>t</i>	4.30	6.41	7.79
<i>P</i>	<0.001	<0.001	<0.001

TABLE II
Change in BVRT error scores with ECT*

	First ECT group	Subsequent ECT group	Total group
<i>n</i>	11	15	26
Mean change	0.3	0.0	0.1
s.d.	5.8	3.3	4.4
Range	-8 to +8	-5 to +6	-8 to +8
Wilcoxon statistic	5.0	59.0	170.5
<i>P</i>	NS	NS	NS

*A negative value indicates less error after ECT.

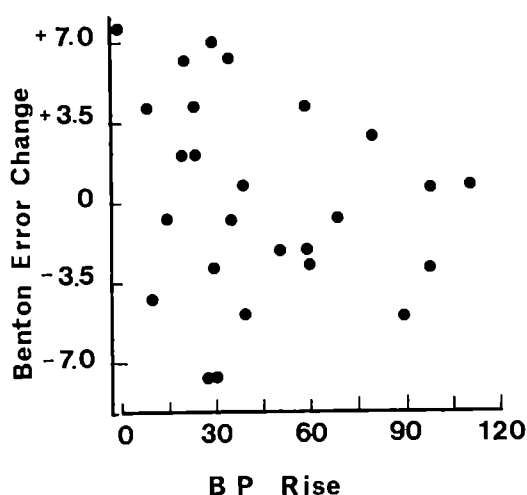


FIG. 1 Plot of systolic blood pressure rise (mmHg) with ECT against change in BVRT error score ($n=26$).

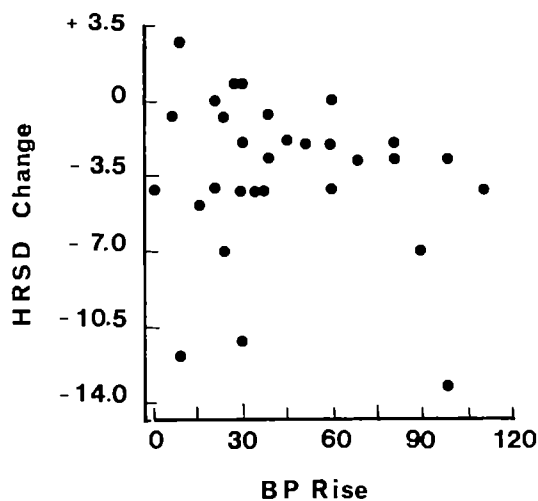


FIG. 2 Plot of systolic blood pressure rise (mmHg) with ECT against change in HRSD score ($n=29$).

Discussion

No difference was found in the number of errors made before and after ECT on the Benton Visual Retention Test. Furthermore, no correlation was established between rise in BP with ECT and BVRT error score change.

These findings are at variance with those of Hamilton *et al* (1979), but are in partial agreement with their study (Stoker *et al*, 1981), which failed to find a significant correlation between peak BP elevation and the BVRT difference score for the total group of patients. We note that post-ECT cognitive assessment in their first study was carried out after three to four hours, and in their second study after four to five hours, while in our study it was after five

to six hours. Ottosson's statement on the need for consistency in the time-intervals between ECT and cognitive testing may well be relevant here.

Stoker *et al* (1981) did, however, report a linear relationship between 'initial' BP rise, occurring within seconds of ECT, and the mean BVRT difference score recorded for their three patient subgroups ('early rise', 'late rise', and 'no response'). We did not find any patients corresponding to their 'late rise' subgroup, for the maximum elevation of BP invariably occurred at the first post-ECT measurement, within seconds of the treatment. We are not able to comment further, therefore, on the significance of the linear relationship between initial BP elevation and cognitive change which they identified. Five of our sample did fall into the 'no response' subgroup—a

similar proportion to that reported by Stoker *et al.* Two of our 'no response' group showed reduced error scores after ECT, but, in contrast to the Stoker *et al* findings, three patients showed increased errors. Due to the small size of this group in each study, it would be premature to draw any firm conclusions, but we cannot support the contention of Stoker *et al* that the 'no response' group show improved BVRT performance.

Our finding of lack of correlation between rise in BP and cognitive change following ECT is similar to the results of Taylor *et al* (1985), and gives a negative

answer to their question as to whether or not the BVRT might be a more sensitive test in this respect. Again, it is relevant that their methodology in relation to the timing and measurement of rise in BP and cognitive change differed from ours and from that of Hamilton's group.

Finally, no correlation was established in our study between rise in BP and changes in the Hamilton and Leeds affective ratings after ECT. These findings are in agreement with Taylor *et al* (1985), and do not support Hamilton's speculation about such an association (Hamilton *et al*, 1979).

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Is There a Lithium Withdrawal Syndrome?

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Patients who discontinued lithium after a mean of 29 ± 21 months prophylaxis were compared with a similar group who were not taking the drug; all had only been in hospital once, and satisfied DSM-III criteria for mania. During the first three months following withdrawal of lithium, significantly more patients relapsed than in the control group; after this period, the rate was identical. These findings suggest that lithium withdrawal exists as a clinical phenomenon and that a period of lithium treatment does not improve the long-term prognosis, once the drug has been stopped.

Discontinuation studies in the early 1970s were concerned with proving the efficacy of lithium as prophylactic medication, and consequently did not look closely at the possibility of relapse being caused by drug withdrawal. Little attention was paid to the temporal relationship between discontinuation and relapse. It was generally assumed that the pattern of relapse reflected a recrudescence of the natural history of the disease process in a unprotected individual, rather than a drug-induced state. Early relapses were noted in some studies (Melia, 1970), but did not occur in sufficient numbers to arouse comment (Grof *et al*, 1970; Baastrup *et al*, 1970). One of the foremost authorities on lithium therapy has stated that he has never observed withdrawal phenomena or rebound illnesses (Schou, 1980). However, if there is a genuine withdrawal phenomenon it would have important implications both for the method used to discontinue patients from their treatment and also for the management of a rapidly recurring episode after stopping lithium.

Lapierre *et al* (1980) reported 20 cases who had their lithium withdrawn on an open basis, for five days; four patients relapsed within this time and required admission to hospital for mania. This report seems to provide some convincing evidence of a rebound phenomenon, although it has to be interpreted with caution: three of the relapsed patients had experienced a mean of two affective episodes per year in the period between first contact with psychiatric services and the prescription of lithium. Their rapid relapses could therefore be a function of their severe illnesses, rather than a result of lithium withdrawal.

Klein *et al* (1981) switched 21 patients to placebo, but it is not clear whether this was under single-blind conditions; the diagnosis was of affective illness, but no criteria for this are stated. Although 11 patients

were reported to have "relapsed into severe psychotic states with paranoid, manic and depressive symptoms within 14 days", it does not appear that any of these were severe enough to warrant admission. All subjects restarted lithium after five weeks.

Christodolou & Lykouras (1982) studied 18 patients (17 of them bipolar) and reported that three relapsed within four days of double-blind placebo substitution. Two of these relapses were reported as severe and one required ECT, but the severity of symptoms was assessed by rating scales, and none of the patients required admission. Among the assessments made on these patients was a scale measuring side-effects such as anxiety, tremor, restlessness, fatigue, irritability, and other symptoms normally associated with a drug withdrawal state. No increase in these was noted.

King & Hullin (1983) described four patients who suffered minor symptoms of heightened anxiety, irritability, and emotional lability as a result of lithium withdrawal; none of the patients had affective relapses, and none required admission to hospital. Withdrawal symptoms were then studied retrospectively by questionnaire, but usable replies were obtained from only 18% of the patients approached, and such a biased sample means that little reliance can be placed on their stated results.

Sashidharan & McGuire (1983), in a carefully conducted retrospective study of 22 patients satisfying Feighner criteria for primary affective disorder, could find no evidence of a rebound phenomenon: only two patients relapsed during the first two months after discontinuation. In all, 15 patients relapsed, the majority staying well for over one year, and they concluded that there was 'a gradual pattern of relapse over a period of months following discontinuation'.

Method

The study group was drawn from patients admitted for the first time between 1 January 1970 and 31 December 1981; all satisfied DSM-III criteria for mania (American Psychiatric Association, 1980). Patients with puerperal illness or concurrent organic brain disease, or who were not resident in the UK at the time of their attack, were excluded. Two patients were excluded who committed suicide – one during a first admission and the other on the day of discharge. Also excluded were patients who did not have a clear gap between the stopping of their lithium and the onset of symptoms, as recorded in their case notes.

To be included in the study, patients in both the experimental and control groups had to remain well for at least three months following discharge, to ensure that any subsequent readmission was for a genuine relapse, as opposed to a recurrence of symptoms from the original episode. This excluded a similar number of patients from each group (29% of those on lithium and 22% of those that were not, $\chi^2 = 0.52$, d.f. = 1, NS). The control group therefore consisted of 50 patients, 42 of whom had never been on lithium, and eight who had only taken it for a period of less than three months; the experimental group consisted of 29 patients, established on prophylactic lithium for at least three months following discharge. Any patient who had three successive serum lithium estimations during follow-up of less than 0.4 mmol/litre was to be excluded from the study, but none were found. The patients subsequently took lithium for a mean of 29 ± 21 months.

From the admission notes and nursing records, demographic and clinical factors were recorded including age, sex, previous number of affective episodes requiring treatment but not admission, duration of admission, and family history of affective disorder. The severity of illness was estimated by the Modified Manic Rating Scale (MMRS) (Blackburn, 1977), which was further modified so that it could be used retrospectively on the admission notes. Inter-rater reliability with a rater blind to the purpose of the study gave a correlation of $r = +0.71$, which is in good agreement with the value quoted by Blackburn for the interview situation ($r = +0.68$ to $+0.79$). All patients and their GPs were contacted to provide a full follow-up picture until the close of the study (31 December 1983). The follow-up thus ranged from 2 to 14 years.

The outcome of the two groups was assessed by the life table method, which is the method of choice for analysing longitudinal data (Fleiss *et al.*, 1976). Relapses during the course of manic-depressive illness have been shown to be exponential (Grof *et al.*, 1970; Schou *et al.*, 1970), and when plotted on a semi-logarithmic scale give a straight line, the slope of which is the 'decay constant'. Data from all patients in the study were used. A common starting point (time 0) was defined; at this point, formal follow-up began. For the control group, time 0 was three months after discharge from hospital, when they were considered to have recovered from their first illness. At this point, the experimental group had been established on lithium for three months; time 0 for this group was the point at which they subsequently discontinued their medication. Successive probabilities of remaining well over three month intervals from these points were

calculated for both groups, relapse being defined as readmission to hospital.

Accurate information regarding the date of discontinuation of lithium was essential. Out of the 17 cases that were readmitted, seven had discontinued their drug in consultation with the medical staff, and this was clearly recorded; they subsequently developed symptoms and were readmitted. In another five cases, it was recorded at the time of their readmission that they had stopped their lithium, and developed symptoms 5, 7, 9, 14 and 21 days later (in two of these cases the dates of stopping lithium and the subsequent re-emergence of symptoms were corroborated by the patients' wives). In a further two cases, information regarding discontinuation and subsequent readmission were supplied by other hospitals. Finally, in three cases, it was recorded that the patients stopped their lithium and then developed symptoms, but the exact date of discontinuation was not clear. In these cases the date of the last serum lithium determination was used, as supplied by the lithium office, which records the lithium levels of all patients in the region. These patients were readmitted two months, one year and four years later. This method is unlikely to have introduced any error into the results, since only one patient was involved in the period of significant difference between the groups. Since the analysis of outcome after discontinuation was calculated in three month periods, this patient would, in any event, appear in this first period.

Any retrospective study is dependent on the quality of the case records. This study was greatly facilitated because in Edinburgh all notes are typed before inclusion in the file, and all first admissions have a full and detailed case summary, prepared after discharge.

Results

Although patients were not randomly allocated to experimental or control groups, they did not differ on any factors that have been proposed as predictive of outcome. Thus, mean age was 34 ± 13.5 years (control 37 ± 14.2 years), number of patients with previous episodes was eight (control seven, $\chi^2 = 2.2$, d.f. = 1, NS), family history of affective disorder 11 (control 12, $\chi^2 = 1.9$, d.f. = 1, NS). Length of hospital admission did not differ: 7.1 ± 3.5 weeks (control 5.7 ± 3.5 weeks, $t = 1.67$, d.f. = 77, NS). The diagnostic breakdown of the relapses in the two groups was similar: of the experimental group, 13 (76%) were manic, compared

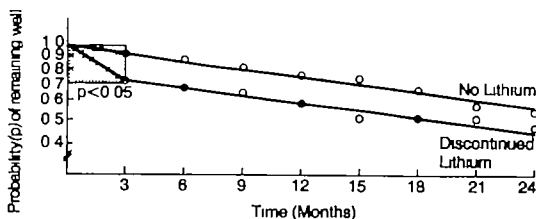


FIG. 1 Semi-logarithmic plot for discontinued lithium ($n = 29$) and control ($n = 50$) groups. Slopes were significantly different over the first 3 months ($P < 0.05$).

TABLE I
Life table for discontinued lithium group ($n=29$)

Interval months	No. starting interval well ($=a$)	No. failing in interval ($=b$)	Interval-specific probability of failure ($=c=b/a$)	Interval-specific probability of remaining well ($=1-c$)	Cumulative probability of remaining well
0-3	29	8	0.28	0.72	0.72
4-6	21	1	0.05	0.95	0.68
7-9	20	1	0.05	0.95	0.65
10-12	19	2	0.11	0.89	0.58
13-15	17	2	0.12	0.88	0.51
16-18	15	0	0.00	1.00	0.51
19-21	15	0	0.00	1.00	0.51
22-24	15	1	0.07	0.93	0.47

TABLE II
Life table for control group ($n=50$)

Interval months	No. starting interval well ($=a$)	No. failing in interval ($=b$)	Interval-specific probability of failure ($=c=b/a$)	Interval-specific probability of remaining well ($=1-c$)	Cumulative probability of remaining well
0-3	50	4	0.08	0.92	0.92
4-6	46	2	0.04	0.96	0.88
7-9	44	3	0.07	0.93	0.82
10-12	41	3	0.07	0.93	0.76
13-15	38	1	0.03	0.97	0.74
16-18	37	4	0.11	0.89	0.66
19-21	33	4	0.12	0.88	0.57
22-24	29	2	0.07	0.93	0.54

with 17 (68%) of the controls. The only criterion on which they did differ was severity of initial illness as rated by the MMRS (experimental group 28.0 ± 7 ; control group 22.4 ± 8 ; $t=2.67$, d.f.=76, $P<0.01$). All chi-squared analyses included Yates' correction factor for continuity; t -tests were 2-tailed for independent samples.

The life tables (Tables I and II, Fig. 1) show that there is an increased risk of relapse over the first three months following the discontinuation of lithium, compared with the first three months for the control group after recovery from their initial illness ($z=2.17$, $P<0.05$). After this period, the decay constants (rates of relapse) for the two groups are identical.

Discussion

This is the first study to report on a group of bipolar I patients after a first illness, with a similar group as a control. Readmission rather than the recurrence of symptoms was chosen as the outcome measure, since it is much more accurate in a retrospective study.

The increased risk of relapse over the first three months following the discontinuation of lithium lends support to the hypothesis that a withdrawal state does exist. As documented above, symptoms tended to recur rapidly, most patients being readmitted within this first follow-up period. It is not possible retrospectively to be certain that the patients were well at the time they stopped their lithium, although great care was taken to exclude any patients where there was any doubt. Independent confirmation of lack of symptoms was obtained for five out of the eight patients that relapsed during this first period (three from medical staff, two from spouses).

The slopes of the graphs after the first three months are identical, indicating that a period of lithium treatment (in this case, a mean of $2\frac{1}{2}$ years) does not improve the prognosis once it has been stopped. There is therefore no justification for short periods of prophylaxis.

Patients were not randomly allocated to treatment

or no-treatment, but differed only on the severity of their initial illness, as rated by the MMRS. This is not surprising, since it is reasonable to assume that patients were put on lithium after their first admission because the clinicians believed (for whatever reason) that they were at an increased risk of future relapse. An alternative explanation for the results then seems possible, but if this group had an increased risk of relapse as a result of having had a more severe initial illness, a straight-line decay graph of a steeper slope than the control group would be expected. This is not the case, however, and no other studies have shown severity of symptoms to be predictive of future prognosis.

The results of this study are in contrast to that of Sashidharan & McGuire (1983), which is the only other one of comparable size. However, the latter shows important differences, with a mix of unipolar and bipolar patients, and it is not clear whether the bipolar patients are I or II; in the present study all patients were bipolar I, and it may be this group that is at high risk of a rebound state developing. This difference might go some way towards explaining the contradictory findings in the literature. Such a possibility is further supported by the fact that seven of the eight relapses in this study in the first three months were for mania (the other being for a mixed affective state), whereas in the earlier study there were an equal number of manic and depressive relapses.

Comparison with the study of Bastrup *et al* (1970) is also interesting: although he states that "there was no indication of rebound or abstinence

effects", the investigation was completed in five months. During that time, 12 out of 22 manic depressive patients who had been switched to placebo relapsed; these also were equally divided between mania and depression, which might be a reflection of the ratio of bipolar to unipolar and bipolar I to bipolar II patients in the study. Although it is stated that "the relapses were spread over this period in a manner subjected to analysis elsewhere", these data are apparently unpublished, and no further conclusions can be drawn about them.

The possibility of a lithium withdrawal state has obvious implications for clinical practice. Early relapses might, in the absence of the phenomenon being recognised, lead to the patient being persuaded to restart lithium, and then to take it for a much longer period than necessary, perhaps for life. However, had the relapse been treated without lithium, the future prognosis might have been good and further prophylaxis unnecessary.

This is a retrospective study, and its data suggest that a prospective double-blind discontinuation study is needed of bipolar I patients, concentrating particularly on the temporal relationship between stopping the drug and the recurrence of symptoms. If the increased risk of relapse during the initial withdrawal period is confirmed, identification of types of individuals prone to this type of reaction would be helpful.

Acknowledgements

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Sodium Bicarbonate Abuse: A Case Report

Sodium bicarbonate abuse is unusual, and rarely reported. A patient was extensively investigated at several hospitals for a recurrent hypokalaemic metabolic alkalosis; it transpired that she had been abusing sodium bicarbonate for 8 years and had gained hospital admission at will by taking large amounts. She also showed features of the Munchausen syndrome.

Case report

In 1980, a 21 year-old hospital secretary was admitted to hospital for investigation of a five year history of intermittent vomiting, weight loss, and episodes of light-headedness, nausea, headache, and tingling around the mouth. She denied overbreathing. Clinical examination was normal, apart from a positive Chvostek's sign; blood pressure was 90/60 mm Hg. Investigations showed a plasma bicarbonate greater than 40 mmol/l (normal range 20-30 mmol/l) and a low plasma potassium at 1.8 mmol/l (normal range 3.4-5.3 mmol/l).

Her hypokalaemic metabolic alkalosis was investigated by a barium meal and follow-through, upper alimentary endoscopy and duodenal biopsy, sigmoidoscopy and rectal biopsy, all of which were normal. Liver and thyroid function tests, and cortisol and Synacthen tests were also normal. The electrocardiogram showed a sinus rhythm with T wave flattening, compatible with hypokalaemia.

No cause for her symptoms was found, and she was prescribed eight effervescent potassium tablets daily (53 mmol K⁺/day) and discharged. She moved to the Midlands, and over the next two years was admitted on five occasions with recurrent hypokalaemic alkalosis, despite out-patient treatment with potassium supplements. Her plasma potassium levels on admission ranged from 1.8-2.2 mmol/l, with plasma bicarbonate levels greater than 40 mmol/l. Blood gas analysis showed a raised pH and raised pCO₂. She complained of diarrhoea, nausea, generalised weakness, palpitations, muscle cramps, and occasional slurred speech. Plasma renin and aldosterone, intravenous pyelography, and urine acidification tests were all normal; one urinary acidification specimen was found to contain orange squash. Urine tests showed she was excreting 120 mmol of sodium and 120 mmol of potassium daily. Her creatinine clearance was 90 ml/min. In view of the recurrent hypokalaemia, Bartter's syndrome and pseudo-Bartter's syndrome were considered (Robb *et al*, 1984). The evidence for Bartter's syndrome was inconclusive, however, and she persistently denied diuretic or laxative abuse. In 1982 she moved to another area, and was admitted to hospital complaining that she had become rapidly tanned on minimal exposure to the sun; computerised tomography scanning of the adrenal glands showed no abnormality.

In 1982 she moved to Devon, and was admitted for further investigation of her recurrent metabolic disturbance. Sucrose was found in her 24-hour urine collection. A psychiatric opinion was requested, and it was during this interview that she admitted to sodium bicarbonate abuse over the previous eight years. She had started taking it to

relieve nausea, but found that large amounts gave her a 'buzz' - a feeling of well-being and alleviation of tension. Her consumption increased to daily amounts ranging from 50-150 g, and attempts at withdrawal produced craving and misery. Furthermore, she realised from her first episode of in-patient investigation that she could gain urgent admission to hospital by deliberately increasing her bicarbonate intake; she was able to avoid stressful events by this means and also to gain medical attention.

Two attempts at in-patient withdrawal were followed by a return to dependency. Over the past 12 months she has had regular out-patient supportive psychotherapy. She has now developed slow hesitant dysphonic speech, with periods of mutism, and displays intermittent pseudo-athetoid movements of her arms: these signs are only present at interview.

Discussion

The pathogenesis of a metabolic alkalosis was well described by Seldin & Rector (1972). The common causes are: vomiting, diarrhoea, laxative abuse, relief of chronic hypercapnia, Cushing's syndrome, primary aldosteronism, Bartter's syndrome, and adrenal steroid therapy.

Recent reports have described a metabolic alkalosis secondary to the topical application of 9-alpha fludroprednisolone (Pachon Diaz *et al*, 1983), following the abuse of a nasal spray (Pepe *et al*, 1983), and with fludrocortisone therapy (Burns *et al*, 1983). A case of sherbet fountain (sodium bicarbonate) addiction presenting as hypokalaemic alkalosis was described by Kaye & Williams (1983). Chronic metabolic alkalosis is frequently accompanied by potassium depletion, due to distal tubular potassium secretion uninhibited by competition for secretion with hydrogen ions, and is associated with a high intracellular potassium concentration. Administration of sodium bicarbonate in doses up to 140 g/d for periods of up to three weeks has been shown to produce a metabolic alkalosis with no injurious renal sequelae (Van Goidsenhoven *et al*, 1954).

Our patient proved a challenging diagnostic problem. For three years, she willingly submitted herself to numerous unpleasant and potentially hazardous investigations, without the real cause of her recurrent metabolic disturbance being detected. She was able

to precipitate urgent hospital admission at will, and spent many weeks at different hospitals undergoing expensive investigations and enjoying medical attention. She exhibited many features of the Munchausen syndrome, and had some medical knowledge through her work as a hospital secretary, with easy access to hospital libraries and medical journals

(O'Shea *et al*, 1984). She varied her symptoms and mode of presentation subtly to ensure that further investigations would be carried out.

Despite regular supportive psychotherapy, behaviour therapy, and treatment with antidepressants and benzodiazepines, she continues to abuse sodium bicarbonate.

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Koro in an Israeli Male

The Koro syndrome, a 'special cultural psychiatric syndrome', is encountered in South China, Malaysia and Indonesia, especially among people of Chinese origin. There are only eight case reports of Koro from the Western hemisphere. We present a typical primary Koro patient from Israel. Jewish cultural mores are a possible contributory factor.

Koro is a syndrome of acute anxiety accompanied by a conviction that there is penile shrinkage and dissolution (Yap, 1965), and by a fear of death. It is considered a special culture-bound psychogenic syndrome, as it is strongly influenced by beliefs of the Yang (male) and Yin (female) humours activity in human beings - principles elucidated first by Chu Chen Hang some 700 years ago (Lee *et al*, 1962). This syndrome has been considered variously as an obsessive-compulsive illness, anxiety neurosis, acute

castration fear, psychotic-like syndrome, depersonalisation syndrome, and hysteria (Yap, 1965; Gwee, 1963, 1968; Kobler, 1948; Marks & Lader, 1973; Rin, 1965), based on patients from the area to which the syndrome is endemic: South China, Malaysia and Indonesia.

In addition to the report of Baasher (1963), who stated that Koro is encountered in Sudanese people, there are only eight Western case reports (Dow & Silver, 1973; Lapierre, 1972; Bychowsky, 1952; Yap,

1965; Edwards, 1970; Barret, 1978; Hes & Nassi, 1977); except for one, these are secondary to organic brain syndromes or major psychiatric disorders such as depressive illness and schizophrenia. Two of the eight case reports mentioned are from Israel (Hes & Nassi, 1977), including the one primary case. Unfortunately, due to language difficulties there is no detailed intrapsychic description of this patient, except for his symptomatology.

Yap (1965) differentiated the syndrome as mainly a depersonalisation state rather than a pure hallucination, in the sense that it is not influenced by affective arousal; however, the patient's insight into the symptom is restricted, unlike that of most depersonalised patients. The insight defect was regarded as a consequence of cultural background and the social norms of beliefs. We present an Israeli Jewish Koro patient whose disorder is primary and who has the same insight restriction, as a result of neurotic intrapsychic structure.

Case Report

A 56 year-old man, married and the father of three children, was admitted to the Geha Psychiatric Hospital because of acute anxiety attacks, consisting of a feeling of penile dissolution and shrinkage, which had started three years before.

Born to a Jewish family in Iraq, the patient was the second son of eight children, his father was a merchant, and the family's economic situation was good. He studied for ten years, and up to his immigration to Israel at the age of 22 he worked as an assistant accountant. He served his full term as a soldier for two years in the Israel Defence Forces, and in later years continued to fulfill his civilian obligations through reserve duty. For the past 25 years, he had worked as a printer. Up to his marriage at the age of 32 he proclaimed himself to be a 'ladies' man', and enjoyed going out with friends.

He married his second cousin, under pressure from his parents. The couple could not afford to buy a flat of their own, and were provided with a place within his parents' house. The patient described himself as happily married, a devoted and a loving family caregiver, "hypersexual", and with a very satisfying marital sexual life.

Some years before his admission the patient's wife started to pressure him to obtain a flat of their own, complaining mainly of lack of privacy. Later on, she developed a delusion of being loved by her previous boss at work, and avoided sexual engagement with her husband in order "not to betray her lover". The wife was finally admitted to hospital for two months, and a diagnosis of schizophrenia was made, after she returned home she showed less sexual drive, and the patient started practising masturbation.

In order to earn more money, to enable him to move from his parents' house, he started to work extra night shifts. The patient claimed that his troubles started after being frightened by a black cat in the dark during one of these

nights. A few days later, pain and a feeling of penile dissolution appeared. In an attempt to avoid shrinkage of the penis, he used to pull it by hand. He did not stop working, but used to sit in a twisted position, with one of his hands holding his penis most of the time.

At times he felt intense pain in his groin, and felt compelled to show his wife or workmates his "contracting penis". He seldom had sexual relations with his wife, and complained about increasing pain following intercourse. He eagerly sought the help of urologists and was intensively checked, but no physical pathology was found – which did not offer the desired relief. He believed that he had a fatal disease. A few months prior to his admission, the patient was dismissed from his job. His new job included lifting up objects, but he claimed that he could not bend, because that sort of movement caused further retraction of his already "shrunk" penis. Gradually social disability took over, as he refrained from going to work to avoid the appearance of the symptoms in public.

Three years after his initial complaint, he was referred to the psychiatric ward. During his mental status examination the patient was seated with his whole body turned to one side, in order "not to sustain pressure on his penis". Most of the time his hands were holding his groin. He gave very detailed information about his illness, and showed marked anxiety and fear concerning his "disappearing penis". He also complained of sleep difficulties and of general weakness. Orientation and memory were intact. There was no evidence of auditory or visual perceptual disturbances, and there were no other depressive or psychotic symptoms.

An underlying depressive state was suspected, and a course of clomipramine (up to 200 mg/day) was given for 40 days, but without clinical improvement. Treatment with phenelzine (up to 45 mg/day) was stopped after three weeks, as the patient developed dizziness, marked anxiety, and psychomotor agitation. A therapeutic trial with chlorprothixene also failed to affect the chief complaints. Minor tranquilisers (oxazepam) moderately reduced the frequency of the anxiety attacks.

The patient explained his anxiety as a consequence of a feeling of penile disappearance and pubic "emptiness". The dissolution of the penis symbolised for him his loss of virility. He had a rigid self-image of masculinity versus femininity – the image of a strong man caring for his weak wife, proving his virility through frequent sexual activity. His self-description of masculine omnipotence was completely contrary to results of psychodiagnostic tests (Rorschach, Thematic Apperception Test, Wechsler Adult Intelligence Test, and Bender); he was found to have very low masculine self-esteem, perceiving women as big and threatening.

The patient was also found to have strong sexual drives, accompanied by aggressive impulses toward the sexual object. These drives evoked severe anxiety controlled by obsessive-compulsive mechanisms.

In psychotherapeutic sessions the patient described a poor family situation, in which his wife started to reject his needs for sexual performance. He felt very hurt, increased his masturbatory habits, and believed he consequently became ill, he saw masturbation as the main reason for his illness, as it "weakens the penis and the body, and causes

loss of sperm". He repressed and denied any connection between the strained relations with his wife and the illness. In addition, he received secondary gain by escaping into illness when there were hard times in his work as a printer, and the danger of losing his job became a reality. He continued to show his penis frequently during attacks of anxiety to his room-mate or the therapist, as he had done before with his wife and workmates.

Discussion

Koro is considered a culture-bound psychogenic syndrome (Lkeinman & Lin, 1981; Robert, 1982). Our patient suffered from a syndrome identical to the Koro syndrome described in the literature. He was Jewish, born in Baghdad, and had no knowledge of Far Eastern cultural beliefs. However, in Jewish tradition there is a prohibition against masturbatory acts, which it is claimed can cause physical problems, although there is no special reference to shrinkage of the penis.

Jewish laws and traditions put great emphasis on the continuation of the family line; within that context lay the prohibition of spilling the seed (including masturbation). Underscoring that attitude is the practice of levirate marriage (Deuteronomy 22, 3-6). According to that custom, if a husband died without heirs his closest male relative was compelled to take the widow as a bride and provide her with children. The Bible tells the story of Onan who, after his brother's death, refused to fulfill his duty and decided "when he went into his brother's wife, that he would spill on the ground lest he should give seed for his brother" (Genesis 38, 8-10). Though scholars still debate whether Onan was practicing masturbation or coitus interruptus, his 'sin' cost him his life, and his name is the source of the Hebrew word for masturbation, *onanut*.

The great Jewish philosopher and physician Moses

Maimonides (1135-1204) is reported by Hess & Nassi (1977) to have said that "to spill the semen is to spill the power of the body and its life and the light of the eyes. The more one spills the more the body's power and life diminish". Our patient's complaints were much in the same vein. He felt the dissolution of the penis with anxiety and had a very restricted insight into the symptom.

Psychotherapy and psychological tests revealed a neurotic constellation over conflicts of sexual identity, castration fears, and loss of control over severe sexual aggressive impulses. The 'dissolution' of the penis appeared to serve a neurotic purpose by letting the patient obsessively deal with his penis without using it in reality, in addition to the obvious secondary gains.

The patient was immature, much like patients from Hong Kong described by Yap (1965); Yap saw it as a depersonalisation syndrome, created by dissociative mechanisms affecting the integrity of the body image. Our patient's poor insight into the symptom was affected by intrapsychic defence mechanisms of denial, repression, and dissociation, and not by cultural beliefs as in classical Koro patients.

The precipitating psychological factors—guilt over masturbation and sexual rejection by his wife—are also often described in the literature of classical Koro patients, but here it could be rarely attributed to the cultural prohibition of onanism, so deeply embedded in the Jewish tradition. The appearance of Koro in an otherwise 'normal' people in the Far East is attributed to cultural beliefs, which may be the cause of the relatively high incidence there. However, appearance of a primary Koro syndrome in other parts of the world is possible: the fact that there have been few case reports up to now may be due to either a genuinely low incidence, or to Western codes of classification.

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Anorexia Nervosa in a Woman Totally Blind Since the Age of Two

A 28 year-old woman, blind since the age of two, had become anorexic at 21. She had eating problems as a child, and weight preoccupation from her teens onwards. Psychiatric admission with other anorexic patients preceded the illness. Inability to see oneself does not protect against anorexia nervosa.

Body image disturbance is one of the hallmarks of anorexia nervosa (AN). Of all the psychiatric syndromes, AN is most closely linked to disturbance in the visual body image. When one thinks of being too fat or too thin, the instantaneous image conjured up is a visual one. Therefore, development of AN in a patient who was blind from an early age not only presents an unusual coincidence of medical problems, but also raises interesting theoretical issues.

Case report

A 27 year-old blind woman was referred for evaluation of possible AN; although in the past she had bradycardia, with a pulse as low as 40, there were no current major medical problems. At the time of evaluation she was 5'2" in height and weighed 44.5 kg—78% of the recommended average weight (RAW) for her height according to the 1983 Metropolitan Life Insurance Company tables. The preceding year she had intentionally lost weight from 44.6 kg (82% RAW) down to 43.9 kg (69% RAW), and at that point became and subsequently remained amenorrhoeic. At 44.5 kg she felt far too fat, and was so terrified and depressed by her weight gain that she frequently felt suicidal. She did two or three hours per day of aerobic exercises, depending on her weight;

she also bought a computerised talking scale that called out her weight, and weighed herself several times a day. (Not only does this scale talk, but it also has a memory, permitting it to say such things as "You've gained a pound since the last time you weighed yourself".) Although she tried vomiting a few times, she found it painful and repulsive. However, she used two types of diuretic tablets, many over-the-counter diet pills, and one or more enemas on a daily basis for at least two years. Her daily caloric intake was estimated to be between 200-600 calories—primarily in the form of vegetables. There were no episodes of bulimia.

She was born, the youngest of three daughters, into a troubled marriage; both parents were intermittently alcoholic, and her mother had been very thin in the past. At the age of 18 months she developed a retinoblastoma in one eye, which was treated by enucleation, and received radiation therapy to the other eye; by the age of two, all residual eyesight in the remaining eye was also lost. She recalls being tied down for the radiation treatment, and developed a fear of being restrained.

From a very early age, her parents always wanted her to eat more; at times she would claim not to like certain foods, even if she did, because she didn't like being forced to eat. She recalls having developed strong aversions to many foods, simply on the basis of not liking the sounds of their names, even if she enjoyed the tastes. For example, the words pizza, crust, and spinach were ugly to her, so she

would never eat those foods. Also, gelatin deserts 'wiggled', so she would not eat them. She rarely tried new foods after the age of six. She remembers people commenting about her weight being too low.

When she was six her parents, always fighting, divorced for the first time they remarried when she was ten, constantly battled, and divorced a second time when she was 20. As a child and through her teens she worshipped her father, but subsequently came to realise that he had always been hyper-critical of both her and her mother. She also learned that her father had been unfaithful to her mother, and developed the idea that "men cheat and try to control you".

Early adolescence was difficult, and for the first time in her life she felt tremendously unhappy. She attended normal schools that had only a few special programmes for the blind. When she was 13, children in school were unpleasant to her, teased her often, and went so far as to steal her cane. At 13 she began to menstruate and develop physically, and became horrified by the idea that she might start to get fat. She said "bikinis were in then, but I would never put on a bathing suit because my body was changing and I was starting to feel fat like a whale". Her first periods frightened her very much, and subsequently she has welcomed the amenorrhea resulting from her weight loss as a boon. All through high school she had poor self-esteem, a great deal of self-hatred, and felt alone and ostracised much of the time.

At the age of 18 she left her family, and went away to college in another state. She had a guide-dog during her first year. She did well, and hoped to have a musical career. At the end of that year many of her friends left, and she transferred to a good music course at a small university near the family's home. She remained there for 18 months, after which for obscure reasons she was abruptly discharged; her family was told that because of her blindness, the teachers didn't know quite how to handle her, and the university refused to provide any further explanation. This expulsion shattered her already fragile self-esteem and she suffered her "nervous breakdown": she became severely depressed, paranoid, withdrawn, and suicidal, and began to slash herself. Having lost her sense of direction and meaning in life, she was diagnosed as having a mixed personality disorder and a secondary depression.

She was admitted to a private psychiatric hospital, where she remained for the next 18 months. She cut her wrists deeply enough to require sutures many times, and developed the idea that the wounds needed to stay open or else she would be a bad person. At this hospital she first encountered women with AN, and initially thought they were "very weird". During this period the idea that she needed to lose weight first took hold; the most she had ever weighed was 50 kg (88% of RAW), and her usual weight had been 47.7 kg. While in hospital she dieted down to 43.9 kg, and felt that she was 'bad' when her weight increased. After 18 months, still at a low weight, she was discharged to a long-term care facility, but within a few days threatened suicide with a loaded gun and was readmitted. Over the next four years she had five additional admissions for impulsive suicidal behaviour.

She began to experience herself as fat through tactile

exploration of her arms and her legs, but her perceptions of her limbs were inconstant: "sometimes they felt thin, and sometimes they didn't, changing sometimes from minute to minute or hour to hour". She was aware that some of the changes were actually related to fluid shifts rather than fat. But, she said, "anything that is weight I don't like. When I feel fat I get depressed and suicidal." During this period she was chronically depressed, and was given the DSM-III diagnosis of atypical depression (American Psychiatric Association, 1980). She made additional multiple suicide gestures and was treated, to no avail, with a variety of antidepressants, including imipramine, amitriptyline, tranylcypromine, phenelzine, and lithium to which triiodothyronine was added. She also had a course of 12 ECTs, which had little effect.

During periods out of the hospital she lived sometimes with her sisters and sometimes with her father; he was very critical of her, and they even had physical fights. She visited her mother (who moved away from the area) only intermittently.

She received out-patient psychotherapy, where DSM-III dysthymic disorder was diagnosed, in addition to her other diagnoses. Ultimately, with this help, she managed to move away from her father to share an apartment with a platonic male friend she had known for several years. In that setting her weight rose, to her horror, to the 44.5 kg she weighed at the time of evaluation.

Family history

Her paternal grandfather committed suicide, and a maternal uncle died of alcoholic cirrhosis. Her oldest sister has episodes of binge eating, is always overweight, and is always on a diet. Her middle sister, a runaway who had once been addicted to drugs, had settled down, and was currently married and doing well.

Mental state

On examination, she was a thin, pleasant woman who spoke easily and openly about her history and mental life. She talked about her past suicidal behaviour and current death fantasies, alternating between blandness and a wry, wistful smile. At times she giggled with mild anxiety, but was otherwise composed and appropriate. She described having had trouble throughout her life in making decisions and in dealing with new situations, particularly social situations, which she found anxiety-provoking. There was no evidence of hallucinations, and the only evident fixed preoccupations were those concerning weight and food.

Her calm appearance during this evaluation was quite different from her mental state as previously recorded in her medical records, in many of which she had been described as depressed, distraught, histrionic, and desperate.

On the Eating Disturbance Inventory (Garner *et al.*, 1983) scores on all eight sub-scales were at or above those reported for patients with AN. Here, too, she demonstrated exaggerated responses to the various sub-scales. Since this measure is not yet widely known, we present her most salient scores, followed by the AN and female comparison

(FC) group averages published by Garner *et al* (or, where pertinent, the restrictor or bulimic sub-groups referred to as ANR or ANB): drive for thinness 21 (AN=15.4; FC=5.0); bulimia 1 (ANR=2.7; ANB=10.8; FC=2.0); body dissatisfaction 21 (ANR=14.2, ANB=17.4, FC=10.2).

Discussion

To the recent proliferation of unusual cases of AN (Buchan & Gregory, 1984), we add what is to our knowledge the first published case of its development in a woman blind since the age of two. In our view the diagnosis is valid, based as it is on the presence of all the major psychological, behavioural, and physical manifestations of AN. That these were preceded and accompanied by other psychiatric difficulties, most notably personality disturbances, does not diminish the accuracy of this diagnosis. The patient's exaggerated responses on the Eating Disturbance Inventory were consistent with her observed behaviour in the home and with the meticulous records she kept regarding her intake. Her fussiness with food, use of food in control struggles with her parents during childhood, and weight preoccupation during early adolescence all provided a background of vulnerability. Her subsequent AN coalesced during a time when she was without a sense of direction or purpose – when her sense of self, always shaky at best, was lowest since she was no longer a musician or university student; AN then provided her with an identity, at a time when she desperately needed one. Although she remained volatile and self-destructive during the 18 months following the development of the AN, it was her view and that of her therapists that the severity of her self-destructive slashing diminished as her AN deepened; it seemed as if, to some extent, she traded the former for the latter. The fact that she modelled herself after patients with AN that she met on the ward is consistent with her shaky sense of self.

The perfectionistic strivings she had as a student and musician, the presence of binge eating episodes in one sister, and the harsh, critical nature of her father are all possible contributing factors. This patient also exhibits many masochistic features, common in patients with AN (Hogan, 1983). From a

psychodynamic point of view, she clearly incorporated the harsh, critical, and unforgiving aspects of her father, and brutalised herself with self-destructive slashing, starving, and unrelenting intrapunitive criticism.

The fact that she was sighted during her early infancy might have contributed to a more accurate self-and-other representation than that usually expected in congenitally blind children, but hers is still seriously impaired. The trauma of the enucleation and of the restraint required for radiotherapy might have deeply influenced her personality development. Conceivably, the radiotherapy might have affected the development of nearby areas of her brain, and thus contributed to her subsequent psychiatric difficulties.

The development of body image in blind children appears to be impaired, relative to sighted children. Given the importance of visual communication between mother and infant in the development of a good relationship between the two and in the development of the infant's sense of self (Robson, 1967), it is remarkable that the personality development of blind children is ordinarily so normal (Freiberg, 1977). This may be due to the great amount of redundancy in the mother-child communications system and in the intrinsic autonomous development of personality. Kinsbourne & Lempert (1980) found that congenitally blind children do internalise a representation of the human body, but compared with that of sighted children it is impoverished and systematically distorted – a defective representation. The other senses permit a spatial representation of the body, but one that is inaccurate. Freiberg (1977) found the development of the sense of self or 'I-ness' to be delayed. In this regard, it would be of interest to know if cases of AN have ever occurred among congenitally blind children, and to assess the extent to which preoccupations with weight and appearance occur in such populations.

It is clear from this case that the absence of sight from an early age and of all the attendant visual information regarding the shape of one's own body relative to that of others does not preclude the development of AN. You do not have to see yourself to want to be thinner.

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Identical Twins Discordant for Presenile Dementia of the Alzheimer Type

In genetically proven identical female twins, discordant for presenile dementia of the Alzheimer type, the affected twin began to dement at the age of 49, and died 15 years later; the diagnosis was confirmed at post-mortem. The surviving twin remains clinically unaffected 20 years after the onset of dementia in her sister. Environmental aetiological factors are postulated to account for this discordance.

Alzheimer's disease (AD), the major cause of dementia, is arbitrarily divided into presenile dementia of the Alzheimer type (PDAT) and senile dementia of the Alzheimer type (SDAT), depending on whether the illness starts before or after the age of 65. Although previous evidence (Newton, 1948; Lauter & Meyer, 1968; Constantinidis, 1978) implied that these forms of AD are essentially identical disorders, differing only in age at onset, recent neuropathological (Bondareff *et al*, 1982) and neurochemical (Rossor *et al*, 1984) findings suggest a nosological distinction between the two.

AD may result a variety of different aetiological factors. Relatives of probands have an increased risk of dementia in later life (Sjogren *et al*, 1952; Larsson *et al*, 1963; Constantinidis, 1978; Heston *et al*, 1981), but the mode of transmission is uncertain. The results of most pedigree studies are compatible with polygenic inheritance, but some families AD appears to be inherited as an autosomal dominant disorder (Breitner & Folstein, 1984).

The study of biological markers such as hapto-globin types (Stam & Op den Velde, 1978; Eikelenboom

et al, 1984), HL-A antigens (Renvoize, 1984), HL-A linked complement components (Neri *et al*, 1984), and ABO and rhesus blood groups (Renvoize, 1985) in AD patients may help to unravel the nature of this inheritance. However, important information on the relative aetiological contributions of heredity and environment in AD might be gained by the more traditional approach of studying concordance rates for the disease in monozygotic (MZ) and dizygotic (DZ) twins, relying on the fact that MZ twins are genetically identical, whereas DZ twins share only half their genes. If an increased concordance rate is observed in MZ compared with DZ twins, a genetic contribution to the aetiology is confirmed.

Kallmann (1956) studied 108 twin pairs in which the index twin had senile dementia, and found an incidence of senile dementia of 42.8% in MZ co-twins, 8% in DZ co-twins, 6.5% in siblings and 3.4% in parents. Though this confirms the role of inheritance in senile dementia, the lack of full concordance in the MZ twins also points to the importance of non-genetic factors. No similar systematic twin studies have been conducted in the presenile form of

AD, and only four cases of PDAT occurring in identical twins have been described (Davidson & Robertson, 1955; Hunter *et al*, 1972; Sharman *et al*, 1979; Kilpatrick *et al*, 1983). This paucity of twin studies therefore prompts us to report a case of genetically proven MZ female twins, discordant for PDAT 20 years after the onset of disease in the affected twin.

Case report

Personal histories

The twins were born following an uncomplicated delivery in 1915, each weighing approximately 6 lbs; the unaffected twin (U) was born first and the affected twin (A) second. From birth they were identical in appearance, and later blood group studies showed a high probability (0.98) of them being monozygous (Table I). The personal development and educational progress of the twins was similar and unremarkable. Both left school at 14 to work as weavers in the same mill. Twin U married at 21 and moved away, while twin A married at 23 and lived locally, but they remained close, visited regularly, and went on holiday together. A was healthy prior to the onset of dementia and, unlike U, never travelled abroad. U had pneumonia at 16, appendicectomy at 20, mastectomy at 29 for a breast abscess, and suffered a left central retinal vein occlusion on the day of A's death at the age of 64. Both experienced the menopause at 44.

Twin U is widowed and lives alone. When interviewed, 20 years after the onset of PDAT in A, she showed no evidence of dementia, and scored maximum points on Hodkinson's Mental Test (1973).

Family history

Their father died of bronchopneumonia at the age of 65, and their mother, who developed insulin-dependent diabetes mellitus in later life, died from heart disease at 75. Neither the parents nor the grandparents appear to have become demented. Sisters of 72 and 60, and a brother of 62, are healthy and show no evidence of dementia.

Each twin had one daughter. The 41 year-old daughter of twin A is well, but the 43 year-old daughter of twin U was diagnosed as suffering from probable Hodgkin's disease at the age of 20. This is of interest in view of reports of an excess of myelo-proliferative disorders among the relatives of sufferers of AD (Heston *et al*, 1981). She presented with swelling in the left inguinal region, and a gland was removed which showed atypical histological features; she was treated with local radiation and made a full recovery, which has been sustained.

History of illness in the affected twin A

In 1964, at the age of 49, twin A began to have difficulty in setting the table, and forgot the names of old friends. Over the next four years there was a gradual decline in memory, intellect, and personality. During a hospital admission in 1968 she was restless, and disorientated in time, place

TABLE I
Results of zygosity testing of the twins

Blood group system	Twin A	Twin U
ABO	0	0
Rh(D)	+	+
Phenotype	ccDEe	ccDEe
Direct anti-globulin	—	—
MS	MSs	MSs
P ₁	—	—
Lu ^a	—	—
K	—	—
Le ^a	—	—
Le ^b	+	+
Fy ^a	—	—
Jk ^a	+	+
Jk ^b	+	+

and person, concentration was poor, general information limited, and short and long-term memory impaired. She stammered and showed severe sensory, nominal and expressive dysphasia, with constructional apraxia and dyscalculia.

Physically she was a plump, normotensive, grey-haired woman, with bilateral arcus senilis, and slightly increased tendon reflexes in the limbs of the left side. Full blood count, erythrocyte sedimentation rate, blood urea, electrolytes, liver function tests, glucose, folic acid and cyanocobalamin levels, thyroid function tests, and Wassermann reaction and Kahn tests were normal. Cerebrospinal fluid examination, including the Lange colloidal gold curve, was normal, as were skull and chest x-rays, air encephalogram, left carotid angiogram, electrocardiogram, and radioactive isotope brain scan. A series of EEGs showed abnormal activity of theta and delta frequency, symmetrically distributed, with subclinical seizure activity in one record.

After discharge she became progressively more dependent upon her husband, requiring help with feeding, washing, and dressing; she was finally admitted to a psychiatric hospital, four years later. She gradually became mute, unresponsive, wasted, doubly incontinent, bed-ridden, and prone to generalised seizures; at this stage, there was generalised hypertonia, hyper-reflexia, and occasional myoclonus. She died at the age of 64, about 15 years after the onset of dementia.

Post-mortem findings

The body was emaciated; all the visceral organs were underweight, and there was a severe bronchopneumonia.

The brain weighed 850 g, and externally showed marked generalised cortical atrophy, maximal at the frontal poles. Coronal sectioning revealed the cerebral ventricles to be dilated and the cerebral cortex thinned, especially in the occipital region, the subcortical structures were relatively normal.

Microscopically, senile plaques were found in the cerebral cortex in sections from the temporal, frontal, parietal, and occipital regions. In frozen sections of the temporal lobe these plaques were numerous, and many of the cortical neurons showed neurofibrillary tangles. The appearances were those of AD.

Discussion

Our case is the third example in the literature of MZ twin pairs discordant for PDAT. Davidson & Robertson (1955) reported a woman with acne rosacea who began to dement at the age of 50, and died 19 years later. The post-mortem confirmed AD; her unaffected twin sister showed no evidence of dementia at the age of 73. These twins were considered to be identical, although no laboratory investigations were made to prove uni-ovularity. The affected twin had suffered from severe influenza with delirium and fever at the age of 38, during the 1918-1919 epidemic; as in our case, the affected twin was the second-born, and there was no family history of dementia. Hunter *et al* (1972) also described a pair of genetically proven MZ female twins discordant for PDAT. The affected twin died aged 64, after a 15-year history of dementia; AD was confirmed at post-mortem. The unaffected twin sister died two years later of carcinoma. As the twins had been adopted in infancy, no details of birth order or family history were available. Our twin A and the affected twins described above have some features in common: all were women with similar ages at onset and with illnesses of unusually long duration.

There are two reports of MZ twins concordant for PDAT. Sharman *et al* (1979) described PDAT in identical twin brothers who simultaneously developed the disease at the age of 34 and who both died four years later. The disease was confirmed histologically in both, but no special genetic studies were carried out to prove monozygosity. Their mother had also developed a similar dementing illness at the age of 37 and died five years later; at post-mortem her brain was found to be atrophied, but no histological examination was performed. More recently,

Kilpatrick *et al* (1983) reported genetically proven identical twin sisters with PDAT, both of whom started to dement at the age of 50 and were still alive six years later. Histological confirmation of the disease was obtained by cortical biopsy in both sisters. Their mother and maternal grandmother had developed a similar illness at the same age. The almost simultaneous onset of dementia and the positive family history in these two twin pairs favours autosomal dominant inheritance.

Although concordance cannot be completely excluded in our twins, it is unlikely after 20 years; as both twins were genetically identical, this discordance points to the importance of environmental influences in the onset of AD in the affected twin. Potential aetiological factors already examined in the disease include disordered immune function (Kellett *et al*, 1982; Pentland *et al*, 1982), conventional virus infections (Whalley *et al*, 1980; Renvoize & Hambling, 1984), chromosomal abnormalities (Wright & Whalley, 1984), and metal intoxications (Crappier *et al*, 1976; McDermott *et al*, 1979), but no clear associations have as yet emerged. In a case-control study of AD, Heyman *et al* (1984) found no differences between patients and controls in toxic environmental exposures, animal contacts, smoking, drinking, or unusual dietary habits; however, AD patients had an increased history of severe head injury, and female patients a higher frequency of prior thyroid disease.

Although the role of inheritance in the aetiology of AD is clearly established, this and previous reports of twins discordant for the disease demonstrate that non-genetic factors must also play a part, at least in some cases. The search to identify these environmental agents should continue.

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Art Group Psychotherapy in a Psychiatric Day Unit

The use of art in the context of a therapy group in a day hospital setting is described, and the literature on art and factors influencing such groups discussed.

Art has been used to illuminate psychopathology (Rorschach, 1913; Guttmann & Maclay, 1937; Maclay *et al.*, 1938; Lehmann & Risquez, 1953; Wadeson & Bunney, 1970; Birtchnell, 1973), as an indicator of maturation (Richards & Ross, 1967), as the main agent in the rehabilitation of psychiatric patients (Dreikurs, 1976), and as an adjuvant in a therapy programme (Edelstein & Kneller, 1976). It has also been used as an aid to counter institutionalisation (Gerace & Rosenberg, 1979) and to sensitise staff to the feelings of their patients (Misner, 1979).

Method

The project was set up to meet the needs of a group of day patients at the Department of Psychological Medicine, Newcastle General Hospital. The group comprised the Senior Registrar in Psychiatry and Art Therapist as therapists, with six patients whom it was felt could benefit from psychodynamic group therapy. It was planned as a 'slow open' group, since members were free to leave after an initial commitment of three months. However, with only one member leaving after six months to find employment outside the city, it became a 'closed' group.

The group met once weekly for one and a half hours, over one year. The first hour was a conventional talking group, followed by about 15 minutes' painting; a further 15 minutes was spent discussing the individual paintings of the patients and therapists.

The group was older (range 40–58 years) and more chronically disturbed (2–8 years of psychiatric treatment) than is usual for therapy groups, but quite characteristic of patients in most day hospitals. The group members each demonstrated a combination of symptomatic disturbance, dependent patient role, interpersonal problems, and problems of self-concept. They were of average or above average intelligence, with varying verbal ability and even more variable psychological mindedness. Assessment for the group was on the basis of each patient's personal characteristics and on their ability to use other group activities on the Unit.

The therapists had detailed discussions about the verbal content and paintings after each therapy session. This self-supervision was enhanced by writing up each session and presenting this to the supervisor each month. An assessment of outcome was made six months after the end of the group, in order to avoid the immediate effects of the ending of a group and yet to be recent enough for the group to be remembered clearly (Bednar & Lawlis, 1971). Moreover, sustained gains are more important than gains apparent immediately after treatment. To avoid therapist bias, the patient and referring psychiatrists rated changes (in mood, interpersonal functioning, and 'overall') since referral to the day unit. Patients rated components of their experience in the group.

Case History and Results

The experience of the group can best be illustrated by describing one of the patients. Analysis of the effects of

therapy with a group of six is necessarily anecdotal rather than statistical.

Mrs F, aged 51, had her first psychiatric illness two years previously, when she was admitted with loss of confidence, apprehension, shakiness, anxiety, and depression; the diagnosis then was depressive illness. She had previously been treated by her GP for five years. On admission, she received tricyclic antidepressants and ECT. She responded to these, but was left with marked loss of confidence and self-esteem, having lost her job on account of prolonged absence from work. She had a broken marriage, but was continuing to stay in the same house with her ex-husband and sons.

The symbols in her paintings expressed her attitude towards the group by portraying them as trees under black skies, representing non-coping people. To convey her feelings of isolation and uncertainties about becoming part of a group she positioned herself as a raft, looking towards the mainland where she felt she should be, but with a hostile sea of emotions to cross before she could reach land. As the group progressed and strong feelings were being expressed, she showed apprehension by painting a crouched figure waiting to be protected, or half-constructed walls, not knowing whether they should be built up or broken down. She also saw herself as a leper hiding in a hooded cloak – a diseased person not acceptable to 'normal' society – and the group as scared people who wore masks in order to appear 'normal'. Her anger was expressed against this society in a drawing of herself as a rat in a cage, being rewarded with a piece of cheese for performing well. As she worked through her feelings, she drew defences which were strong enough to cope with landslides of the future. As the group approached its end, she was afraid she would not cope without its support, and showed anger at the impending loss. She compared herself to a toad which hibernates in a hole during adverse conditions, where it stays, feeds, and swells so much that it cannot leave the hole when it wants to. However, she worked through the pain of the loss, the effort involved being symbolised by climbing a long steep ladder to leave. Mrs F painted quickly and spontaneously, covering most of the paper. In discussions she could express anger and distress, which also emerged in the painting, and in times of distress she could use the painting as a means of cathartic release, communicating to the group her conflicts and her responses. She showed an ability to respond to other group members, and to integrate her interactions so as to gain understanding of her feelings.

Six months after the group, Mrs F felt much better generally in her mood and in her relationships. She had found all aspects of the group helpful, and was only disappointed that it had not gone on longer. The psychiatrist had discharged her from his care, feeling that she had improved greatly overall and in her mood, but only slightly in her relationships; he noted that the group had helped her self-esteem and enabled her to express her anger and other emotions instead of storing them up.

Another group member showed gains comparable to Mrs F, and a further member gave every indication of similar improvement before his death from a myocardial infarct three months after the end of the group. The patient who left after six months had made significant improvements; the other two members rated themselves as unchanged or

worse, though their referring psychiatrists thought that their functioning was marginally better. Their lack of progress after the group had been reflected in a lack of progression in the themes of their paintings during therapy. It is of interest that this is in keeping with Kiesler's view (1971) that indicators of progress can be indicators of eventual outcome.

Discussion

A veture of this kind raises such questions as the balance between caring and curing for patients who are older, less flexible, and have less overall strengths to contribute to the life of the group, yet who bring a longer lifespan of experience and stability to therapy (Hildebrand, 1982). Questions that are related to this issue are: How 'real' and accessible should the therapist be? Is therapy to be skewed in the direction of 'non-specific' therapeutic factors (Yalom, 1975; Bloch, 1979), such as catharsis, mutual support, universality of problems and altruism between group members (all very evident in this group), or of relating present, 'here and now' feelings to transference issues in the past and current external present (Malan, 1979)?

Transference issues appear less important in older patients (Hildebrand, 1982). This is reinforced both by a closing down in the range of options available and by painful feelings in relation to a longer adult life, in which mistakes have been made whose transference implications have been overlaid with more practical consequences. This difference of perspective is easily lost on therapists more youthful than their patients unless, as in this group, they have had considerable experience with older patients.

As part of the assessment procedure of the day unit, all patients were encouraged to be aware of the interpersonal aspects of their problems, and to regard their situation in wider terms than 'illness' and the relief of symptoms. The very nature of conducting the group in a day unit programme both enriched, supported, and complicated the therapy. It enriched it by common day-by-day experience shared by patients and therapists. It supported it, as shown by the very high attendance and negligible dropout rate, and complicated it by presenting opportunities for acting out group phenomena on the unit. Meeting between sessions in a fairly structured setting provided an opportunity to work through material informally. While the general intervention of the therapists was directed to commenting on here-and-now events in the group, relating these

to current and past events, their role as interpreters was affected by their roles outside the group.

The supervisor addressed himself to the written reported content of the sessions and to the paintings as they were produced; in this way the therapists were encouraged to work over the issues between sessions, committing their thoughts to paper. Since the supervisor saw his role as that of supervisor and not therapist to the therapists, his comments on the therapists' paintings were handled in much the same way as the reported verbal material.

For this group the art component played diverse roles. The paintings gave insight into the personality styles of the group members and the degree to which these changed. As well as these 'diagnostic' perspectives, the art reinforced a sense of identity among group members, which contrasted with their former role as recipient patients, and thus it discouraged institutionalisation and encouraged group cohesion. It was an adjuvant to the therapy itself, serving to sensitise staff to the feelings of their patients, both in an overall sense and also towards the changing feelings within the group. While the therapists' paintings reflected their roles and feelings in relation to the group processes, the patient's paintings could be interpreted both personally and as carrying some aspect of the group dynamic.

For some group members, painting enabled them to communicate with the group in a way that would have been denied them had the group been entirely verbal. This is important for people who, for intellectual, educational, or cultural reasons, do not readily express themselves verbally. For others, art represented a sublimated discharge of the feelings mobilised in the course of therapy. It may be questioned whether such a group safeguarded some members from acting out their feelings in a less controlled manner in their lives. To people who have suffered considerable emotional and material deprivation, apparently uninvolved therapists can be seen as being further depriving, and are likely to be ignored or reacted to with much hostility (Meares & Hobson, 1977). The therapists avoided this problem by combining an interpretive with a more giving role, both by sharing the act of painting and by contact with group members in the day unit at other times.

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A Comparison of the Antidepressant Action of Citalopram and Amitriptyline

The response of patients with major depressive illness to citalopram of amitriptyline was compared in a double-blind multi-centre trial. No differences in efficacy were observed, but citalopram had less hypnotic effect, and a remarkably lower profile of side-effects.

Citalopram is unrelated to tricyclic, tetracyclic, or other known antidepressants, and is a specific blocker of neuronal uptake of 5-hydroxytryptamine at the presynaptic membrane, with no significant action on catecholaminergic systems. Its pharmacological properties suggested that it should be non-toxic to the heart, and without anticholinergic side-effects.

Method

The trial was conducted in five hospitals in South Wales, on in-patients and out-patients aged between 18 and 70 who met the DSM-III criteria for major depressive illness (American Psychiatric Association, 1980), scored 18 or more on the 17-item Hamilton Rating Scale for Depression (HRSD) (Hamilton, 1967), and gave informed consent. All were entered into the trial within 36 hours of admission (48 hours at weekends).

The trial was double-blind and randomised in blocks of four. Matching tablets contained either 10 mg of citalopram or 37.5 mg of amitriptyline. For the first three days the dose was two tablets at night, increasing to four until the end of the third week, and thereafter 3–6 tablets (30–60 mg citalopram, 112.5–225 mg amitriptyline). Average doses given in the sixth week were 46 mg for citalopram and 148 mg for amitriptyline. The only other drugs allowed were diazepam and temazepam, for night sedation and for control of anxiety.

The trial lasted six weeks, but some patients were withdrawn earlier for troublesome side-effects persisting after reducing the dose (one patient on citalopram, five on amitriptyline), for deterioration or failure to improve (four and five, respectively) and for other reasons (two and five, respectively).

The following assessments were made: the Newcastle Scale (Carney *et al.*, 1965), the HRSD; the Montgomery-Åsberg Depression Rating Scale (MADRAS) (Montgomery & Åsberg, 1979); and the Leeds Self-Rating Depression Scale (Snaith *et al.*, 1976). The results were analysed using the Mann-Whitney U-test, the Wilcoxon matched pairs signed ranks test, and the chi-squared test.

The assessments of efficacy were run as end-point analyses, transferring the latest available rating to later assessments. Of the 24 allocated to citalopram, one was assessed only up to three weeks, one to the fourth week of treatment, and the remainder completed the six weeks' course. Two on amitriptyline were assessed for the last time at three weeks, three at four weeks, and the remainder of the 20 remained on the trial for six weeks.

The mean data for the HDRS are illustrated in Fig. 1. The other assessments gave similar results, and have not been included. There were no significant differences between the groups in the average starting scores on any of these ratings, or between any of the subsequent scores. All ratings showed a significant improvement at the 5% level from seven days onwards (Wilcoxon matched pairs).

Side effects

Most patients experienced at least one additional side-effect over baseline during treatment. In general, reported side-effects were much more common in those on amitriptyline (110) than in those taking citalopram (52) (Fig. 2), and the profiles of complaints appeared to be due to the anticholinergic properties of amitriptyline (dry mouth, blurred vision, sweating, urinary retention, and constipation). Of patients on citalopram, 16 had no such symptoms and 11 had one or more; of those on amitriptyline, only five were free of anticholinergic problems and 20 recorded one or more (χ^2 , $P < 0.01$). With citalopram, five side-effects of severe degree were reported, compared with twelve with amitriptyline.

Discussion

No difference in therapeutic effects of citalopram and

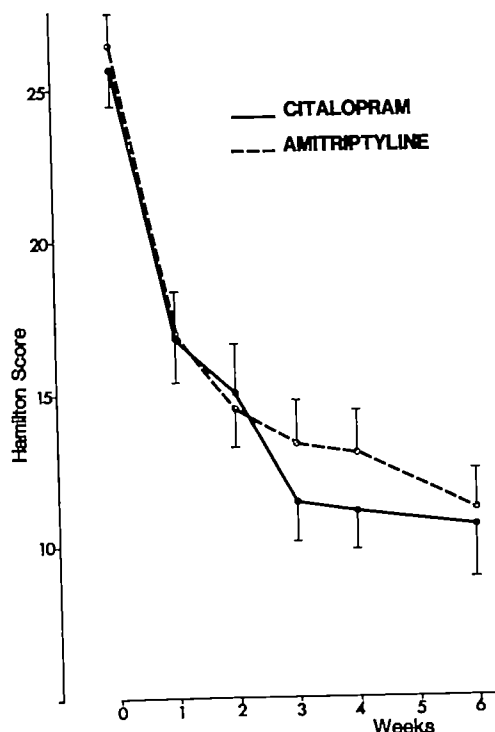


FIG. 1 Mean HDRS scores for citalopram ($n = 24$) and amitriptyline ($n = 20$). Error bars show the standard errors

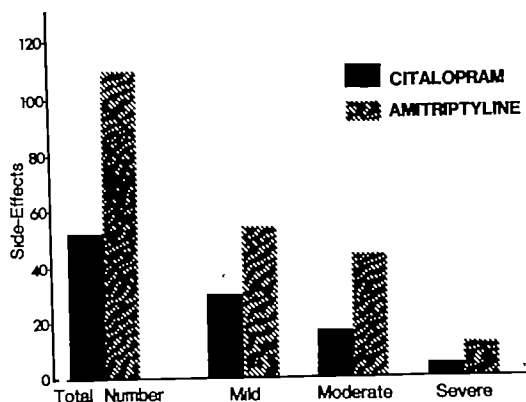


FIG. 2 Side-effects not present at baseline

amitriptyline appeared, but citalopram was clearly the less troublesome drug with regard to side-effects, in that side-effects above baseline were seen twice as

often in the amitriptyline group. Amitriptyline's problems in this, as anticipated, were anticholinergic. Thus, the results of this trial suggest that citalopram is an effective and acceptable antidepressant.

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Books Reconsidered

*Psychotherapy by Reciprocal Inhibition: Joseph Wolpe**

I first came across this particular book during my training at Boston City Hospital in 1970. It was by that time 12 years old. Skinnerian operant conditioning was the rage in the psychological and psychiatric circles; reciprocal inhibition seemed at the time to be considered *passé*. The psychoanalysts had already discounted any suggestion that the only effect of insight-oriented therapy was to provide a therapeutic setting in which reciprocal inhibition took place. Behaviour therapists were busy working out complex positive and negative reinforcement schedules for illnesses as diverse as schizophrenia and alcoholism. Dr Laing put in an appearance in Boston at that particular time, extolling the virtues of the existential benefits of madness. But, I imagine that in its day, this book was highly controversial, since it challenges the central premise of psychoanalysis—that the essence of psychotherapy is uncovering and expression of the repressed.

Wolpe presented a new theory of psychotherapy based on learning theory: neurotic behaviour is presented as originating in learning, and the theory predicts that its elimination is a matter of unlearning. Thus, the gauntlet was thrown down and psychoanalysis challenged. Wolpe introduces his ideas with experimental observations which were made on cats in which lasting neurotic states had been induced. Should anyone doubt that cats can be made neurotic, they need only discuss their current doubts with our sister profession, The Pet Psychiatrists. An antagonism between feeding responses and anxiety was found to occur.

It is this finding which was transposed into the realms of psychotherapy with humans. The therapeutic principle of reciprocal inhibition is as follows: "If a response antagonistic to anxiety can be made to occur in the presence of anxiety-evoking stimuli, so that it is accompanied by a complete or partial suppression of the anxiety responses, the bond between these stimuli and the anxiety responses will be weakened."

Wolpe's basic method was simple. An anxiety hierarchy was constructed with patients, and the patient was then made to relax (reciprocally inhibited), while

imagining items in the anxiety hierarchy. A 90% cure was claimed with neuroses from simple phobias through to character neuroses, which were believed to consist of intricate systems of phobias. The reciprocal inhibition which was used ranged from assertive responses, sexual responses, relaxation responses, respiratory responses, 'anxiety-relief' responses, conditioned motor responses, 'pleasant' responses in the life situation [with drug enhancement], and interview-induced emotional responses. These were the responses that the disposal of the therapist by which therapeutic change may deliberately be brought about.

Justification for this method is coherent and logical. A background explanation of the relationships between anatomical structures in behaviour, discussion about the making and unmaking of functional neural connections, in other words—learning and unlearning, are followed by a definition of neurotic behaviour—"any persistent habit of unadaptive behaviour acquired by learning in a physiologically normal organism."

My main criticism of *Reciprocal Inhibition* is that it oversimplifies the method by which human beings acquire neurotic illnesses. This oversimplification leads to a blinkered epistemology of the totality of human experience. Psychotherapy by reciprocal inhibition attempts to break the links between the person and extra-personal stimuli. The mistake of psychoanalysis initially was to ignore this very important area. The mistake in psychotherapy by reciprocal inhibition is to ignore the fact that human beings can create within themselves a world whose inner reality and experience bears little resemblance, if any, to the outer reality of the world within which that person lives. Hence, people have strong beliefs in religion, strong cravings for and addictions to various forms of art and culture; they also can internally (intrapsychically) create and maintain belief which leads to neurotic behaviour. Looking at a systemic level, the theory of reciprocal inhibition by-passes the power inherent in a system. Systems may condone certain types of symptoms, but condemn others. For example, one family may consider it is perfectly permissible for the entire family to bathe together in the nude, while another family exists in which such behaviour would be cause for

* (1958) Stanford University Press, California

instant referral to the nearest psychiatrist. In one therapeutic group it is permissible to stand up, shout, move about, roll on the ground, and regress to birth while in another therapeutic group, any such movement could lead to instant dismissal from the group.

But where is the strength of this book? I believe it lies in the systematic way that innovative, interesting, and successfully used therapeutic techniques and methods of treatment are presented. Why should it be re-read? In much of psychiatric practice, there remains extreme ignorance about creative ways of working with people. Psychotherapy using reciprocal inhibition actually includes the therapeutic relationship itself. I regret that much of the ignorance is within the psychiatric profession, while much of the psychological work is being done by psychologists,

nurses, social workers, and occupational therapists. Many of them will already have absorbed the principles of psychotherapy by reciprocal inhibition, and because they are unable to prescribe and use drugs, they have forged ahead in their abilities to use their relationship with patients in an effective way.

When the Royal College of Psychiatrists feels that psychotherapy training of any sort should not be made mandatory for junior psychiatrists, it will not surprise me that recommendations that this book be read by every junior psychiatrist would also fall on deaf ears. Nevertheless, I would make this recommendation. It is a book well worth reading and was written well before the polemics about behavioural therapy versus "analytical psychotherapy" destroyed any concordance that could have occurred within the psychiatric profession.

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Dominance Hierarchies in Psychotherapy Groups

Sir: The article by Kennedy and McKenzie (*Journal*, June 1986, 148, 625-631) is fundamentally flawed. The underlying assumption of the authors is that social behaviour has biological determinants, and that the social behaviour of the highest organism phylogenetically will include the social behaviour of the lower primates, such as chimpanzees. In particular, the authors say there is a "natural" tendency to form hierarchies and furthermore, these hierarchies have a regulatory function, resolving the tension between the development of cohesion and conflict. The central error is to ignore the importance of culture and variation in human social organisation. It is not true that humans are "naturally" hierarchical. There is much research by social anthropologists describing in detail how many societies of people who live by hunting and gathering are, remarkably, non-hierarchical (e.g., Lee & DeVore, 1968). Amongst some hunters and gatherers there is a leader whose position is attributable as much to personality factors as his skill in hunting; however there are no ways to impose his authority, and his position is very tenuous. When conflicts arise the hunting band is likely to divide.

Many anthropologists (e.g., Sahlins, 1977) consider direct extrapolation from ethnological studies to human behaviour to be an abuse of the method. One reason why this occurs is that aspects of social organisation, the culture, of the writers are seen in the animal society organisation.

This is made relevant in considering the subject of hierarchies in psychotherapy groups: these groups share the culture of the society of which they are a part. When the authors see hierarchy in psychotherapy groups they will consider it to be "natural". They are, in fact, seeing the way the group reproduces the outside social structure.

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ECT as a Contributor to the Production of Delusional Misidentification

Sir: I read with interest the conclusion of Dr Hay's paper (*Journal*, June 1986, 148, 667-669).

I have seen a 58 year old woman who presented with a three week history of delusional misrepresentation which began after she had an anaesthetic. She had become constipated with the onset of a depressive illness two months previously, and believed that she had cancer. She sought a surgical opinion. A sigmoidoscopy was performed under anaesthetic which excluded bowel pathology, but confirmed constipation. She had suffered no previous psychiatric illness. Both the patient and her husband agreed that the delusional misidentification began after the sigmoidoscopy. This suggests that an anaesthetic agent rather than electrical stimulation may be the cause of such symptoms.

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Lithium Augmentation of Tricyclics

Sir: Pai *et al* (*Journal*, June 1986, 148, 736-738) describe a further series of patients where it is claimed that lithium augments the effects of another antidepressant. Nearly all the other studies making this claim, including those cited in the report, seem to be describing the same phenomenon. Namely, that some severely depressed patients who have completely failed to respond to a first-line antidepressant dramatically respond within anything from a few days to a couple of weeks to the addition of lithium. The simplest and most economical explanation of nearly all of the cases reported is that lithium has had a major acute antidepressant effect. Although most of the authors consider this possibility, it is usually dismissed and some complicated explanation for either summation or potentiation is invoked to explain the observation.

All of the controlled studies of the acute antidepressant effects of lithium, subsequent to the negative result of Stokes *et al* (1971) have shown lithium to have a major acute antidepressant effect, substantially better than placebo and at least, if not better, than an antidepressant such as imipramine.

Two controlled trials conducted by myself and colleagues in Dundee (Worrall *et al.*, 1979) are representative and discussed the issues at that time. One of those trials, in which tryptophan was compared with lithium and tryptophan in combination, is regularly quoted in the subsequent combined studies and used as evidence for lithium having an augmenting effect on other antidepressants. We specifically stated that the design of that trial could not possibly prove that. The simplest explanation in that trial was that lithium was acting alone and, in truth, tryptophan was only used because we did not obtain ethical permission for a placebo comparison.

What seems to have happened is that influential figures twenty years ago stated that lithium did not have important acute antidepressant effects and, with minor modifications, that statement has been authoritatively repeated in nearly all reviews since. What the combined studies have shown is that when a first-line treatment has proved ineffective and the clinician has then tried lithium, he has not believed his own eyes when he has seen a major acute antidepressant response, and some form of potentiation or augmentation of the first drug by lithium is instead suggested.

Why expose patients simultaneously to two drugs without first trying each separately, especially if, as is likely, continuation treatment is going to be needed? It may be that in a few patients lithium does need to be used along with another antidepressant, or a neuroleptic, and it would be surprising if on occasion two antidepressants from a different class did not have more effect than one. A controlled trial to prove that would be feasible, but before going to that trouble an open-minded reappraisal of the effects of lithium alone might make such a trial less necessary.

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At Risk Drinking

Sir: I was delighted to read the article by Dr King (*Journal*, May 1986, **148**, 533-540). In this and other articles Dr King has used the CAGE Questionnaire.

Unfortunately, he has repeated a failure of other authors to attribute the questionnaire to the correct source. The CAGE questions were developed by me and my colleagues at the University of North Carolina in the late 1960s. The CAGE questions have been used in many different studies by now, and recently (Ewing, 1984) I published a paper in the *Journal of the American Medical Association* describing their origins, clinical use and efficacy. That reference might be the best one for Dr King and others to use in future.

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Anhedonic Depression

Sir: Young *et al.* (*Journal*, March 1986, **148**, 257-267) have suggested the possible existence of two sub-types of endogenous depression—anhedonic sub-type and vegetative sub-type. Earlier, Hibbert *et al.* (1984) reported that symptoms of reduced interest and pleasure and of low mood represent features of the state of depression which are related to each other, but are not much related to the more 'biological' symptoms of reduced sleep and appetite.

In a study of 34 RDC major depressives, I classified the patients as anhedonic and non-anhedonic based on high or low scores on the sub-scale anhedonia-asociality of the Scale for the Assessment of Negative symptoms (SANS) (Andreasen, 1981). The anhedonic sub-type had a significantly ($P < 0.001$) longer duration of illness than the non-anhedonic sub-type. Almost 20% of anhedonic depressives had a duration of illness longer than nine months with treatment. The response to tricyclic antidepressants was unsatisfactory in the anhedonic depressives and the need for adjuvant therapy with electroconvulsive shocks and antipsychotics was significantly ($P < 0.01$) more often required for them. Anhedonic depressives also required a much longer duration of treatment than the non-anhedonic depressives (Chaturvedi & Sarmukaddam, 1986). Interestingly, anhedonic depressives had significantly higher total scores on SANS than non-anhedonic depressives.

These findings help further in identifying an anhedonic sub-type of depression. However, in my study, no demographic differences were observed

between the two groups, especially as regards, age, sex, education, occupation or marital status. There is a need for research on biological parameters to further validate the sub-types.

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Quetelet's Index and Appetite and Weight Change in the Context of Depression

Sir: A study of 168 mild to moderately depressed out-patients presenting over a period of six years at St Thomas's Hospital, London, and involved in a series of antidepressant trials, showed that factors linked to appetite and weight change were previous appetite and weight history. Patients gaining appetite and weight with depression had a previous history of overeating in response to the normal stresses and strains of everyday life, and of variable weight history (Harris *et al*, 1984a). With recovery after six weeks' treatment, these patients tended to lose appetite and weight (Harris *et al*, 1984b). A possible explanation for this is to be found in the concept of restraint. According to Polivy & Herman (1976) restraint is characterised by preoccupation with the caloric values of food, dieting, and guilt feeling on diet-breaking. It is high in obese and latently obese subjects, and in normal or even under-weight individuals who respond to societal pressure to be slimmer and run below their "set-points" of body weight. They hypothesised that with anxiety and depression such individuals lose restraint, and there is increase in food intake and weight. Obviously, in large populations of depressives, there will be overlap with factors linked to obesity, and the further question arises as to whether or not it is the fatter patients (i.e., fat relative to height, not fat relative to pre-episode weight), who gain appetite and weight with depression and then lose appetite and weight with recovery. To examine this, we looked at subjects in terms of Quetelet's Index (QI) Weight/Height^2 (Quetelet, 1869). QI has recently been validated as a convenient formula and reliable indicator of obesity (Garrow & Webster, 1985): 20-24.9 representing

desirable weight, 25-30 mild overweight, and over 30 being clinically relevant.

The method of the study has been reported elsewhere (Harris *et al*, 1984a). At presentation, height and weight were measured, and after six weeks' treatment, patients were weighed again. QI was calculated in terms of the patient's stated normal weight. Appetite was scored at presentation and at six weeks, relative to patient's own norm. Standard statistical methods were used, including the unpaired t-test and the product-moment correlation coefficient.

Appetite and QI. At presentation, appetite was more often reduced than increased, and the relative appetite was positively correlated with QI ($r=0.15$ $0.05 < P < 0.1$). After six weeks' treatment this was reversed and there was a negative association between appetite and QI ($r=0.18$ $P < 0.05$). The patients were also divided according to appetite into groups with marked increase, moderate change and marked decrease. These groups were delineated by boundaries approximately one standard deviation either side of the mean. If delineation was by initial appetite, the differences between groups were not significant. When groups defined by appetite at six weeks were examined, subjects whose appetite was severely decreased were significantly fatter (QI mean 28.2) in comparison with those who had both marked increase and moderate change ($P < 0.01$). When projected back to what they were at presentation, the same group had had relatively high appetite.

Weight and QI. Weight at presentation showed no significant associations with QI. Patients were also divided into three groups: those with marked increase in weight (109% of their normal weight), those with moderate change (95 to 109% of normal), and those with marked decrease in weight (95% of normal weight). These groups were delineated by one standard deviation beyond the mean (102%). There were no significant differences at presentation but at six weeks the group currently less than 95% of customary body weight had a higher mean customary QI of 25.8 ($P < 0.05$).

A number of practical implications result from our findings concerning QI. First, there is a tendency for fatter individuals to increase appetite with depression. Therefore, identification and management of depressive mood changes is an important factor in the management of obesity. Secondly, the question arises as to whether or not further weight gain will occur with antidepressant treatment, e.g., due to tricyclic-induced carbohydrate craving. Our findings indicate that in the short term this is not the case, but that loss of appetite occurs, probably due to regain of control. It might be possible, therefore, to use a first line antidepressant drug such as a tricyclic

for a short period without producing further appetite and weight gain.

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Smoking in Chronic Schizophrenia

Sir: While studying obesity in our patients (Gopalaswamy & Morgan, 1985) we asked them about their smoking habits and 100 men and 70 women replied: 123 of them had chronic schizophrenia, most were in their 50's and 60's with many years of illness behind them, 82% had never been married, and the majority were working class.

Smoking was more prevalent among the men ($P < 0.05$) and among the schizophrenic patients ($P < 0.001$): 87% of the men and 74% of the women schizophrenic patients smoked, compared with 60% and 58% respectively among the non-schizophrenic patients. These differences within the patient sample were trivial compared with the significant differences among the patients (83% of all men and 67% of all women smoked) and among the general population, of whom 36% of men and 32% women are known to smoke.

The proportion of smokers in the general population falls year by year in response to anti-smoking propaganda which most patients choose not to heed. This is very understandable. There may be few pleasures left in life for a person with chronic schizophrenia and smoking may be one of them. Indeed, for the schizophrenic it may be even more pleasurable than for a healthy person. Nicotine in large doses depresses a person's level of arousal. Schizophrenics suffer from heightened arousal and for them a packet of cigarettes represents a way of suppressing the discomfort of hyper-arousal which they may find more acceptable and effective than the major tranquillisers which we prescribe for them.

Our findings match quite closely those of Masterson & O'Shea (1984) who reported smoking by 92% of their male and 82% of their female schizophrenics. Rice (1979) questioned how heavily his schizophrenic patients smoked and whether they ever developed lung cancer. The proportion of patients with schizophrenia who die of lung cancer is surprisingly no higher, and may even be slightly lower, than in the general population—when it seems that it ought to be double or treble (O'Shea, 1984 Craig & Lin, 1981). This suggests either that people with schizophrenia enjoy partial protection against developing lung cancer or that cigarette smoking is not such an important aetiological factor as is thought. Either way the subject requires fuller investigation.

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Psychotherapy in the Third Reich

Sir: Henry Rollin (*Journal*, March 1986, 148, 345-346) reminds us that the ghost of Hitler still stalks the world. I am reminded of an anecdote told by the late Willie Mayer-Gross, himself a victim of Nazi oppression, about the high esteem in which Hitler was held by some psychiatrists. A German research fellow working at the Maudsley came to see Mapother after the Reichstag fire and the plebiscite in 1932. He wanted to discontinue his fellowship and return to the Fatherland. "If 92% of the German people have voted for Hitler, he must be a great man". Mapother replied: "I am of Irish stock, if 92% of the Irish would vote for one man I would be certain that he is worthless".

Mayer-Gross' own researches into the fate of neurotics under the Third Reich confirmed the pathologic influence of culture on the manifestations of neuroses. Comparatively few German servicemen were shot for "lack of moral fibre"; neurotic anxiety and depression were somatised, generally taking the

form of disorders of the gastrointestinal tract. So great were their numbers that special "stomach battalions" were required to accommodate them and their disabilities. Hitler thought he had eliminated neurosis by decree: he did not succeed.

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Tardive Dystonia and Pisa Syndrome

Sir: I wish to report a case of tardive dystonia presenting as Pisa syndrome and induced by depot neuroleptic.

Case report A 37 year old male patient with paranoid schizophrenia was on a maintenance dose of fluphenazine decanoate, 25 mg intramuscular every two weeks since May, 1981. Before that he had been treated as an in-patient during 1979 and 1981 with chlorpromazine up to 600 mg per day and five modified bilateral ECT. He had no family history of psychosis or any movement disorder. In June 1984 he was noticed to be keeping his upper trunk bent towards his left side and rotated backwards. This symptom had appeared insidiously and had been progressing slowly for about two months before he was brought to hospital. On examination, his left shoulder was tilted approximately four inches lower and two inches backwards compared to the right one. The patient could maintain normal posture only for a few minutes, with effort. There was no tremor or dyskinesic movement of any part of the body. The patient did not feel distressed or disabled by the symptom and, on questioning, showed marked lack of concern about his grossly abnormal posture.

His fluphenazine injections were discontinued and he was prescribed oral trihexyphenidyl 2 mg three times a day. However, there was no significant relief over four weeks and the drug was stopped. The patient was kept on drug-free follow-up. After another two months the symptom started improving slowly and disappeared completely by March, 1985. Since then there has been no recurrence and only residual schizophrenic symptoms are present for which he is not receiving medication.

The insidious onset of dystonia in this patient suggests the diagnosis of tardive dystonia. The symptom in this case matches almost exactly with the description of Pisa Syndrome (Ekblom *et al*, 1972; Yassa, 1985). Significant points to be noted here are absence of any acute dystonia or tardive dyskinesia at any stage, complete lack of concern shown by the patient to the symptom, unresponsiveness to anticholinergic therapy, and delayed but complete spontaneous recovery on discontinuation of fluphenazine.

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Yassa, R. (1985) The Pisa syndrome: a report of two cases *British Journal of Psychiatry*, **146**, 93-95

Procyclidine Abuse

Sir: We wish to report five cases of procyclidine abuse.

Case reports. (i) A 39 year old man with a personality disorder (JB) insisted he needed chlorpromazine and procyclidine. When all his medication was stopped, his wife, a 26 year old schizophrenic adequately maintained on a depot neuroleptic, began requesting increasing amounts of procyclidine for persistent Parkinsonian side-effects. She later admitted that JB was using all her supply of the drug.

(ii) A 39 year old woman with schizophrenia regularly persuaded medical staff to prescribe procyclidine by telling tall stories such as: "the wind has blown away my prescription" and "the dog has eaten my tablets".

(iii) A 22 year old man with a personality disorder and a long history of polydrug abuse frequently presented to the casualty department at weekends feigning rigidity and oculogyric crises in order to obtain procyclidine.

(iv) A 58 year old man with schizophrenia refused procyclidine during his last hospital admission preferring instead to tolerate severe Parkinsonian side-effects. He said he was afraid of becoming addicted to procyclidine again.

(v) A 39 year old woman with schizophrenia refused to consider any reduction in her procyclidine dose even though she had no obvious side-effects from phenothiazine medication.

These cases illustrate abuse of procyclidine and the lengths to which patients may go in order to obtain it. The euphoric side-effects documented in the *British National Formulary* were cited by the patients as the main reason for their misuse of the drug. Dependence and abuse of procyclidine are not reported in the latest edition of the BNF.

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Transcultural Psychiatry

Sir: Further to my anxieties about the use of psychiatry to confirm Western folklore concerning the *zombie* (*Journal*, March 1986, **148**, 340-341): it now appears that the Harvard research project on tetrodotoxin in Haiti was funded by the Broadway producer, David Merrick, and that the whole study is currently being turned into a feature film starring the

psychopharmacologist, his Haitian informants and, presumably, a *zombie* (Gleason, 1986). Apparently the original idea was that the drug, when isolated, would be used by NASA to place astronauts in a state of suspended animation during long voyages (Davis, 1986).

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CORRECTION

In the letter from Anthony B. Joseph (*Journal*, June 1986, 148, 749) 5th para, 8th line, the reference should be (Joseph, 1986) not (Weinstein *et al*, 1954).

A HUNDRED YEARS AGO

The Idiots Bill

The framers of this measure should at once determine, not only in their own minds but by an intelligible definition of the term 'idiots' what class of persons they intend to deal with. As the Bill is drawn it is a highly unsatisfactory measure, affording opportunities for dealing with persons of feeble mind with only one certificate instead of two, and with the least possible protection. It will be a scandal if this measure passes with no more attempt to distinguish between two classes extremely easily confounded than the

absurd intimations that 'idiots' or 'imbeciles' do not include 'lunatics' or 'lunatic' does not mean or include 'idiot' or 'imbecile'. The definition is not one whit more intelligible as a means of analysis than the famous synthetical formula, "Caesar and Pompey are alike, especially Pompey."

Reference

- Lancet* (April 3, 1886) pp. 656

Researched by Henry Rollin. Emeritus Consultant Psychiatrist., Horton Hospital, Surrey.

Book Reviews

Foucault. By J. G. MERQUIOR. London: Fontana Press. 1985. Pp 188. £3.50.

Foucault was one of the celebrated twentieth century hermeneutic social thinkers who sought massive forces at work behind the unassuming social practices of the *bourgeoisie*. Like Marx, he was interested principally in power relationships (there is a chapter in this book on Foucault's 'cratology': his theory of power) but he ended up in Freudian territory in the power relationships which are codified in sexual practice.

Foucault is of interest to the psychiatrist as an ideologue and as a historian of insanity. Merquior is principally concerned to present the former side of him in this book, but does devote a chapter to *Madness and Civilization: A History of Insanity in the Age of Reason*, the gadfly book in which Foucault takes the great steps forward in psychiatry made by Pinel, Tuke and the rest, and argues for them being steps in "that gigantic moral imprisonment which we are in the habit of calling, doubtless by antiphrasis, the liberation of the insane by Pinel and Tuke" (*Madness and Civilization*, p. 278 in the Vintage Books edition). Merquior's discussion of this book closely follows the lines of Sedgwick's in *Psycho Politics*, but adds more evidence against Foucault's historical accuracy. In this chapter and later in the book, Merquior concludes that Foucault was not a reliable historian and that the true history of European psychiatry does not sustain his thesis.

Even so, the effect of *Madness and Civilization* lingers on, and not only in the camps of the irrationalists, the antipsychiatrists and the counter-culture to whom it made, according to Merquior, its first appeal. The reason for this may lie in the seductiveness of Foucault's central claims. Two of them are that modern man is an alienated and distorted descendant of men when unreason flourished; and that enlightened social reform, humane medical practice, sexual hygiene and scientific rationalism are some of the main means by which this distortion has come about since they represent the imposition on society of the apparatus to control unreason developed by the *bourgeoisie*.

Merquior assesses these and similar claims with impartiality although he makes clear that he is not a Foucaultian at the end of the book. It is a densely written monograph which draws on an impressive amount of research for its small compass. As is so

often the case, Foucault's style has affected the style of writing about Foucault and parts of the book seem purely rhetorical ("Like Nietzsche, Foucault holds the individual's will to power as a datum which is also an unavoidable fatum"). The learning is also sometimes too obtrusive, and the references to Foucault's books are sometimes to their French and sometimes to their very freely translated English titles, which makes for confusion. These minor criticisms aside, I enjoyed this book and I am glad to have read it. I would recommend it to any psychiatrist with an interest in the history of ideas or in recent currents of French intellectualism. It is also, insofar as I can judge, an excellent, if unsympathetic, introduction to the work of Foucault although it omits any indication of the relationship of that work to his life.

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A DSM-III Casebook of Differential Therapeutics.

By SAMUEL PERRY, ALLEN FRANCES and JOHN CLARKIN. New York: Brunner/Mazel. 1985. Pp 370.

Case vignettes of 53 adult patients chosen to represent the spectrum of DSM-III diagnoses form the material upon which three authors from Cornell University display their skills in choice of psychiatric therapies. Their own cases are seasoned at times by those of distinguished colleagues from elsewhere in the USA and some cases of famous psychiatrists (Freud, Kraepelin and Alzheimer), whose management is now discussed in modern day terms. The purpose of this book is to illustrate the rationale of the various psychiatric therapies and, through their examples of putting therapies in to practice, to be educative. The book opens with a chapter that describes the various therapeutic options and closes with some personalised guidelines from the authors.

One of the strengths of this book is that it is well written with a lively style interspersed with humour. The cases read like short "who-dunnits", the outcome being held back while the clinician thinks aloud and chooses the therapeutic approach. The impact of that approach and the outcome for the patient is then presented. The second strength is that this book teaches us something about American/British differences in practice. Their treatments seem quite doctor

and clinic orientated, the relationship between the doctor and his patient being the key.

Paramedical and community workers are given little prominence. The approach by the psychiatrist is eclectic (somatic, behavioural and psychotherapeutic techniques all being used); but the bias is in favour of the last. In the introductory section to various therapies, 23 pages are given over to a discussion of interpersonal techniques while 3 are given to somatic therapies. However, for these authors the doctor/patient relationship is a precision instrument specified under the headings of format, setting, duration and frequency and technique. The authors point out possible dangers from the patient in misapplied relationships, so that throughout the book their therapeutic approach is carefully planned and monitored. Such critical analysis of the doctor/patient relationship, shown to be different in aim, type and duration for each of the patients they describe, is refreshing and very informative. The English reader can learn something here.

The weakness of the book comes perhaps from an implication in the title *A DSM-III Casebook*. While each case is classified along the five current DSM-III axes before treatment is discussed, the authors don't refer to this diagnostic exercise again. It would have been an excellent opportunity to evaluate DSM-III in terms of its ease of application in each of the cases and whether or not the classificatory exercise helped them in choice of treatment or had any predictive validity. This seems a wasted opportunity to show DSM-III in practice to an international readership. Time spent in critique would have perhaps given this book some balance because the overall effect is somewhat glossy. However, one mustn't carp too much, for it's rare to find a psychiatric text book that is enjoyable to read.

ANTHONY H. MANN, *Reader in Psychiatry,*
Royal Free Hospital School of Medicine, London

Essential Papers on Borderline Disorders. Edited by
MICHAEL H. STONE. New York: New York
University Press. 1986. Pp 580. \$55.00.

The time is ripe for an anthology of papers on the so-called borderline states or disorders. This volume provides an anthology which is the choice of a psychiatrist with broad interests in both the biological and psychoanalytic aspects of the problem, and it can therefore be read with interest by psychiatrists of all persuasions or none. It is one of a series of essential papers on psychoanalysis, the others being about object relations, narcissism, and depression respectively. Subtitled *One Hundred Years at the Border* it

takes up the story in 1835 with an extract from James Prichard's *Treatise on Insanity*. This is the third interpretation of Prichard's less than specific work, the others being the notions that he was writing about either psychopathy or manic depression.

Other papers in the 1880's to 1920's section are by Hughes and Rosse. Each section is preceded by a lengthy but helpful introduction written by the editor, setting the papers in historical context and trying to clarify the enormous logical and taxonomic difficulties which beset the field. Indeed these sections are perhaps value to the inexperienced reader than the selections themselves.

The second section deals with the analytic 30's and 40's, with contributions by Stern, Deutsch, Schmideberg, and Hoch and Polatin. Here the disorders in question are variously described as varieties of schizophrenia, psychopathy, and neuroses.

The 1950's and 60's contain papers by Grinker, Kety, Eriksen, Kernberg, and Balint, and show the beginnings of a biological approach and the flowering of the analytic. The 1970's and 80's are concerned with the nosological status as well as the tricky countertransference issues. The final paper, by Akiskal, carries the most telling title for borderline disorders—'Borderline: An Adjective in Search of a Noun'. It is to the credit of this volume that it does not try to shirk the problem of definition even though it may not throw much new light upon it.

The many different notions of what borderline is jostle each other in a nosological dustbin, sometimes perhaps without sufficient acknowledgment of the inconsistencies between them. They range from "moderately severe psychiatric disorders", through "mild, recovered and compensated schizophrenics" to *forme frustes* of either schizophrenia or, perhaps, manic depression (the bias, if any, of the editor). On a more personal note they may be "difficult narcissistic patients with whom one (ie the analyst) fails" or "angry depressed and unreasonable" patients. They may otherwise be classified as "hysteroid dysphoria", and even "mothers of borderline cases" are also said to be likely to be borderline.

Patients who show defence mechanisms of "disavowal and denial" or who have "serious problems in handling separation and in achieving an adequate sense of autonomy and self definition" can be called borderline. It fills "the space between existing and more sharply discriminable entities like schizophrenia and manic depression" and is neighbour to "concepts of psychoneurosis and schizophrenia", as well as having the two meanings carried in DSM-III of 'schizotypal' or 'unstable' personality. Only the concepts and definitions of Kohut are definitely rejected.

With such a plethora of descriptions the reader may be tempted to draw the conclusion that after more than a century at the borderline we still haven't got a map.

CHRISTOPHER THOMPSON, *Senior Lecturer,
Charing Cross and Westminster Medical School,
London*

Psychopharmacology: Recent Advances and Future Prospects. Edited by SUSAN D. IVERSEN. Oxford: Oxford University Press. 1985. Pp 332. £25.00.

For their tenth anniversary the British Association of Psychopharmacology decided on a broad review of their subject. The proceedings show variety as the striking feature, and specialists in modern psychopharmacology fill 27 chapters with history, progress, growing points, and further hopes and needs.

The work is divided into five parts, including affective disorders, anxiolytics, psychopharmacology of cognition, schizophrenia, and euphorians. In all it gives an impressive account of the status of the subject seen largely from the research-worker's point of view. The discussion of historical matters and areas of broader consideration will be readily enjoyed by all, but the more detailed chemistry is strong stuff, to be taken, if not slowly, in divided doses. Surprisingly, in the light of commercial pharmacological progress, affective disorders do not steal the limelight and the five sections are well balanced and thorough in approach.

The first section is concerned with the effectiveness of anti-depressants, and a helpful wide view of the treatment of depressive illness is taken, although the contributing factors in treatment resistance are not stressed. This is followed by consideration of the biology of depressive illness and the role of 5-HT. A detailed review of the role of platelets in the neurochemical field follows and there are clear and helpful descriptions of animal models of depression in which their limitations are fully acknowledged. Antidepressants of the future are considered.

In the second part, anxiolytic compounds are reviewed mainly from the point of view of the benzodiazepines; in particular where they act and their differences. There are chapters on the production of anxiety by drugs, and the prospects for treatment. This section is introduced by a stimulating and searching review of the uses of anxiolytic substances in society.

An additional treat is provided by a full section on cognitive disorders. Not only is the content important, but the introduction of this topic to the 'front window' of a major text may be "a first".

Schizophrenia receives a well-deserved share of research and particular attention is drawn to the contributions of genetics and environment, structural changes in the brain, the basis of antipsychotic drug activity, Dopamine D₂ receptors, and the case for integrated viral genes.

Medical consideration of stimulant drugs is still active despite their sunken public image. Perhaps the suggestion that there is little place for stimulant drugs in treatment may carry the baby with the bath water, for some find a limited place for them in excitatory abreaction techniques and the diagnosis of covert psychosis. The subject of alcohol is well covered and yields a worthy message. Caffeine, nicotine and aphrodisiacs are included here.

The variety of topics is equalled by the range of penetration and perspective shared by the chapters. Each justifies its inclusion. There are parts where the varied use of terms is irritating, but this is inevitable when drugs, chemistry or treatments themselves are the prime focus. In covering so wide a field the sentiments of the editor, Susan Iversen, bring us down to earth when she reminds us that the drugs discussed may improve brain function or restore dysfunction, but may also be instruments of self destruction. She should be congratulated, for this is a collection for the *cognoscent*, a clearly visible milestone of which the authors and British Association of Psychopharmacology should be proud.

JOHN POLLITT, *Consultant Psychiatrist,
Hayes Grove Priory Hospital*

Public Drinking and Public Policy. Edited by E. SINGLE and T. STORM. Toronto: Addiction Research Foundation. 1985. Pp 294. \$25.00 (hb), \$20.00 (pb).

Those of us involved in the delivery of services for people with drink-related problems find it increasingly hard to define the boundaries of our proper professional concern. We used to treat patients in hospital. Now we counsel customers in the Community. Concurrently we are becoming more interested in social contexts and conduct which may be prejudicial to reasonably problem free drinking. As our Community Services bite deeper and deeper into our drinking cultures, we cannot help but notice the regularity with which those for whom we care frequent the same drinking milieu, drink in the same peer groups with anti-domestic beliefs and/or which support and validate markedly high levels of alcoholisation. If, as we are urged, primary prevention and education are increasingly to become our business then so is an understanding and eventually perhaps a

moulding of public drinking behaviour. It is on that quite remote horizon of clinical practice that *Public Drinking and Public Policy* looms.

The book is the edited proceedings of an A.R.F. Conference held in Canada in April 1984 and obviously the participants had an exciting and illuminating time for the post-hoc introduction talks of the value of bringing together different groups and disciplines and the hope that the dialogue will be continuing. Generation of such sentiment is the life force of conferences but it is usually not sustained so that by the time the proceedings eventually see the light of day they are for the participants akin to old photographs and for readers who were not there, somebody else's photographs. So this book contains all the usual proceedings problems of repetition, inconsistency of style and striking differences of focus.

Most of the seventeen papers were given by social researchers and their techniques vary greatly. Some just sat on a bar stool drinking and watching what was going on (or participant observation as it is called) whereas others did huge international questionnaire surveys. What is pleasing is that regardless of the angle from which it was photographed the looming on the horizon was not formless. Young single men are the dominant pub goers; patrons' conduct is modified by the public house design and site; the bar staff have a major influence on drinking behaviour; price of drink and special offers such as "happy hours" do influence consumption and may lead to people making a decision to drink rather than do something else; neighbourhood taverns ("locals") are also about sociability and of course many pub environments are sexist. That's all pretty universal and has preventive potential. But the more unsystematised and quirky papers were the most illuminating. Pekka Sulkunen's chapter on a suburban pub in Finland and its darts club from the perspective firstly of the patrons and then of their wives or ex-wives is a delightful analysis. James Shaefer's highly original and idiosyncratic chapter which even includes a relationship between the tempo of country music and rate of consumption is a gem. (They are inversely correlated, probably because the slower is the music the more maudlin is the lyric.) Thomas Barbor's chapter on 'Alcohol, Economics and the Ecological Fallacy' is very condensed but well worth the effort and the final two chapters raise the interesting hare of the publican's liability if patrons drink and drive after leaving the establishment. This latter issue is exactly the kind—among thousands of others—to which David Robinson and Philip Tether address themselves in their important new book on local strategies for prevention of alcohol problems.

(*Preventing Alcohol Problems A Guide to Local Action*. London: Tavistock. 1985.)

So there is much here of interest but only to a tiny minority of specialists with a particular orientation. For the majority its main value would be to help them to understand what kind of environment they were in if after a hard week of "real psychiatry" they drop down for an hour or two to the Red Lion.

DOUGLAS CAMERON, *Consultant Psychiatrist,
Leicestershire Community Alcohol Service*

The Case-Study Method in Psychology and Related Disciplines. By D. B. BROMLEY. Chichester. John Wiley. 1986. Pp. 351. £24.50

Bromley's main contention is that the accepted methods of natural science, such as laboratory experiments and social surveys, cannot be easily applied to phenomena which have developed and occur in the 'real' world independently of investigators, particularly in individual cases. He believes that jurisprudence, especially the aspect concerned with 'best evidence', provides the basis of 'an explicit conceptual and methodological framework' (p. 312) which will solve the problem; hence his notion of the 'quasi-judicial' case-study - 'quasi' because of important differences between psychological and judicial case-studies. Bromley sees the case-study as utilising: i) all relevant information; ii) concepts and procedures derived from research, iii) the philosophy of science; and iv) the methods of other professional disciplines.

Bromley believes that the quasi-judicial case-study not only facilitates practical work, but can produce important generalisations, analogous to case-law. The case-study should therefore become one of the main investigative procedures of psychological science, being taught at both under- and post-graduate levels, publishing its own journals, and being prominent in formal academic discussions.

He does not demonstrate the exact relation of jurisprudence to psychological case-study method, and provides no empirical examples of the effective application of the method. We have only the discussion, very difficult to understand, of certain methodological and theoretical aspects of some unsystematic studies, which are only summarily described. The general account of the method is clearly written and very useful, but this account does not seem to differ, in essence, from the long-established practice of many clinical psychologists and psychiatrists.

The uniquely original aspect of this book lies in its conception of the scientific importance of the case study in general and clinical psychology, the exposition of which is enlivened by the imaginativeness of much of the writing. Further exploration of this conception could have important consequences for both psychiatry and clinical psychology.

M. B. SHAPIRO, *Honorary Research Fellow in Psychiatry,
University College, London*



Anxiety: Psychological Perspectives on Panic and Agoraphobia. By Richard S. Hallam. London: Academic Press 1985. Pp. 210 £30.

Panic: Facing Fears, Phobias and Anxiety. By Stewart Agras. New York: W. H. Freeman 1985. Pp. 151. £22.95 (hb), £13.95 (pb).

The first of these books is a long theoretical exploration of anxiety or rather certain aspects of that state of experience which the author chooses to call panic anxiety. Despite the title the first part of the work considers somatic aspects and medico-psychiatric views and constructs. It is at times difficult to be certain what the author means by the term 'panic-anxiety' but a chapter devoted to three illustrative accounts by subjects (sufferers) of their experience indicates that the area of discourse is concerned with sudden onset of anxiety which the subject recognises as an abnormal experience. Despite this the author strives to discount the abnormal, or morbid, nature of such an experience. The many strands of thought that entwine the topic are followed and this results in a useful survey of present views although the author admits his prejudice and some are more fully developed than others. The personal contribution of the authors is hard to discern. It is a book for a theoretician and not for a clinician.

Dr Agras' book is, by contrast, addressed to the intelligent person who suffers, or who has a relative suffering, from phobias. It might be read with benefit but it is constricted by the somewhat naive theoretical concepts of classical behaviour therapy and fails to develop the more enlightening cognitive reinterpretation of these views and clinical concepts. Despite the title there is no clear description of the state of panic disorder and no development of the observations which have led to an understanding of the development of agoraphobia from early panic attacks. Consequently the reader is uncertain as to the basic difference between the agoraphobic syndrome and other simple phobic states. The attempt to conceive of compulsive symptoms as a simple extension of phobic states has been discounted and the author confuses 'implosion therapy' with exposure to anxiety inducing situations at maximum intensity which is sometimes called flooding. Certainly implosion therapy did not originate from the Institute of Psychiatry, London. Despite these and other minor objections the book would do no harm if read by a phobic patient.

PHILIP SNAITH, *Senior Lecturer in Psychiatry,*
The University of Leeds

Preclinical Psychopharmacology. Vol. 2, Part 1 Edited by D. G. GRAHAM-SMITH and P. J. COWEN. Amsterdam Elsevier. 1985. Pp. 471. \$111.00, Dfl. 300.00.

"The route that pharmacology offers for a biological understanding of severe mental illness remains a difficult one", wrote one of the authors. Despite the interest and activity centered on psychopharmacology and biological psychiatry in recent years, and the exponentially increasing volume of literature, the major clinical breakthrough is as illusive as ever. This may change in the near future when numerous new and significantly different compounds,

currently under development by the drug industry, complete their clinical evaluations.

This multiauthor book, covers in depth, topics in pre-clinical psychopharmacology. The first part is about the basic classes of drugs with chapters on antidepressants, monoamine oxidase inhibitors, lithium, tranquillisers, psychostimulants, and neuroleptics. The second part is devoted to a review of drugs of abuse covering the psychopharmacology of the opiates, hallucinogens, cannabis, alcohol and cocaine and there is also a section on behavioural teratology and toxicology.

The large section on drugs of abuse including a chapter on PCP is topical. Interesting issues such as the often alluded but rarely quoted relationship between opiates, acupuncture and the placebo effect are also covered.

Issues which may be of particular interest to the psychiatrist include discussions about the 4 postulated dopamine receptors, and the several benzodiazepine ligands, their positive and negative actions and the possible clinical implications. New therapeutic approaches hinted at, include the search for an endogenous ligand for imipramine, presumably along the lines of an endorphin, and the suggestion that imipramine acts as an intraneural false transmitter.

The literary style is of a scientific review with copious references and cross references. One excellent chapter has over 600. Despite that, the book is readable and engagingly interesting. It does, however, presuppose a considerable background knowledge of the specifics of the subject in order to derive full benefit from the text.

This book deserves to be in every medical library and the bookshelves of those clinicians, academics and researchers with an active interest in the area. The slant of the book is clearly towards the preclinical aspects. It is not the book for a casual browser but an authoritative reference work.

COSMO HALLSTRÖM, *Consultant Psychiatrist,*
Charing Cross Hospital, London

Psychopathology Among Mentally Retarded Children and Adolescents - Developmental Clinical Psychology and Psychiatry 6. By JOHNNY L. MATSON and CYNTHIA L. FRAME. London. Sage Publications. 1986. Pp. 119. £19.75. (hb), £10.00 (pb).

This is one of a series of books on problems of children and adolescents designed to present the latest research, theory and trends in clinical practice together with the likely future direction of research in the field. This book, sixth in the series looks at behaviour disorders and emotional problems in children and adolescents with a mental handicap.

The point is made that there is so far little in the way of research and this has concentrated mainly on the less handicapped and the older child. Little has been done to understand the behavioural difficulties of the profoundly handicapped child and in particular the younger age group. The book makes the point that until recently behaviour disorders and mental retardation were often regarded as mutually exclusive diagnoses and points out the current trend of interest in this field which has already shown that the mentally retarded child has a greater risk of behaviour disorder in its widest sense than the child with normal intelligence.

There is a preoccupation with finding suitable objective measurements such as rating scales or adapting those in use in a population of normal intelligence. Clinicians in the UK will be unfamiliar with many of those discussed and will not have found the lack of such instruments a handicap in their everyday practice. It is surprising that many of the drugs mentioned in the section on pharmacotherapy are described by their trade name, which may limit the value of this section in the UK.

Child psychiatrists in training in this country are now being encouraged to include the mentally handicapped child and his family in their practice when there are psychological problems. Many established child psychiatrists are reluctant to encourage this trend, fearing that the mentally handicapped child will present with a discrete and insoluble range of difficulties bearing no relationship to the child of normal intelligence. I do not think that this book will help to dispel this myth and there is the risk that this book will prove de-killing to those who wish to know more about the child with a mental handicap in this country.

JOAN BICKNELL, *Professor of Mentally Subnormality, St George's Hospital Medical School, London*

Depression: A Psychobiological Synthesis. By Paul Willner. Chichester: John Wiley. 1985. Pp. 597. £57.50 \$80.65.

This book has been long overdue: it provides a near exhaustive review of the literature pertaining to the psychological and biological strands of depression with the aim of providing an explanatory psychobiological framework for the complex interaction of these aspects. The author has selectively sampled the literature on psychology, social aspects, physiology, chemical pathology and pharmacology of depression with a particular reference to animal models in a search for links between these strata of analysis before providing a synthesis. The contents of the book are well organised to achieve this aim. In Part 1, the nature of psychobiological explanations is exposed and this is followed by reviews of nosology, cognitive functioning, neuropsychology and of animal models. The chapters on animal models are excellent and examine the comparative validity of these models as they relate to the human situation. In Part 3, the involvement of specific transmitters is reviewed from standpoints of theory, the clinical situation and neuropharmacology, ending with a chapter on their interactions. These reviews, however, are essentially non-critical: shortcomings of methodology, sources of conflicting results of various studies were rarely exposed, which is germane to the aim of this monumental work if the construct provided is to have stability. Part 5, provides elements of a synthesis under aspects of etiology, psychopathology, natural history and effects of treatment ending with a brief summary and a statement on future directions. This final part is very succinctly and lucidly written, but would have been enhanced if the author had provided flow charts and diagrams to outline his framework. The model provided has heuristic value for researchers into 'missing links' and provides new insights into the phenomenon of depression. I found the book stimulating and it should appeal to clinicians and researchers with a broad approach

to the study and understanding of depression. The book has a very rich bibliography of over 2000 references.

M. T. ABOU-SALEH, *Senior Lecturer in Psychiatry, University of Liverpool*

The Unfinished Revolution in Italian Psychiatry: An Italian Perspective. Edited by RICHARD F. MOLLIKA. *International Journal of Mental Health*. New York: M. E. Sharpe 1985. Pp. 212. \$46.20

I was hoping that when I opened this book that it would bring a touch of colour to the stark black and white picture of Italian psychiatry that has been portrayed in the psychiatric literature over the past five years. This volume comprises two issues of the *International Journal of Mental Health*, and opens with Dr Mollica, who sets the scene well by explaining how Franco Basaglia's ideas developed from the political philosophy of Antonio Gramsci, who died in one of Mussolini's prisons in 1937. From this, one understands how politics and psychiatry in Italy are inextricably intertwined and why the Italian reform movement is so pre-occupied with the "second disease", the process whereby psychiatric patients became outcasts because of their segregation from society (Emarginazione). Basaglia's movement was not concerned with the primary treatment of psychiatric illness, only the form and setting in which it took place.

Unfortunately, after this promising start, we return to black and white accounts of the consequence of mental health reforms since Law 180 was passed in 1978. Thus Basaglia's widow (Franca), Jervis, Paparo and Bacigalupi, Carrino and Crepet all write as committed supporters of the reform and blame administrative incompetence or financial strictures in areas (e.g. the South of Italy) where the reform has limited or no benefit. Cassano, Mauri and Petracca, Hochmann, Jones and Poletti all oppose the reform and predictably find evidence to support their views. The most colourful contribution comes from Mario Tobino, who is both novelist and a psychiatrist, and who writes eloquently about the "foul wind" that has created chaos in Italian psychiatry.

But the colour comes from his literary style, not from the quality or information that the psychiatrist needs in judging the Italian reform. If this book was a tankard of beer it would be unfit for sale, because its contents are 80% "head" and only 20% beer. Most of the beer comes from our own Douglas Bennett, who is the only contributor who confines comment to the interpretation of data. He describes the changes that took place in the psychiatric services of Trieste between 1968 and 1978. The number of patients was reduced from 1260 to 366 and this was achieved without any significant change in staffing numbers. Although a great deal of information is missing about these patients one cannot help coming to the conclusion that for the typical psychiatric patient in Trieste the ideas that lay behind the reform of 1978 have been of benefit.

As for the rest of Italy, the "unfinished revolution" referred to in the title has an even less finished perspective. Psychiatrists there who are genuinely committed to improving psychiatric services find at least something of value in

the reform and those whose work is confined to hospital or private practice continue to criticise. Law 180 has confirmed the existence of three other laws; (1) good psychiatry cannot be created by legislation, (2) community psychiatry cannot be carried out without community resources, and (3) rhetoric is no substitute for data in the evaluation of psychiatric services. This book represents the opening speeches of prosecution and defence in the "Case of Italian Psychiatry". The evidence has yet to be presented.

PETER TYRER, *Consultant Psychiatrist,
Mapperley Hospital, Nottingham*

Metal Ions in Neurology and Psychiatry. Edited by SABIT GRAY, JOSEPH HARRIS, BENG T. HO. New York. ALAN R. LISS. 1985. Pp. 409. £72 00

This volume, which is the fifteenth in the series *Neurology and Neurobiology* has sought to highlight the role of metal ions in the field of neurology and psychiatry.

Recent advances in the chemical and pharmacological fields have increased our ability to measure and study the role of metal ions in various biological situations, thereby increasing our understanding of their role in human health and disease.

The contributors are all eminent scientists in their fields. The coverage is wide, though frequently lacking in depth. The quality and style of presentation also are inconsistent. For example, there is one chapter with over two hundred references and another with none. The chapters are organised on the functional aspects of metal ions so that a clinician who wanted to study the role of metal ions in, say, psychosis may need a careful compilation of pieces of information from several chapters. There are a few excellent chapters of interest to the psychiatrist like 'Vanadium and Affective Disorders' by G. J. Naylor and 'The Problem of Lithium Mechanisms' by J. L. Crammer. These apart, information of an immediate interest to a psychiatrist is contained with a varying proportion in the other chapters.

There are glaring omissions like the work of Carl Pfeiffer on the role of zinc and copper in schizophrenics, however much others may consider this as controversial.

These criticisms apart, the book has a wealth of information, more appealing perhaps to neurologists and pharmacologists. The editors have done well with a difficult task by bringing together a wide range of topics. They have succeeded in their declared interest of 'spotlighting current developments' and 'attempting to bridge the gap between clinical and fundamental approaches'. Their only worry should be, would that be sufficient to make a reader dip into his pocket to find the cover price of £72?

D. P. SRINIVASAN, *Consultant Psychiatrist,
Garlands Hospital, Carlisle*

Love and Tradition: Marriage between Jews and Christians. By EGON MAYER. New York: Plenum 1985. Pp. 311. \$17.95.

Egon Mayer is Professor of Sociology at Brookland College and also President of the Association for the Sociological

study of Jewry. The book is based on a survey of 450 intermarried couples, and attempts to describe what happens when an American Jew marries a Christian. It looks at how they approach their differences in religious observance, family life, and the customs surrounding births, deaths, marriages and rites of passage. It argues love does not conquer all, for tradition dies hard because it serves important human needs.

The book goes a long way towards dispelling the anxiety that intermarriages will eventually lead to the assimilation and therefore loss of Jewish culture and traditions. On the contrary, it seems that in a large percentage (33%) of intermarriages the Christian partner converts to Judaism, while only 10% of the Jewish partners convert to Christianity. In most marriages (43%) neither partner converts but they make an attempt to blend their two heritages and many Jewish customs and traditions are adopted and worked into the family life of the new couple.

This is not a book about marital therapy, nor is it about marital discord. It is a look from a sociological perspective at how the tension between love and tradition is resolved in marriages between Jews and Christians. As there is a tension in all marriages the book is of general interest. It is illustrated with lots of case studies. How applicable the findings are to the mixed marriages that are generated by our own multiracial society is of course open to question. The partners in many of these marriages often come from traditions that are much more difficult to bridge.

The book is worth a read to those interested in the problem of intermarriage and in the inevitable and complex interplay between love and tradition.

C. R. WHYTE, *Consultant Psychotherapist,
Humberstone Grange Clinic, Leicester*

Crisis Intervention - Theory and Methodology. By DONNA C. AGUILERA and JANICE M. MESSICK. Oxford: Blackwell Scientific Publications. 1986. Pp. 253. £14.50.

This small volume has been very popular amongst workers in crisis intervention settings since it was first published in 1970. It is now in its 5th edition, and there are some important changes both in style and content. Previous editions, have, in my view, been only of limited value to psychiatrists and have been written principally for other workers on teams. They have also been stronger on the practical applications of crisis intervention than on theoretical and academic aspects of the subject.

The authors (originally nurses) state in the preface to this edition that "it was not our intent in the previous editions of this textbook to imply that crisis intervention was limited only to one profession", and it is clear that this has been a common criticism. They have responded to this by adding some sections and enlarging others, and usefully by changing the style from a somewhat annoying and patronising second person to the more usual third person. In addition, the references are now at the end of each chapter and are extremely comprehensive.

This book is about 50% longer than before and most of this is taken up with an expansion of the section on dealing with crises.

The authors present a new model for conceptualising the problem in a diagrammatic form, which may be useful as a framework for some and is certainly simple and rational in its approach. The case histories, as always, are presented in a lively and sympathetic way are the strength of the work. The process of intervention is clearly explained and both the practical results and theoretical perspective are discussed.

There are two new chapters—one dealing with 'burn-out'—probably an essential component of any forward-looking book of the eighties—and one discussing socio-cultural influences on therapy which exclusively deals with cultural patterns in the USA and so is of limited value to a British readership.

As a basic introduction to crisis intervention for relatively inexperienced workers from many disciplines, this work is good value. It remains, however, a basic and superficial work and will not be very helpful to anyone with experience in the field.

GILLIAN WALDRON, *Consultant Psychiatrist,
Tower Hamlets Health Authority, London*

Diagnostic Interviewing. Edited by MICHEL HERSEN and SAMUEL M. TURNER. New York. Plenum Press. 1985. Pp. 365. \$37.50.

Ordinarily, diagnostic cookbooks in psychiatry, particularly if centering around a highly formulaic system such as the DSM-III, tend to be dull and boring. This volume, in which a group of American psychiatrists and psychologists put together the theory and practice of diagnostic interviewing, is an exception. The book is divided into three parts. The first part covers basic interviewing techniques and mental status examination. The rest of the book is devoted to the application of these principles to the major psychiatric disorders and special populations such as children and the elderly. Each chapter provides a DSM-II oriented description of the condition in question, followed by guidelines on how to collect and organise the necessary information to arrive at a diagnosis. The contents of the book are clearly and systematically presented.

The main strength of the book lies in the richness of clinical experience portrayed. The contributors share, with the reader, the "special strategies" of interviewing that they have developed in their clinical practice, and illustrate the do's and don'ts of the diagnostic process by suitable case vignettes. Though the presentations are uniformly good, the chapters on Affective Disorders (Van Valkenberg and Akiskal), Personality Disorders (Soloff), and Psychophysiological Disorders (Ward and Chesney) deserve special mention. While most chapters offer an eclectic, "medical model" approach to diagnosis, some contributors, notably Dupree and Patterson (assessing the elderly) present an unashamedly behavioural approach to assessment (not diagnostic) interviewing.

The book is missing in some areas. Neuroses, except anxiety, receive scant mention, there is no section on organic mental disorders, and the assessment of the suicidal and the dangerous mentally ill is hardly discussed. The book is technically well produced, but there are occasional errors.

The editors' stated aim is to provide a "how to do it" manual for beginning practitioners. This purpose is served

eminently by the book in its country of origin. For a novice trainee in Britain where clinical, particularly diagnostic practice is often very different, the contrast reflected in this book may be confusing. For the experienced clinician, however, such a refreshing contrast could be of great interest, and could help to sharpen his diagnostic tools. This book deserves a firm place in all psychiatric libraries.

M. S. KESHAVAN, *Assistant Professor in Psychiatry,
Wayne State University, Detroit*

Social Controls and the Medical Profession. Edited by JUDITH P. SWAZEY and STEPHEN R. SCHER. London: Eurospan Group of Publishers. 1985. Pp. 268. £27.50.

This book consists of a number of papers given at a series of conferences held between 1980 and 1982 as part of the Project on Social Controls and the Medical Profession established by a body known as Medicine in the Public Interest Inc. Little information is given about this interesting organisation beyond telling the reader that it is involved in studying current issues relating to medicine and science in the United States. The project was set up "in response to a perceived need to better define and analyse the governance of physicians as professionals" and, as one might expect from such a large topic, the contributions cover an enormous range from moral philosophy to an engaging piece entitled 'Laying down the scalpel: reflections on competence in a surgeon's career' by the Clinical Professor of Surgery Emeritus at Harvard. There are also papers on becoming a lawyer and on competence and impairment in the clergy which might, at first glance, seem out of place but, in fact, provide an interesting comparative element.

Competence and impairment in the medical profession seems to be the major theme of the book and many of the papers will be of interest to those involved in, and observing, this aspect of medical practice in the United Kingdom. The paper on impaired physician committees (IPC's) by Professor Ruddick of the Department of Philosophy at New York University entitled "Tough Love", Physician Advocacy and Moral Diffidence: Philosophical Reflections is of particular interest even though written in the context of American medicine. There is additionally a 34 page bibliography on impaired professionals and medical competence which will be of benefit to those wishing to pursue these topics. All in all this is an interesting book about certain aspects of medical practice in the United States. Much of what is written cannot be transferred to the British scene in total but may represent signs of things to come.

BILL MORGAN, *Lecturer, Department of Social Policy,
Newcastle University*

Clinical Neurology for Psychiatrists. 2nd Edition. By DAVID M. KAUFMAN. Orlando, Florida: Grune & Stratton. 1985. Pp. 431. \$44.50.

What is distinctive about a neurology text-book directed specifically at psychiatrists? Taking this one as the paradigm it means a beautifully-produced, clearly-written comprehensive neurological text enlivened by skilful and

informative line drawings. More attention is perhaps devoted to the differentiation of psychogenic disorders and the causes of dementia than in the average neurological text-book and readers are instructed to prepare formulations rather than make diagnoses but few other concessions to psychiatrists are discernible.

The contents are in two parts. Part 1 contains six chapters that cover classical anatomical neurology including peripheral nerve and muscle disorders. Particularly useful is the introductory chapter entitled 'First Encounter with a Patient' which provides helpful advice on structuring the interview and examination. The chapter on psychogenic disorders wisely advocates a sceptical approach to the diagnosis of hysteria and warns of the frequent concurrence of organic and psychogenic dysfunction in the same patient.

Part 2 covers major neurological symptoms including headache, seizures, pain, visual disturbance, aphasia and agnosia, sleep disorders, intellectual deterioration and sexual dysfunction as well as specific disorders such as multiple sclerosis, congenital abnormalities, cerebrovascular disease and brain tumour. Topicality is demonstrated by the inclusion of a section on AIDS and an illustrated chapter on investigative techniques including computerised scanning, positron emission tomography and nuclear magnetic resonance.

There are few aspects to criticise. The author is sceptical about epileptic psychoses and the statement "brain tumours rarely occur in young and middle-aged adults" is misleading. The fifty or so questions and answers at the end of each chapter and the 275 at the end of the book, although a useful aid to learning and revision, do materially inflate the size and presumably the price of the book.

Overall, however, this book is a pleasure to read and would benefit candidates for the MRCP as much as the MRCPsych. It can be recommended to clinical tutors as suitable library material.

K. DAVISON, *Consultant Psychiatrist and Lecturer,
University of Newcastle-upon-Tyne*

Psychological Applications in Psychiatry. Edited by
BRENDAN P. BRADLEY and CHRIS THOMPSON. Chichester:
John Wiley and Sons Ltd. 1985. Pp. 247. £19.50.

The editors of this volume have asked contributors to describe psychological approaches to understanding and treating disorders which correspond to psychiatric conditions and to base their discussion on research findings. The chapters cover obsessional-compulsive disorders, anxiety states, depression, schizophrenia, drug and alcohol dependence, neuropsychology, childhood disorders, psychogeriatrics, rehabilitation, and assessment.

The contributors provide well edited, up-to-date reviews. For such a concise volume it is surprisingly comprehensive and it serves as an excellent introduction to both psychiatry and clinical psychology.

Certain themes and trends predominate throughout the book. For example in his excellent chapter describing anxiety states Mathews argues that these disorders should be viewed as emerging from biophysical, psychosocial and cognitive-behavioural chains of events. Consequently, interpretation and treatment should take account of all

these factors or processes in a way that compliments or even replaces an emphasis on diagnostic categories and a one dimensional, medical approach. Similarly, in his description of the models of drug and alcohol dependence and his summary of treatment approaches, Gossop is highly critical of traditional views and methods. In their place he favours an alternative model using relapse prevention procedures in the form of cognitive-behavioural self-control skill training. The orientation adopted by Mathews and Gossop is echoed by Hemsley in his chapter on schizophrenia. He too concentrates on the importance of assessing the psychological and environmental factors using a functional analysis. Furthermore, he describes how among research and treatment possibilities one of the most interesting, exciting and promising newer approaches involves modifying cognitive and behavioural impairment via training in coping strategies. According to Powell a reorientation of this sort is also occurring in the field of neuropsychology. He describes a change in the role of the neuropsychologist from offering a diagnosis to that of providing an assessment of real life functioning in order to initiate management programmes and remedial training, for example using memory training for amnesics and biofeedback to reduce epileptic convulsions.

This book provides a real sense of the major concerns of clinical psychology and psychiatry in the 1980s. Very highly recommended.

PETER BUTCHER, *Principal Clinical Psychologist,
The London Hospital, Whitechapel, London*

Psychiatry: The State of the Art. Volume 7 - Epidemiology and Community Psychiatry. Edited by P. PICHOT, P. BERNER, R. WOLF and K. THAU. New York: Plenum Press. Pp. 693. £75.00.

Conscientious attendance at large academic conferences is hard work - the range of subjects is wide, the time given to each often short, and what appears in prospect, to be a deliciously varied menu all too often turns out to be a rapid procession of dishes which are all sampled but none really enjoyed before the next flavour appears. Proceedings of such conferences can produce the same effect as a jaded palate, without the pleasure of the social intercourse available at the live event.

The VII World Congress of Psychiatry, held in Vienna in 1983, has produced a series of 8 large volumes, of which this is the seventh. The compilation of all the contributions, from plenary sessions to poster presentations, into a coherent whole, has been a mammoth task of editorship, which has not, at least in this volume, been entirely successful. Perhaps it is by nature a somewhat 'catch-all' volume, but there is not a great deal of epidemiology in the work, which largely concentrates on community psychiatric issues. Some sections might have been placed with advantage with parts of other volumes, for example the papers under 'General Hospital Psychiatry' with the 'Psychosomatic Medicine' section in Volume IV. Similarly, some titles seem to have no particular relevance to social and community issues - the section on antidepressants in particular, which contains two or three papers entirely concentrating on psychopharmacology.

The most interesting sections are those on mental health services planning and delivery, where I found papers by Goldberg and Wing to be particularly helpful reviews. The most coherent and discrete section considers ways of helping the family cope with schizophrenia, and this contains some interesting international research findings which taken as a whole may truly reflect 'the state of the art' for this topic.

It is an opportunity to read of work done by a wider international authorship than that usually brought together in one volume, but in general the result is unsatisfactory. There are 103 papers in 18 sometimes arbitrary sections—some of the papers are reviews, some preliminary reports of research projects—the standard of English is immensely variable, the typesetting and reference style inconsistent. In conclusion, I would not think that many people would find enough original material presented in sufficient depth to justify its high price.

GILLIAN WALDRON, *Consultant Psychiatrist,
Tower Hamlets Health Authority, London*

Depressive States and Their Treatment. Edited by VAMIK VOLKAN. New York: Jason Aronson. 1985. Pp. 456. \$40.00.

This book, edited by Vamik Volkan, Professor of Psychiatry and Training Analyst aims to bring together the psychodynamic and psychobiological views of depression. In so doing, the contributions demonstrate the interplay of environmental stresses; psychodynamic factors; genetic, endocrine and biochemical factors in the causation of depression. Though each author offers some interpretation of the interplay, they do so from their own particular focus. Volkan rationalises these by dividing the books into five parts: Basic Concepts: Mourning and Depression in the Life-Cycle: Life Situations: Psychotherapy: Somatic Issues.

The basic idea of the book works quite well and each part may be read in its own right. This is with the exception of Part III on Life Situations which seems incomplete and somewhat contrived in comparison with the others. Basic Concepts provides introductory text, starting as it does from Freud's 'Mourning and Melancholia'. D Wilfred Abse offers two illuminating chapters examining depression from depth psychology. The next part on Life-Cycle works best when it focuses on early childhood experiences as precursors to later adult depression.

The part on psychotherapy confirmed by fears; that everyone finds depressives a demanding and constantly unrewarding group of patients with whom to work. Volkan gives us a concise exposition of his re-grief therapy, but perhaps the most imaginative chapter is by Sheldon Heath, warning us of the risk we all take of becoming a container for our patients' depression.

The final part of the book offers excellent reviews on the psychobiology of depression and its somatic treatment. However, by way of contrast, Morewitz warns that in

primary health care depression may be disguised by somatic presentation. As Morewitz notes the doctor has a tendency to see the patient as a body/mind split and as a whole this book goes a long way towards integrating this split as regards depression.

ALAN PRODGERS, *Senior Psychotherapist,
The Red House (Psychotherapy Services),
Salford Area Health Authority*

Handbook of Psychiatry 8th edition, ROBERT PRIEST and JARROLD WOOLFSON. London: William Heinemann Medical Books. 1986. Pp. 211. £7.95.

This is the 8th edition of a popular standard undergraduate textbook which was originally Minski's *Handbook of Psychiatry for Students and Nurses*. Despite several facelifts, or perhaps because of them, it remains appropriate both for undergraduate students of medicine and for nurses.

The book was last revised in 1979 and the two authors have made a number of changes for this 8th edition. Minski's name has been dropped and the book has an attractive new jacket design. The chief merit of the book in previous editions has always been its simplicity and clarity. Keywords are picked out in bold text and most of the difficult controversial areas of psychiatry are avoided or reduced to their noncontroversial essentials. The 8th edition retains these features.

The chapter on Psychiatry and the Law has been completely rewritten by John Hamilton to take account of the changes caused by the new Mental Health Act. There is also a new chapter on psychosomatic disorders in which the concept is illustrated by three eating disorders, anorexia nervosa, bulimica nervosa and obesity. A section of defence mechanisms has been added to the introduction and a short section on psychiatric disorders of pregnancy and the puerperium. A brief section on adolescence has been added to the child psychiatry chapter.

The main changes have been in the treatment chapter. Sections on barbiturates, other older hypnotics, insulin-coma and modified insulin therapy have all been dropped at last. Some of the newer antidepressants have been added, although it is likely that at least one of them will have to be precipitately dropped by the next edition. The substituted benzamides have been added as a class of neuroleptic and the classification of antipsychotic drugs has been clarified. The authors have also added a section on care of the dying patient to the psychotherapy chapter.

In summary, this 8th edition of a popular textbook, has brought the text up to date and it seems that as the authors gain confidence in their ability to simplify without distortion they are expanding the subject matter into more controversial areas.

C. THOMPSON, *Senior Lecturer in Psychiatry,
Charing Cross and Westminster Medical School, London*

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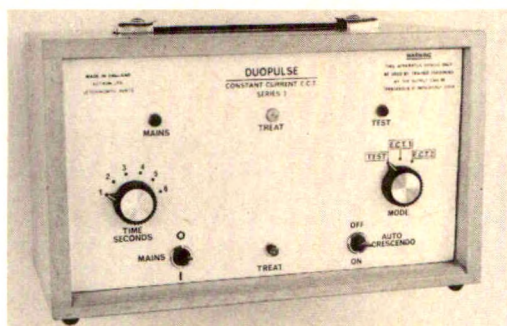
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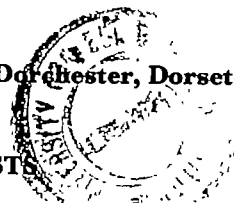
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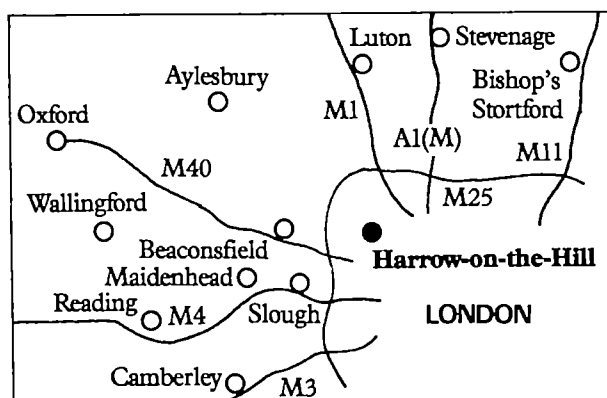
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Annotation

Long-term Psychiatric Patients in the Community

K. JONES, M. ROBINSON and M. GOLIGHTLEY

The reduction of mental hospital populations in Britain and the United States has generated a considerable amount of literature on policy, but detailed studies of the effects on patients and the conditions under which they live after discharge are rare. In the United States, a National Institute of Mental Health review of the literature commented that "the question of what actually happens to patients who leave mental hospitals and re-enter the community is largely unanswered" (Bachrach, 1976).

In Britain, there have been three main research approaches:

- (a) computerised case-register studies based on the Camberwell model (Wing & Hailey, 1972), of which Cheadle *et al* (1978), Howat & Kontny (1982) and Sturt (1984) are recent examples
- (b) studies of patient groups from particular agencies, such as day hospitals (Milne, 1984), assessment units (Eagles & Gilleard, 1984), district general hospital units (Gibbons *et al*, 1984) or ward support schemes (Mitchell & Birley, 1983)
- (c) studies of the input of specific professional groups, such as general practitioners (GPs) (Shepherd, 1976), social workers (Fisher *et al*, 1984), or community psychiatric nurses (Paykel *et al*, 1982).

All three types of study are service-based. They provide a valuable approach to the evaluation of services, but give only limited information on what the patients in the ill-defined area called 'the community' actually experience. Some patients may be receiving a complex of services from different agencies, while others receive few or no services. Detailed tracer studies which take the patient rather than the service as a focus are needed. Who helps? Who fails to help? How do hospital-based and GP services fit together with social work, private, and informal (family care) services? The move from hospital-based to community-based services involves a sizeable re-

definition of patients' problems from 'medical' to 'social'. Does it work in practice?

Studies involving interviewing ex-patients and their relatives or other carers are difficult, and therefore rare. Patients move, and are sometimes difficult to trace, so there is a risk of attrition in the sample. Some prefer to forget their time in hospital, and do not wish to be interviewed; some, in continuing distress or confusion, may misunderstand the purpose of the exercise, and require sensitive handling; and some may not give coherent replies. The study of non-medical services is new to most medical research workers. The few recent and relevant follow-up studies (McCreadie, 1982; Sadavoy & Reiman-Sheldon, 1983; Johnstone *et al*, 1984) have all been concerned with symptomatology rather than with the quality or mix of care. An opportunity to carry out a study for York Health District on patients discharged from the three York mental hospitals (Clifton, Naburn, and Bootham Park) into the York area was therefore welcomed as a pilot project. A full report on this project is available (Jones, 1985).

The two samples chosen for the follow-up consisted of:

- (a) the long-stay group: all patients who had been continuously in hospital for over one year, and who were discharged in the period 1 April 1982 to 31 March 1984 ($n=50$)
- (b) the elderly confused group: 100 patients selected randomly from a total of 203 aged 65 and over with a diagnosis of senile dementia (ICD categories 290.0-290.9, World Health Organization, 1978) and discharged during the same period.

Perhaps the most important finding is that the project was viable. All but two of the patients (elderly confused who had left the district) were traced, and no major objections to the research procedure were encountered. The project was approved by the local Medical Ethical Committee. In all cases, the Responsible Medical Officer agreed to the follow-up.

Patients' general practitioners were consulted, and in only four cases did they think that an interview would be too stressful for the patient: these cases were removed from the sample. The interviewers (two senior social workers, two college lecturers, and a psychologist) were instructed to withdraw at once if either patients or their carers had any objection to the interview procedure: this occurred in only 12 cases. Most were happy to talk, and many seemed to find a friendly visit helpful. The only complaint came from an ex-patient who had *not* been included in the sample.

In-depth interviews, based on checklists, lasted from one to three hours. The interviewers completed both the checklist and a detailed case study for each patient. The case studies produced a wealth of data from which it would be possible to derive a substantially refined research instrument for future studies, with an improved computer database.

Results

The long-stay sample

Patients interviewed were evenly divided between men and women, and covered a wide age-range—from 24 to 82. The length of stay in hospital varied from just over a year to 43 years. Over half had a diagnosis of schizophrenia. The older patients tended to have one admission and a long hospital stay, while the younger ones had multiple admissions and shorter stays. At the time of interview, only five were living in their own homes or a family home. The rest were scattered between local authority homes or hostels, private registered homes, group flats, and lodgings. None were in prison; one was sleeping rough, and two were back in mental hospital. Placements had been made with great care (York has a homefinder who believes in 'intuitive matching' between the patient and the setting—a highly individual process) and most patients appeared to have made a reasonable adjustment.

The elderly confused sample

Of this group of 100, 35 had died before the time of interview, and 23 were back in hospital. Three of these patients had been discharged in their nineties. Only ten of the sample were found to be living in their own homes or a family home. Others had been discharged to local authority or private registered homes, or to sheltered housing. The elderly confused in their own homes were much less well-supported than the younger long-stay patients. Where there was only a mild degree of senile dementia, the family was

large enough to spread the burden of care, and there was enough money to cushion the situation, the problems were manageable; but where the mental condition was severe and disruptive, there were physical complications such as blindness or incontinence, the burden of care fell exclusively on one relative (usually a husband or wife, also old and often in failing health), and the financial resources were strained, the situation could be nearly intolerable. Neighbours offered little help, and friends were scarce. Although the health and social services were initially involved, visits tended to tail off after a few months. The expectation of the current model of community care is that patients will improve after discharge from hospital; senile dementia does not fit the pattern.

Hospital discharge plans

These were faithfully drawn up, but tended to be unrealistic. They made no mention of where the patient was expected to live (presumably on the assumption that they would all return to their own homes). They did not list GP or out-patient clinic services, and there was a marked under-use of social services. Most discharge plans recommended the use of only one community-based service, although patients in fact needed several in combination. The situation after discharge was often unstable, and the need for support more complex than the discharge plan data suggested. We concluded that what was needed was not a 'discharge plan' (which clears a hospital bed), but a community care plan which offers a possibility of stable living conditions with adequate support.

Success of placement

The patients in these samples did not bear out the gloomier prognostications of those who distrust care in the community—similar surveys in other areas might have less positive results. Of the total of 38 who died before the interviews took place, 35 died of natural causes (two elderly confused patients died as a result of falls, one in hospital and one in a home, and one long-stay patient committed suicide). But were the rest happier in or out of hospital? It is often taken as axiomatic that any kind of life 'in the community' is better than staying in a mental hospital; as the survey results indicate, however, patients go out to a variety of settings, and not all of them are necessarily beneficial.

Quality of life study

The research team attempted to devise a scale

sufficiently flexible to cover both life in hospital and life in the variety of settings to which patients were discharged, and the matching of conditions for each patient in both settings. While many quality-of-life scales have been constructed for different purposes, the combination of hard and soft data poses severe problems of measurement, and there are difficulties in weighing individual items. Scales based on some concept of 'normal living in the community' are open to the objection that we have little idea what is 'normal' for severely handicapped or disturbed people. After studying existing scales, the team came to the conclusion that the construction of a new and simplified scale was the only useful approach. The York Scale is based on Maslow's (1943) hierarchy of human needs. It measures four groups of needs (survival, safety/security, purpose, and independence) on three dimensions: needs met, needs partly met, and needs unmet. The scale needs further development, but it was found that

- (a) patients' own homes scored well on choice and autonomy, but not on health care, personal care, activities, and companionship
- (b) local authority homes provided personal care, but scored poorly on health care, privacy, and activities
- (c) private homes (usually run by psychiatric nurses) provided good personal and health care and activities, but were often cramped and lacking in facilities
- (d) mental hospital wards provided best for basic survival needs, health care, and activities, but not for personal choice, privacy, or autonomy. Nor did they provide for stability, since patients might be moved to another ward or discharged at any time.

Conclusions

The findings of the survey should be treated with caution, since the samples were restricted in scope and comparatively small. However, they provide pointers for practice and for future research.

Most of these patients were still living in fairly institutional surroundings. Whether they were better or worse off than in hospital depends on a variety of factors relating to the individual patient and the individual setting – there is no clear general answer indicating the superiority of 'hospital care' or 'community care'.

The questionnaire had been constructed in the expectation that most of the patients in the samples would be found in their own homes, and that if they

were fit for discharge, they would be able to sustain an interview. Both of these assumptions were disproved. Only one in ten of the patients were in their own homes, and few were capable of answering questions. Most of the information came from people such as relatives, officers in charge of homes, landladies, hostel wardens, and hospital staff. Often even professional staff were uncertain in their replies – they could not remember the names of GPs, or say when a visitor had last called, or whether patients had relatives. Patients who are incoherent, or passive and undemanding, do not seem to inspire good record-keeping, although they may need it more than most.

Even in York, a fairly compact city, the study revealed the great complexity of care in the community: the difficulties which health and social services have in working together when they have different legislative bases, different working styles, different patient groupings, different planning and budgeting cycles, and different professional backgrounds; the problems of the small voluntary societies in trying to work with the statutory 'giants'; the isolated pockets of private care, and the difficulties of co-ordinating all these services with GP care. The 90 patients interviewed – all living within a limited area – had 67 different GPs. Repeated official exhortations to collaboration and co-operation (Department of Health and Social Security, 1975, 1978, 1981) will be of little effect unless attention is given to finding administrative solutions which provide a mix of services where needed.

Modest research projects of this kind, which can be of practical and immediate use to the psychiatric services, may be of particular value when a major policy shift is being implemented. They need to be tightly scheduled, and to provide quick returns. The survey reported here was planned, executed, and reported back to York Health District within twelve months. It took place in the context of rapidly-developing services; in the past year, two additional consultant posts have been established, with specific responsibilities to develop after-care services for younger patients with chronic handicaps, and for the elderly confused; numbers of community psychiatric nurses have been increased; an 'at risk' register has been set up; funds have been released to social services to provide for collaboratively planned day care; community mental health teams are being formed to work with GPs in designated localities; and plans are well ahead for the provision of four community resource centres and eight community units for the elderly. Small-scale in-depth research can help to maintain the momentum of change, and provide rapid feedback on groups in special need.

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Lithium Treatment: A Refresher Course*

M. SCHOU

Updated guidelines are presented for the safe and effective use of lithium; covering indications, treatment management, duration of prophylactic treatment, side-effects and their prevention, overdosage, interactions, and contraindications. Alternatives to lithium and patient-doctor co-operation are discussed.

Many patients are given lithium treatment for long periods of time, and questions may arise for patient and doctor before and during treatment. This paper is an attempt to provide answers to questions often asked; answers are also proposed for questions which may not be asked but should be. The didactic aim and space limitations have necessitated a certain bluntness of statement. The opinions presented are the author's own; others may hold different and equally valid views.

Indications

Although given for some somatic diseases (neutropenia, cluster headache), lithium is used primarily for the treatment of psychiatric illness.

Mania

As an antimanic treatment, lithium preceded the neuroleptic drugs by about five years, and its efficacy has been firmly established. Lithium nevertheless appears to be used rather sparsely on this indication, and one wonders whether the preference for neuroleptics may not to some extent stem from impatience on the part of staff and doctors. The full antimanic effect of lithium develops gradually over about a week. Acute or fulminant mania must therefore be treated with a quick-acting neuroleptic or with ECT, but in less fierce cases, lithium is preferable from the patients' point of view, because it induces less feeling of restraint.

In mania of strong intensity, it may be advantageous to start treatment with a neuroleptic drug in combination with lithium, and then to reduce the

dosage of the former over the following week, as lithium takes effect. Therapeutic intervention is rarely popular with manic patients, but avoidance of the hard-hitting neuroleptics may make treatment more acceptable.

Depression

Early clinical observations and trials failed to show antidepressant effects of lithium, but later studies, including some with placebo-treated controls, have revealed that lithium does in fact exert some therapeutic effect during a depressive episode. Depressions occurring as part of a bipolar illness may respond better than those with a unipolar course. The effect of lithium may set in later than that of the conventional antidepressants. There are at present few who use lithium as first choice antidepressant treatment, but in patients with a tendency to postdepressive hypomanic upswings, this therapy may merit consideration.

The antidepressant properties of lithium seem to be of value in a different setting. Several studies have shown rapid improvement when lithium was added to antidepressants in patients whose depression had been refractory to treatment with the latter drugs alone. Whether this is the case also for those depressions which tend to take a chronic course remains to be seen.

Recurrent manic-depressive illness

The prophylactic effect of lithium in manic-depressive illness is by now one of the best documented in psychiatric pharmacotherapy, and it must be considered a grave medical error not to offer prophylactic treatment with lithium (or one of its alternatives) to patients with frequent and severe recurrences.

*Based on a lecture given to the Royal College of Psychiatrists in January 1986

The following points are clinically relevant.

- (a) Prophylactic lithium treatment is equally effective in women and men and, provided appropriate dosage adjustments are made, in young and old.
- (b) In bipolar patients, the prophylactic action has been found better in some studies against depressive than against manic relapses. Other studies have shown the opposite. Most studies, however, indicate equal efficacy against the two types of relapse.
- (c) In some patients, the effect seems to be primarily a reduction of episode frequency, in others a reduction of episode intensity. In many patients the two are combined.
- (d) Patients with frequent episodes, more than 3–4 per year, may respond poorly to prophylactic treatment, but sometimes they respond excellently.
- (e) In some patients, the prophylactic action of lithium sets in gradually, so that the full effect is seen only after 6–12 months of treatment; relapses do occur during this period, but with gradually decreasing frequency and intensity. In other patients, no relapses are seen once lithium treatment has been started.
- (f) Lithium does not seem to lose its effect.
- (g) Lithium is not addictive.

The efficacy of lithium prophylaxis in unipolar illness has been the subject of debate, but dissent has been more of opinion than of evidence. A few reports have indicated that lithium is less effective in unipolar than in bipolar illness, but most studies show it equally effective in the two forms of the disease. It is perhaps significant that doubts about efficacy in unipolar illness have been raised primarily in the United States, where the concept of unipolar illness may be used more loosely than in Europe.

The question of when prophylactic treatment should be instituted has received different answers, and a general one may not exist. Early prophylactic trials were carried out on patients with two or more episodes within the last two years, but this criterion clearly excludes too many patients who might benefit from the treatment. Another suggested startpoint is when bipolar patients have suffered their third and when unipolar patients have suffered their fourth episode, and even this may be too late. Suicides often occur earlier. Individual factors must be taken into account, such as family and work situations, severity and duration of episodes, and response to conventional therapy. The decision about prophylactic treatment should be made jointly by the psychiatrist and the well-informed patient and spouse.

Aggressiveness

At an early stage, lithium was designated 'specific' for manic-depressive illness, because it acted on both the manic and the depressive manifestations of the disease and because it did not have the broad therapeutic spectrum of the neuroleptics. Lithium effects have nevertheless been clearly demonstrated also in psychopathological conditions which cannot (or can only by stretching the term) be called manic-depressive. Foremost among these is episodic pathological aggressiveness with explosive behaviour. Such effects have been seen in persons with unstable character disorder, in sociopathic prison inmates, in children, and in mentally retarded persons. In the last-mentioned, the antiaggressive action was apparent both when the aggression took the form of self-mutilation and when others were attacked. It is not clear whether the effect of lithium is exerted primarily on the aggressiveness itself or on the – often irregular – periodicity.

Documentation of lithium effects on periodic aggressiveness is so solid, based on double-blind as well as on single-blind studies with large numbers of treated subjects, that one would expect lithium to be used rather widely on this indication. That has not been the case, and a reason may be the prevailing general hesitation about administering drugs to children and to inmates in penal or ward institutions. It may be worth remembering, however, that those responding to lithium reported substantial subjective relief from their previously uncontrollable bouts of rage and violence.

Other

The evidence concerning lithium effects in schizoaffective illness and schizophrenia ranges from clearly negative results to highly gratifying treatment outcome. The latter concerns not only affective elements but also, according to some studies, schizophrenic core symptoms. The question requires further enquiry, but lithium treatment, alone or in combination with neuroleptics, may be worth consideration in patients where the psychopathology shows periodicity, regular or irregular.

Positive reports about lithium effects in alcoholism not secondary to recurrent affective illness are balanced by clearly negative studies. Uncontrolled trials have indicated lithium prevention or amelioration of premenstrual dysphoria, but placebo-controlled studies on larger patient groups have not.

Treatment management

Preparations and dosage pattern

It is of little importance whether lithium is taken in tablet form, in capsule form, or as a syrup. Nor is it of consequence whether one employs lithium carbonate, lithium citrate, lithium sulphate, or any other lithium salt, as long as the preparation used has constant release properties, and releases the full content of lithium during the passage through the gastrointestinal tract. Low or variable release involves risk of underdosage with relapses or overdosage with intoxication.

There has been discussion about the relative advantages of two treatment regimens: (a) one with a softly undulating course of the serum lithium concentration over 24 hours, as produced by administration of sustained-release lithium tablets or by the use of two or more daily doses, or both, and (b) one with higher peaks and lower troughs of the serum lithium curve, as produced by administration of conventional tablets once per day. The question has not been definitively resolved, and it is complex, because wanted effects and side-effects may respond differently to different treatment regimens, and different side-effects may not respond in the same manner. Comparisons between clinics have been carried out, but final answers can be obtained only through prospective within-clinic trials with random allocation of regimens.

Even though we have taken an active interest in the problem at our hospital, we do not at present plan such studies, because it is our impression that the general dosage level is much more important than the treatment regimen. About five years ago, we reduced dosages and serum levels by about 20%, from an average dose of 30 mmol/day to 24 mmol/day and from an average serum level of 0.85 mmol/l to 0.70 mmol/l. This has not led to any marked reduction in prophylactic efficacy, but a number of side-effects have fallen drastically in frequency and intensity. In many patients, side-effects have ceased to be a problem.

Dosage adjustment

Renal lithium clearance and sensitivity to lithium vary from one person to another, and individual dosage adjustment is necessary. Methods for predicting the maintenance dose from the serum lithium level 24 hours after a test dose have been devised. Alternatively, a stepwise procedure may be used: (a) administration of a low test dose—1 or 2 tablets daily—for one week; (b) determination of the serum

lithium concentration and adjustment of the dosage to produce a serum concentration between 0.5 and 0.8 mmol/l; (c) individual after-adjustment if necessary: increase on the occurrence of relapses, decrease in case of troublesome side-effects.

Laboratory monitoring

Laboratory tests before lithium treatment may include examination of the urine, serum creatinine and thyroid stimulating hormone (TSH), haemoglobin, sedimentation rate, blood pressure, electrocardiogram, and body weight. These should be supplemented by others if there is a medical history or suspicion of somatic illness.

During treatment, the serum lithium concentration should be determined once per week during the first weeks, and thereafter at longer intervals. The blood samples for serum lithium determination should be drawn in the morning, 11–13 h after the last intake of lithium. A serum concentration between 0.5 and 0.8 mmol/l is appropriate for most patients, but individual adjustment to levels outside this standard range may be necessary. Supplementary examinations, for example of serum creatinine and TSH, may provide extra safety, and suspicion of intoxication or complications requires additional laboratory control.

The frequency of the control visits is usually determined by clinical considerations. It is important for the co-operation between patient and doctor that they meet at intervals to discuss treatment progress, side-effects, and dosage adjustment, as well as possible social or personal problems associated with the illness and the treatment. The main importance of the laboratory visits may lie in the provision of a structured framework for these contacts. Intervals between routine checkups therefore depend on the patients' personality, intelligence, and educational level, the extent of information and instruction they have been given, the clinic routine, etc.

Recommendations given here deal with minimum requirements for laboratory monitoring under ordinary circumstances. However, sometimes such 'ordinary' circumstances are extraordinary. In developing countries, even strongly indicated lithium treatment may not be instituted because laboratory facilities are lacking, and in countries without a socialised health system, poor patients may be deprived of this treatment because they cannot afford the cost of regular laboratory examinations. In my opinion, it is not irresponsible to prescribe lithium treatment even without laboratory monitoring if the alternative is that no prophylactic therapy is given.

Duration of prophylactic lithium treatment

Statistical evidence indicates that the frequency of manic-depressive relapses tends to rise with increasing illness duration, and more rapidly in bipolar than in unipolar patients. It is therefore likely that a patient who is started on lithium owing to frequent episodes within recent years will also need lithium during years to come. It seems, however, psychologically important that psychiatrists avoid the blunt 'for life' statement to patients who start, or consider starting, lithium treatment. The question should be kept open for future discussion in the light of experience.

In some patients, subclinical 'reminders' reveal that the disease is still active underneath and that discontinuation of lithium is inadvisable. In other patients there are no signs of ongoing illness, and the patient's wish to try stopping lithium may become urgent. Discontinuation under strict supervision can in some instances be executed with impunity, but in most cases recurrences develop sooner or later, and the patient goes back on lithium, so much the wiser.

Lithium can usually be stopped abruptly without the appearance of withdrawal symptoms. In a few patients withdrawal is followed by anxiety; stepwise discontinuation and psychological support may be indicated. In some studies, the early appearance of relapses after discontinuation of lithium has suggested that rebound may occur. Other studies, including some with large patient groups and double-blind design, showed no evidence of rebound.

Side-effects

Psychological side-effects

In view of its striking effects on manic and depressive episodes of abnormal mood, lithium exerts remarkably little action on normal mental faculties. Most patients in long-term lithium treatment feel normal and function normally. There are patients who complain of impaired memory or reduced reaction speed, and although in some instances this may reflect deprivation of manic hyperacuity, there is reason to believe that lithium itself occasionally exerts such actions in sensitive persons. The same effects have been noted by normal subjects who took high lithium doses.

Lithium treatment counteracts not only manic and depressive episodes of abnormal mood; sometimes it also produces a more stable mood during the intervals between episodes. This is felt by some patients as a gain, providing less fluctuation and better self-control. Others miss their previous enthusiasm, and

feel that life under lithium has become less interesting and colourful.

The effect of lithium treatment on artistic productivity has been the subject of several investigations. The most extensive study reported that out of 24 manic-depressive artists in whom lithium treatment had brought the disease under control, six found that their creative power had diminished, six that it was unaltered, and twelve that they created more and in some instances also better when prevention of manic and depressive episodes permitted a more even working rhythm and better artistic discipline.

Lithium effects on mental functions are readily reversible, and disappear when lithium is discontinued.

Somatic side-effects

Some patients interrupt lithium treatment because they suffer troublesome somatic side-effects, but this is relatively rare when one uses the lower dosage and serum lithium ranges mentioned above. Proper attention should nevertheless be given to even trivial physical side-effects, because in long-term treatment, they may influence life quality significantly.

Many side-effects are more pronounced when the serum lithium level is high, and the principal remedy should be an attempt to use lower doses and serum levels. Fine adjustment in each patient is essential, and sometimes the reduction of serum lithium by 0.1–0.2 mmol/l may make all the difference to the patient's well-being.

Supplementary treatment of side-effects may include beta-blocking agents for lithium-induced tremor, and it seems that intake 'for the occasion', for example 10–20 mg of propranolol 30 minutes before the start of a meeting or the beginning of a party, is more effective and less liable to development of tolerance than continuous intake.

Lithium-induced hypothyroidism may be detected earlier by regular determinations of TSH than by clinical observation alone, and it should not go untreated. Administration of small doses of thyroxine, given concurrently with lithium, effectively lowers TSH, reduces the size of a goitre, and ameliorates the symptoms of hypothyroidism.

Morphological changes have been seen in the kidneys of patients given long-term lithium treatment, but the occurrence of similar changes in patients about to start, but not yet having started lithium treatment has raised doubts about their specificity. The findings nevertheless led to extensive studies of kidney function during lithium treatment, both transverse and longitudinal. They confirmed previous experience that lithium may induce lowering of

the renal concentrating ability by making the distal tubules refractory to the action of the antidiuretic hormone, and this may lead to development of polyuria and polydipsia. The studies have in addition documented that lithium treatment, even when given for many years, does not lead to progressive reduction of the glomerular filtration rate. No patient has died from lithium-induced renal insufficiency and terminal azotaemia.

If patients develop lowered renal concentrating ability, they should be instructed to respect feelings of thirst and to avoid situations involving risk of dehydration, but even a low maximum urine osmolality does not constitute grounds for discontinuing psychiatrically indicated and otherwise well tolerated lithium treatment. Administration of diuretics can lower the urine volume, but the procedure is dangerous; amiloride may be safer than other diuretics.

Weight gain may be a result of hypothyroidism, which should be treated, or it may be secondary to lithium-induced thirst, and the patients should then avoid quenching their thirst with calorie-rich drinks. Sometimes the weight gain has no explanation other than the lithium treatment itself. Restriction of calorie intake and increased exercise are appropriate remedies, but often difficult to implement.

Intoxication

The risk of long-term lithium treatment is development of intoxication which, if severe, may lead to death or to long-lasting cerebellar damage. It is important to note, however, that lithium intoxication does not occur capriciously and does not develop suddenly.

Some patients consume a large lithium dose by mistake or because they want to kill themselves. We should perhaps impress on our patients that lithium is neither an effective nor a pleasant self-destructive agent. More often, lithium intoxication is gradual and caused by a too low elimination rate. Usually, the renal lithium clearance is one-quarter of the creatinine clearance, and it falls when the glomerular filtration rate falls, for example as a consequence of kidney disease. However, occasionally the lithium clearance falls disproportionately; under circumstances of negative sodium and water balance. Risk situations during lithium treatment accordingly include physical disease with fever, heavy sweating, vomiting and diarrhoea, low-salt diet, drastic slimming, and treatment with diuretics. Fluid should be administered parenterally to patients in lithium treatment if they vomit massively, if they are uncon-

scious for many hours, and during the night before major operations with narcosis, when oral fluid intake is prohibited.

There are signs and symptoms which herald incipient poisoning, and if any of them becomes prominent, serum lithium should be checked to ascertain the condition. They include dullness, sleepiness, difficulty concentrating, muscle weakness or muscle twitches, unsteady gait, coarse hand tremor, indistinct speech, nausea, and diarrhoea,

Interactions

Interaction of lithium with particular situations is usually more important than interaction with other drugs. Lithium is excreted exclusively through the kidneys, and the lithium clearance is under most circumstances a constant fraction of the creatinine clearance. There is accordingly danger associated with circumstances which lead to a fall of the glomerular filtration rate, for example kidney disease or cardiac failure. There is also risk in situations where the renal lithium clearance falls independently of or more than the filtration rate, for example during dehydration or as a result of a negative sodium balance. Doctors should be aware of the special precautions and safety measures outlined above.

Adverse interaction with other drugs may be mediated through interference with renal lithium elimination. Diuretics are an important example: they are often sold as over-the-counter drugs and consumed without medical control, and several cases of lithium intoxication have ensued because the doctor failed to give an appropriate warning. Interaction with non-steroid antirheumatics such as indomethacin and phenylbutazone has also been seen, and although the underlying mechanism is not clear, interference with renal elimination is probably involved.

Lithium is often administered together with antidepressants or neuroleptics or both, and in most cases this does not present problems. Combination with antidepressants may sometimes lead to aggravation of hand tremor, and combination with large doses of neuroleptics, particularly haloperidol, has occasionally given rise to confusion and intoxication-like states. Combination with low doses of neuroleptics, e.g. haloperidol in doses lower than 20 mg/day, does not seem to present problems.

There does not seem to be interaction between lithium and anxiolytics, hypnotics, analgesics, vitamins, or contraceptive agents. Nor does lithium treatment interact with drugs against epilepsy, thrombosis, or diabetes. Lithium does not ordinarily change the effects of alcohol.

Contraindications

Since constant kidney function is a prerequisite for safe lithium treatment, lithium is contraindicated in patients who suffer from glomerulonephritis, severe pyelonephritis, or cardiac insufficiency. The glomerular filtration rate falls with advancing years, but as this is a gradual process that does not involve fluctuations, age as such is no contraindication against lithium: elderly persons merely require lower doses to achieve the same serum lithium levels. Patients undergoing maintenance haemodialysis have been given lithium safely, in some cases by adding lithium to the dialysis fluid.

Surgeons would be wise to discontinue lithium treatment some days before major operations which may interfere with fluid and electrolyte balance, but it is important that lithium administration is resumed when kidney function and electrolyte balance have become normal again.

Previous liver disease does not constitute a contraindication against lithium. Heart disease does not prevent psychiatrically indicated lithium treatment, but consultation with a cardiologist may be prudent. Lithium treatment should be discontinued or the dosage reduced temporarily in patients suffering from somatic illness with fever, as well as in patients on a low salt diet, on a strict slimming regimen, or starting treatment with diuretics.

A moderate rise in the frequency of congenital malformations has been seen among children born of women given lithium during the pregnancy. As a general rule, it must be considered advisable that fertile women use contraceptive measures during lithium treatment, that lithium is stopped by women who plan to become pregnant, and that it is discontinued as soon as an unplanned pregnancy has been discovered. The individual patient's situation must be considered, however, and sometimes the doctor and prospective parents may decide to continue lithium treatment in spite of pregnancy when previous discontinuations have led to rapid development of severe manic or depressive relapses.

Alternatives to lithium

Not all manic-depressive patients respond to lithium, and some do not tolerate it. A search for prophylactic alternatives is therefore indicated.

It is now well established that continuation treatment with antidepressant drugs prevents depressive recurrences significantly better than placebo. A prophylactic effect against manic relapses has, however, not been demonstrated, and there is evidence from systematic studies, confirming clinical impressions,

that long-term treatment with antidepressants may precipitate manias. Some studies have shown lithium to prevent depressive recurrences more effectively than antidepressants; other studies have shown the opposite. In either case, patients with a unipolar disease course have an option; if one kind of treatment does not work or is not tolerated, then the other can be tried with a fair chance of success.

Until recently, bipolar patients have been in a different position. Long-term administration of neuroleptics may prevent or ameliorate manic recurrences, but does not prevent depressions. Promising results have, however, been obtained with the antiepileptic drugs carbamazepine and valproic acid. The studies have mostly been carried out on patients who were refractory to lithium. It has not yet been clarified whether these drugs are most effective alone or in combination with lithium, nor whether there are circumstances when one of them should be the first choice prophylactic agent.

Many other drugs or procedures have been suggested as prophylactic alternatives to lithium. They include repeated electroconvulsions, sleep deprivation, benzodiazepines, L-tryptophan, L-hydroxytryptophan, rubidium, beta-blockers, and L-thyroxine. Prophylactic action better than that of placebo has not been unequivocally demonstrated for any of them.

Patient-physician co-operation

If long-term maintenance treatment is to give satisfactory results, it must be based on close and confident co-operation between doctor, patient, and family. This is important with manic-depressive illness, a disease that profoundly affects personality and interpersonal relations, and it is essential with lithium, often perceived as a drug with effects directly on the self. Many problems arise along the treatment course which may tempt the patients to interrupt therapy or to be noncompliant.

The very efficacy of lithium prophylaxis may be a reason why patients tend to discontinue treatment. When there have been no relapses for a long time, the patients think that treatment is no longer necessary and stop it, often with dire results. Doctors should specifically warn against the risk of relapse.

Patients with a bipolar disease course may, during effective lithium prophylaxis, miss the productivity, surplus energy, and social ease of the light manias, and for this reason stop the treatment. It is not always the elation that is missed; the family may blame the patient for lack of depth when he or she no longer has depressive episodes of sadness and humility.

Patients may discontinue lithium because it does not work, or does not work as well as they had expected. They may be disappointed because the treatment did not solve all personal and marital problems. There may be factors in the patient's life which tend to maintain the disease pattern and the patient role. The patient may not have taken the tablets regularly, or may have been treated for an insufficient time. Patient and doctor must together try to analyse reasons for incomplete response and discontent.

There are patients who have difficulty accepting that they must be in long-term drug treatment. Sometimes pronounced anti-drug attitudes prevail among family and friends, who urge the patient not to start

treatment or to discontinue it against medical advice. Psychiatrists should spend ample time correcting such misconceptions through the provision of full and sober information about the illness and the treatment.

Careful information and instruction are cornerstones in successful long-term lithium treatment. The patients should be given information in written form as well as verbally, and the doctors must be prepared to discuss problems as they arise. Books written specifically for patients and relatives are available in several languages; they may serve as a basis for discussions between doctor, patient, and family.

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Lecture

Some Relationships Between Psychiatry and the Social Sciences*

D. MECHANIC

Beyond assuring biological survival, every society must have structures that nurture the young, prepare them for social roles and responsibilities, and that successfully integrate them with a reasonable level of motivation into ongoing patterns of activity. Societies must also have institutions that reinforce a sense of personal commitment to everyday affairs: the lack of a stake in social processes contributes to personal demoralisation and deviant behaviour. Deviance may also occur because of biological vulnerability or handicap, because socialisation fails, because demands exceed capacities, or as a result of sub-group identification being in conflict with dominant values. Psychiatric disorders are a sub-set of deviant behaviour.

The incidence and course of most psychiatric disorders are a product of complex interactions between biological propensities and social, cultural, economic, and demographic forces. The human organism is extraordinarily adaptable to variations in geography, climate, culture, and social organisation; most people, whatever the environment, adapt without mental illness. Some populations undergo horrendous conditions of deprivation and adversity, at times resulting in extraordinary rates of impoverishment, hunger, physical abuse, and mortality; the remarkable fact is the resilience of the human organism, and its ability to persevere despite massive assault. Persistently high levels of stress can push most individuals to their psychological breaking points, but even severe limitations of physical security and assaults on psychological well-being have differential effects on those subjected to these conditions, depending on biological vulnerabilities, coping capacities, supportive relationships, and

the meanings which individuals attribute to their situations.

The social sciences are broad in their concerns, examining social structures and change, as well as developmental processes over the life span. Their concern with the determinants of adaptation and how it is shaped by social institutions such as the family, economic arrangements, and social relationships are intimately linked to basic concerns of psychiatry. How societies structure the learning and decision-making necessary for successful adjustment, and how they programme everyday life in the family, at school, at work, and in many other interactional contexts, influences the psychological skills, predispositions, and personal inclinations that are linked to social functioning, psychological well-being, physical health, and mortality. Societies generate different rates and patterns of pathology, depending on demographic, economic, and psychosocial influences that interact with the biological potentials and limitations of human organisms.

I propose to discuss some general perspectives that encompass much effort in the social sciences which is relevant to psychiatry. These include: the concept of the life course; the influence of social stratification; stress and coping processes; social selection; and illness behaviour.

Social scientists in a variety of disciplines have adopted a life-course perspective which examines individuals and the important units of which they are part (such as the family) within a longitudinal frame of reference (Elder, 1985). The ageing of both individuals and these social units has a unique history, and this reminds us of the extraordinary differences in formative influences experienced by varying generations (Ryder, 1965). This perspective also takes account of the abundant evidence that an individual's life-course, and experience at any point, is dependent on earlier influences and choices that affect the range of options at subsequent points. Choices about schooling, job, marriage, childbear-

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ing, and their timing, establish the conditions for future transitions (Brown, 1986). Thus, while the life-course perspective teaches us that there are many ways to age, it also illustrates the inter-connection of events and how life chances are narrowed by particular experiences and choices.

The study of birth cohort differences has been a major analytical advance in social science: each cohort has varying opportunities, depending on demographic factors and the unique historical conditions it experiences, as it progresses through the life cycle. Even the size of a particular birth cohort (Easterlin, 1980), relative to others, in interaction with economic conditions and values, can either provide opportunities or set limits on life chances. If jobs are few, a large cohort of entrants to the job market relative to retirees can affect the fulfilment of aspirations and economic and occupational success. Similarly, the size of cohorts, in combination with cultural conventions for men to marry women younger than themselves, may restrict marital opportunities; when this is combined with existing patterns of differential mortality by sex, it becomes inevitable that many women in Western societies will have long periods as widows.

In psychiatry, as in medicine more generally, age is an important marker because of the different times of onset of varying illnesses, and the age-structure therefore in part determines the burden of illness. With improvements in longevity and the reduction of fertility, many of the world's populations are significantly ageing. In Western industrial societies, these demographic transformations, resulting in proportionate increases of elderly populations and increasingly of the oldest sub-groups, substantially affect the prevalence of Alzheimer's disease, other dementias, and chronic disability (Gruenberg, 1977). These patterns of frailty put extraordinary burdens on families whose capacities for caring have already been weakened by changing household structure, divorce, and women's increased participation in the labour force. Mental confusion, and the behaviours associated with it, are major precipitants of institutional admission, thus putting large economic strain on health and social welfare budgets in many nations.

The size of the large cohorts born after World War II in the United States and other Western countries has provoked much analysis of their impact on future eligibility for social welfare benefits. From a psychiatric perspective, these large birth cohorts have now reached ages of greater risk for major psychiatric disorders. If risk factors remain the same or are even somewhat reduced, the larger populations at risk still result in a greater burden of mental illness. Thus,

these differences in size of birth cohorts become extraordinarily important for psychiatric planning.

There are further implications from a life-course perspective. The behavioural profiles of varying cohorts of psychiatric patients reflect the social ideologies and treatment patterns that prevailed during their formative development. Psychiatrists in Western countries increasingly complain of the new young chronic patients who often are uncooperative, disruptive, and intractable as patients. Many of these younger patients grew up in a cultural context characterised by deinstitutionalisation, a strong civil liberties orientation, and ideologies denying the validity of mental illness or psychiatric knowledge. Such patients have different illness behaviour from earlier cohorts, and have had varying experiences with mental health services. Their greater intractability and possible alternative approaches to their care are best understood in the context of attitudes and values that are shaped culturally during periods of formative development.

The composition of the population of psychiatric patients at any point in time depends on the experiences of different cohorts, as well as on the organisation of treatment and social welfare. The growth of the hospitalised mentally ill in Western countries in the first half of this century was due in part to increasing longevity and the growing number of elderly patients with dementias, to the accumulation of patients in hospitals with tertiary syphilis, and the lack of custodial alternatives (Grob, 1983). In the United States, deinstitutionalisation did not gain momentum until 1966—more than ten years after the widespread introduction of neuroleptic drugs: the accelerated trend to community care followed important social welfare legislation that paid for alternative institutional care and community subsistence (Gronfein, 1985). As a consequence, elderly patients with dementias were relocated to nursing homes, while many younger patients returned to the community, subsisting on their disability benefits.

The life-course perspective has provided a deeper understanding of the continuity of experience, and of the differences between chronological age and social and biological age. Even birth cohorts distanced by as little as five years can have different biological outcomes at a given age (Svanborg, 1986). By identifying risk factors early, and by changing habits and social practices, we have the ability to modify outcomes throughout the life-span. Most important from the conceptual point of view, however, is that we have learned to differentiate age from disease, despite their statistical association, and increasingly find that incapacities typically attributed to ageing are the consequence of disease and not age. We

are also learning better how to maintain the capacities of the elderly, including memory and motor capacities, by teaching coping skills and by training members of their supportive networks to help reinforce behaviours which are consistent with enhanced functioning.

Our achievements, thus far, have been more in sharpening our questions, improving our research instruments, and developing more powerful analytical strategies, than in identifying successfully the complex pathways between biology, society and culture, and states of mind. We now deal more typically in probability and risk, and less in causal certainty, but the precise mechanisms that operate among these important realms of experience remain to be elucidated. In recent years, there has been much work relating social and psychological events to physical end-points such as cardiac functioning and immune response; this suggests that we are getting closer to truly developing a biopsychosocial model (Mechanic, 1986a).

The current research literatures on such issues as stressful life events, appraisal and coping processes, social support, and the sense of personal efficacy and control represent new ways of confronting long-standing issues. The study of how individuals relate to one another, to social groups, and to systems of symbolic meaning, and the implications of these relationships for pathology has been an important focus of study since Durkheim's classic work on suicide in 1897 (Durkheim, 1951). The study of social networks and social supports—a vigorous area of research activity—continues to examine issues examined by Durkheim (Cohen & Syme, 1985). Even in the most extreme environments, persons often increase their adaptability by developing social connections that provide acceptable meanings, information and assistance, and support and protection. These informal organisations, at times taking elaborate forms, have been described in concentration camps, prisons, custodial mental hospitals, refugee camps, ghettos, and areas experiencing disaster (Mechanic, 1974). Social networks and processes of social support have been studied in almost every conceivable situation, and the literature provides much evidence that deficiencies in networks are related to a lack of social well-being, psychological distress, the occurrence of illness, and even rates of mortality (Cohen & Syme, 1985). But the evidence also suggests that social connections are part of a larger, more complicated constellation of influences, and increasing attention focuses on processes of stress and coping.

Each of the relevant component areas of the stress-coping paradigm (the measurement of stress, the

description of social networks, and depiction of the coping process) is characterised by vigorous debates about assumptions, conceptualisation, and methodology, but these debates have significantly sharpened thinking. In the measurement of stress, divergent views abound on the emphasis to be given respectively to positive and negative life events, subjective and objective measures, and major life events as compared with daily irritations. There also has been controversy as to the methodology of measuring events (whether by respondent report or independent evaluations), the significance of precisely timing the event, and the need to distinguish carefully between events totally outside the individual's control, as compared with those to which the person could have contributed such as divorce, loss of job, or economic difficulties (Brown & Harris, 1978; Dohrenwend & Dohrenwend, 1981; Lazarus & Folkman, 1984). The value of focussing on independent events is clear, but relatively few stressors are completely independent of a person's past history or behaviour.

The concept of social support has been subjected to similar types of debates. While the evidence linking measures of social support to health outcomes has been substantial, neither the measurement of the concept nor the results have been fully consistent. The concept may refer to the extent and structure of social networks, the availability of intimate others, social contacts, voluntary community participation, and similar phenomena. Theories underlying alternative approaches to measurement have not always been clear, and the specific causal mechanisms intervening between support and outcomes are not well developed, although specification of alternative statistical models has advanced (Wheaton, 1985). Studies of support have been relatively one-sided, with little attention having been paid to the constraints, responsibilities, and stresses often associated with kinship ties and other close interpersonal relations.

Decisions about measurement often involve assumptions that close the opportunities to examine particular theoretical ideas. Life event scales that failed to differentiate clearly between positive and negative life changes made it impossible in many studies to test the assumption that life changes, independent of positive or negative features, contributed to morbidity. Existing evidence suggests that loss events relate to depressive illness, but that a wider range of events may be relevant to triggering schizophrenic episodes. Measurement approaches must allow testing of the underlying assumptions that motivate investigation in the first place. Similarly, the counting or scoring of life change events, in examining direct relationships between life change

and health outcomes, ignores much developmental research which suggests that growth and competence are attained through mastering challenging life events. Within this context, stress describes a relationship between a situation and an individual's resources to deal with it, and not a specific event (Lazarus & Folkman, 1984; Mechanic, 1978). The most typical measurement models are not always well constructed to capture the richness of our ideas.

The examination of people's sense of personal control, and the exercise of 'mastery', is a large and promising research area; this has immediate practical implications because it is possible to intervene in many social contexts to enhance people's control over their immediate environments (Rodin, 1986). It is also consistent with developments in clinical psychology and cognitive therapy which relate to helplessness and depression. In some contexts studied, such as nursing homes, small changes in personal control have an impressive effect on psychological response, health, and even mortality (Rodin & Langer, 1977), but questions about this remain unanswered. It is not apparent to what degree these effects operate within the normal range of human control, compared with situations of deprivation. Nor is it evident how enhanced control interacts with personality, attribution styles, cultural values, age, and other variables. The area remains an important one for continuing research and theoretical development.

The study of social stratification and psychiatric disorder has had a central place in sociology and psychiatric epidemiology for several decades, and encompasses a range of theoretical perspectives and orientations. These vary from Marxian views on stratification and alienation to more pragmatic concepts that identify strata by status, income, ethnicity, and gender, which are presumed to differ in relative power and resources (Dohrenwend & Dohrenwend, 1969). Stratification is used as a basis to study the differential incidence and prevalence of disorder, pathways into treatment, types of treatment received, retention in treatment and referral, and outcomes (Mechanic, 1978).

Lower social class continues as one of the most influential predictors of the prevalence of a wide variety of psychiatric disorders, including psychotic illness, substance abuse, anti-social behaviour, and high levels of psychological distress. Social class further influences such factors as access to care, pathways into treatment, types of intervention, and retention in treatment. Class, typically measured by such indicators as income, education, and occupational status, encompasses wide variation in living conditions, power relationships, social re-

sources, child-rearing practices, attitudes and values, social stresses, and other important life processes; these relationships with social class may vary by culture and nationality, as well as over time. In community studies, measures of psychological distress have been found to be inversely and independently associated with education, income, and occupational status. The relative influences of these predictors vary by sex and labour force participation, indicating complex interactions (Kessler, 1982). In short, stratification differences reflect a rich microcosm, in which the interplay between personal vulnerabilities, social adversities, psychological resources, social supports, attitudes and values, and coping processes are played out.

In the study of gender differences, most attention has been focused on the higher risks among women for depression and psychophysiological distress, with relatively less given to the excess prevalence of antisocial behaviour, personality disorder, and substance abuse among men. It is clear that gender differences depend substantially on the disorder under consideration, and efforts to identify predictors of psychiatric disorder in the aggregate open up issues for which we have no conceptual solution. Some argue that the acting-out behaviours which are characteristic of men counterbalance the female excess in depression and related symptoms, but we have no adequate specification of mechanisms to explain the processes through which comparable tensions may lead to alternative patterns of expression of distress. Current interest in vocabularies of distress and processes of somatisation across cultures provide a better grasp of how misery is managed within the limits of cultural constraints (Kleinman, 1986); but given our limited knowledge of mechanisms, research tends to focus on gender differences as they affect specific disorders or types of expression.

All human societies undergo continuing processes of sorting and resorting of their populations, influenced by biological differences, social norms, and individual inclinations and preferences. Some important sub-areas concerned with such selection include the study of assortative mating, geographical migration, and educational and occupational achievement. The study of selection gives attention to the characteristics of individuals and groups that make them different, and to the way they interact with others, exchanging information about their social characteristics, skills and limitations, and personal qualities. Efforts are also made to understand the underlying opportunity structures that may provide more or less options, and that facilitate or limit certain types of choices and outcomes.

Many scientists find social selection an irritation; because it is so influential, no hypothesis is credible unless selection is excluded as a prior explanation. Thus, in examining the effects of organisational environments, therapeutic interventions, educational efforts, etc. selection is the first hypothesis that must be investigated. These effects are widely believed to be so persistent that only randomised studies can credibly deal with them. The less obvious point is that understanding selection itself contributes importantly to psychiatric planning.

Social selection, for example, continues to be a persuasive explanation for the relationship between occupation and the prevalence of schizophrenia; environmental, social, and occupational factors may interact with biological vulnerabilities, affecting its onset to some degree, but the large social class differences are most credibly explained by the disabling consequences of this condition. Similarly, there has been long-standing interest in the uneven geographical distribution of severe mental illness: most evidence points to a migratory process, whereby disabled individuals relocate into areas where they are allowed to function at marginal levels, where tolerance for deviance is more obvious, or where social and welfare support services are more readily available.

Social selection processes also play an important role in bringing patients into treatment and into particular care pathways. Repeated epidemiological studies show that significant proportions of patients with serious psychiatric illness seek no care specifically for these problems, and most psychiatric disorder is treated within the general medical sector, if at all (Shapiro *et al*, 1984; Leaf *et al*, 1985). The processes of selection into care, and into varying sources of help, depend not only on the character and severity of symptoms but also on values, attitudes and knowledge, social influences, and the financial and social accessibility of service providers (Greenley & Mechanic, 1976). It is well established that much psychological disorder is conceptualised and presented in somatic terms, and such patients commonly resist psychiatric diagnosis.

One important sub-area of selection involves the processes of identifying, conceptualising, and responding to symptoms. Such study of illness behaviour is of growing importance in understanding the expression of illness, chronic pain, and social disability. The study of illness behaviour seeks to identify the socio-cultural, psychological, and situational determinants that make people aware of symptoms, the cognitive schemata used to interpret them, and the ways in which the financing and organ-

isation of services facilitate or impede varying kinds of care-seeking (Mechanic, 1982).

A crucial premise in the study of illness behaviour is that illness, as well as illness experience, is shaped by socio-cultural and social-psychological factors, irrespective of their genetic, physiological, or other biological bases. Illness is often used to achieve a variety of social and personal objectives which have little to do with biological systems or the pathogenesis of disease. The boundaries of illness and its definitions are extraordinarily broad, and the illness process can be used to negotiate a range of cultural, social, and personal tensions. Illness behaviour is one of many alternatives for coping with personal and social tensions and conflict (Mechanic, 1986b).

Life in modern societies is characterised by extraordinarily rapid changes in technology and social structures, by almost instantaneous transmission of information across societies and cultures, and by high levels of individualism and persistent questioning of values, life styles, and institutional patterns. Many jobs are perceived as alienating, and such important institutions as the family are undergoing transformation. In American and similar societies, a larger proportion of all births are out of wedlock, having increased in the United States from 5% in 1960 to 18% in 1980. Given recent divorce rates, we can anticipate that more than two-fifths of all children will experience divorce of their parents before reaching the age of 16 (Preston, 1985). In many instances, these children are deprived of significant attention and nurturance from at least one parent, and increasing proportions of children in single-parent families are growing up in poverty. It is difficult to believe that these transformations of family life will not pose significant risks to psychological and social development and to the acquisition of self-esteem, competence, and well-being.

Social organisations embody a wide variety of opportunities and demands that potentially challenge cognitive and behavioural capacities. They may be the source of overwhelming stress and immobilisation, or else stimulate a high level of psychological flexibility and personal competence (Kohn, 1977). The demands implicit in social roles shape psychological and cognitive orientations, complexity of thought processes, and coping abilities; thus, the social structures that emerge have extraordinary mental health implications. The challenge to the social and behavioural sciences is to describe these changing structures and their effects on human potential and individual disorder, and to elucidate the dynamics that link these macro processes with specific important health outcomes.

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Aspects of the Epidemiology of Schizophrenia*

E. HARE

Reasons are given why the hospital diagnosis of schizophrenia may be accurate enough for the proper study of certain aspects of its epidemiology. Studies in the seasonality of admissions and of births in schizophrenic patients have produced consistent results in different countries and at different times. The results are summarized, and causal hypotheses based on them are discussed. The idea that schizophrenia is a relatively recent disease is compatible with these hypotheses.

Is there an epidemiology of schizophrenia?

Given the uncertainty of diagnosis, it is an open question whether there can be a useful epidemiology of schizophrenia—conflicting opinions have recently been expressed in two leading American journals (Freedman, 1984; Shepherd, 1984). The diagnostic problem is most apparent in attempts to determine the incidence rate in a general population. Because it is a comparatively rare condition, a large population is needed to give sufficient cases to establish a firm rate. In practice this means that not every individual at risk can be personally examined by a psychiatrist; this gives rise to questions of how far the use of lay interviewers and structured questionnaires are appropriate (Helzer, *et al*, 1985).

However, there are other uses of epidemiology, for which the problems of method are of a different, less formidable kind. This paper discusses evidence which suggests (a) that worthwhile epidemiological studies of schizophrenia can be based on hospital data, and (b) that the findings of such studies lead to causal hypotheses which have considerable explanatory power and point to further lines of research. It might be argued that such studies are not properly 'epidemiological', but I have presumed the epidemiological method to be the search for associations between rates of illness and levels of environmental factors in different populations.

The *advantage* of hospital data is that in every case the diagnosis has been determined from clinical interview of the patient by a psychiatrist. A series of cases can be restricted either to the first admission or to the most recent admission of persons born within a par-

ticular geographical area and resident there at the time of admission, so that a suitable population base is available. From a large geographical area (i.e. a nation or state) large numbers of cases of schizophrenia will be available for statistical analysis, and there will also be large numbers of cases in other broad diagnostic groups drawn from the same population. It has been argued (Ødegaard, 1952) that almost all schizophrenic patients will be hospitalized at some stage. Even where that is not so, a hospital series of cases will represent the more severe instances of the disorder, and thus the probability of 'correct' diagnosis will be greater. This will be true even when the chances of hospitalization vary in different areas and at different times, and when the proportion of cases diagnosed as schizophrenia on first rather than on subsequent admissions is variable.

Of the *disadvantages* of hospital data, the most obvious is that an unknown number of cases in the population will not be hospitalized; however, for the studies considered in this paper, complete case finding is not an essential requirement. Another disadvantage might be that the larger the geographical area, the greater will be the number of psychiatrists concerned with diagnosis and so the greater the probability they will use differing criteria. It seems likely, however, that in a relatively small state or nation (e.g. the UK or the Scandinavian countries), diagnostic criteria will be similar throughout, and there is evidence to support this (Copeland *et al*, 1971). For long-term studies there is the further problem that diagnostic criteria may undergo secular changes.

If, in sets of hospital cases, the associations found between rates of illness and levels of certain environmental factors are similar in different countries and at different periods of time, then this will tend to confirm the value of the method.

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Consistency of results

There is no such thing as complete diagnostic accuracy. What is needed for epidemiological analysis is a degree of accuracy sufficient to yield consistent results. A straightforward objective test may be useful here. For example, consider the differential diagnosis of schizophrenia from neurosis: the test is that cases diagnosed as schizophrenia should be distinguishable (statistically) from cases diagnosed as neurosis by a criterion which has not been involved in the diagnostic process. It isn't always easy to find such a criterion: for example, schizophrenia and neurosis differ considerably in their age-sex distribution, but it might well be claimed that a psychiatrist takes some account of these factors in reaching his diagnosis. However, no-one would claim that the month of a patient's admission to hospital, or the month of his birth, or the age of his parents at the time of his birth, are likely to have been concerned in the diagnostic process. On each of these criteria there is very strong evidence that cases diagnosed as schizophrenia show significant differences in distribution from those diagnosed as neurosis. Thus schizophrenia passes this test. Moreover, there has been considerable consistency in these findings when they have been replicated at different times and in different countries. The summer peak of admissions to mental hospitals was noted in France 150 years ago (Esquirol, 1838), and has been repeatedly confirmed for schizophrenia. An excess of winter births among mental hospital patients was first reported from Switzerland on patients admitted between the years 1870 and 1910 (Tramer, 1929), and the great majority of subsequent studies have confirmed this for schizophrenia, not only in different countries but in parts of the world where diagnostic criteria are known to have differed somewhat. Moreover, in both these instances the seasonal excess over the yearly average has generally been of the same degree: 8–10% for summer admissions, 5–8% for winter births.

The search for diagnostic precision

There is another reason why we should not shy away from an epidemiology of schizophrenia. Attempts are continually being made to increase the precision of clinical diagnosis – formerly by classification into subtypes, and more recently by laying down specific criteria. Yet it should be borne in mind that the nature of schizophrenia may be such that these attempts can never be more than partially successful. If we consider the hypothesis – a not unreasonable one at present – that schizophrenia is due to a diffuse damage to certain cerebral cells or synapses, and that

the distribution and severity of such damage may depend on many factors, such as constitution, time, or simply chance, then we should not expect to find any clear-cut syndrome. Thus the quest for precision in the clinical diagnosis of schizophrenia may be futile.

We should note too that although the diagnosis of schizophrenia remains somewhat unsatisfactory, the diagnosis of depression is very satisfactory. Most clinicians would agree that unipolar depression shows every gradation from an obvious psychosis to an ordinary bout of low spirits. In such circumstances, the decision of where to draw the line between an illness and a normal variant can only be an arbitrary one. The same would seem to apply to states of anxiety. Mania, on the other hand, can be diagnosed with more confidence, and the fact that cases initially diagnosed as mania may later come to be called schizophrenia – and vice versa – does not matter if these are being considered under the general group of functional psychoses.

Two historical examples

There are historical reasons for thinking that diagnosis need not be precise in order to yield useful ideas about possible environmental causes. For example, although colic is a well-defined symptom, its diagnosis – the nature of the syndrome of which it forms a part – would have been far from precise in England in the eighteenth century. Yet, by straightforward epidemiological methods (based largely on hospital cases), George Baker (1767) was able to show that, in those parts of England where cider was made and where severe colic particularly occurred, there was a close association between the incidence of colic and the presence of lead metal in the cider presses. He then went on to show, by chemical tests, that lead was in fact present in the cider from such presses, and that in places where the cider was free of lead there were few or no cases of the 'Devonshire' colic. He also remarked that, in outbreaks of colic on the continent some decades earlier, the supposition that these were caused by adulteration of the wine with salts of lead (used to diminish the risk of acid fermentation) was at first doubted on the grounds that the diagnosis of colic was very imperfect.

During the nineteenth century the diagnosis of general paralysis was, as Kraepelin (1913) noted, often in doubt – indeed, the apparent increase in its incidence during the last decades of the century was put down to the use of wider criteria for its diagnosis. Yet, as we can see in retrospect, useful epidemiological studies were made. Chapman (1879), by a simple analysis of asylum cases, showed that the ratio of the

rates for single to married patients was about three to one for all cases, but one to one for cases of general paralysis. He concluded that general paralysis was not a constitutional disorder (as most cases of insanity seemed to be), but was probably caused by factors acting after birth. Earlier than this, Esmark & Jessen (1857) found that patients with general paralysis gave a previous history of syphilis more often than other patients. This pointer was not taken seriously at the time, perhaps because it was then outside medical experience for 5–20 years to elapse between infection and the onset of mental sequelae. By the beginning of the twentieth century, however, the evidence of an association between general paralysis and a history of syphilis was strong enough to convince the statistically-minded – including Kraepelin – that general paralysis must be a syphilitic disease (and this was some years before Wassermann's test of the cerebrospinal fluid was discovered). The epidemiological evidence and the conclusion drawn from it was a stimulus to further laboratory research.

Seasonality studies

If we accept that there can be a useful hospital-based epidemiology of the functional psychoses, and in particular of schizophrenia, then the next step is to consider some of the findings and their possible implications.

Among the best-established findings are those of *seasonality*: seasonal variations in incidence which differ from those shown by other mental disorders or by the general population. This phenomenon of seasonality is a good reason for thinking the functional psychoses to be real conditions, and not merely social labels as some writers have maintained. Because seasonality is more simply explained in environmental terms than in genetic ones, it provides an argument for there being an environmental factor in the cause of the psychoses.

Seasonality of suicide

Affective psychosis is implicated in a high proportion of suicides; in many countries the suicide rate has been shown to vary with season, with a peak rate in the spring. The spring peak of suicides was noticed in London in the seventeenth century, and has been widely confirmed in many countries and over long periods of time. In England in recent years suicide has been the only common condition with a highest death rate in the second quarter of the year. Few people would question the reality of this phenomenon, but it is hard to account for. Durkheim (1897) offered a

social explanation – that the increasing life and light in spring-time made those who were already miserable feel increasingly cut off from their fellows – but this is a type of explanation hard to disprove. It seems more reasonable perhaps – particularly in view of the evidence (mentioned below) of seasonality in the onset of the functional psychoses – that seasonality of suicide is secondary to seasonality in the incidence of affective psychosis, as Morselli (1881) had earlier proposed.

Seasonality of onset

The date of onset of a functional psychosis is often hard to determine, but the date of a patient's admission to hospital may reasonably be taken as an index of onset. The seasonal variations in admission rate to psychiatric hospitals, with the maximum in early summer, is a well-known phenomenon (Abe, 1963) but one to which little attention has been paid. Pinel mentioned it in his *Traité* of 1801 and Esquirol (1838) demonstrated it statistically at the Sal Pêtrière Hospital in Paris for the years 1806–14 (Fig. 1): In

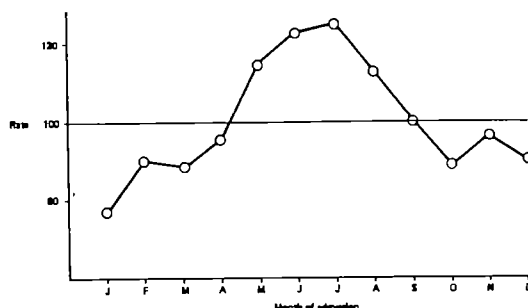


FIG 1 Monthly admission rates to the Sal Pêtrière Hospital, Paris, 1806–1814. There were 2499 admissions. The rates given are the average daily rate for the month expressed as a percentage of the average daily rate for the year (Data from Esquirol, 1838, reproduced from Hare, 1980)

England, monthly admissions to all asylums were tabulated by the Lunacy Commissioners for the years 1895–97; they showed a summer peak for cases both of mania and of melancholia (Fig 2). Recent data for England and Wales, based on the statistics of the Mental Health Enquiry, confirm this pattern and show it to hold both for schizophrenia and for mania (Hare & Walter, 1978). The seasonal pattern is less marked for depressive illness (possibly because of the diagnostic difficulty in separating psychotic from neurotic depression), and hardly appears for neurosis or personality disorder. No one has seriously claimed to explain seasonal variations in admission

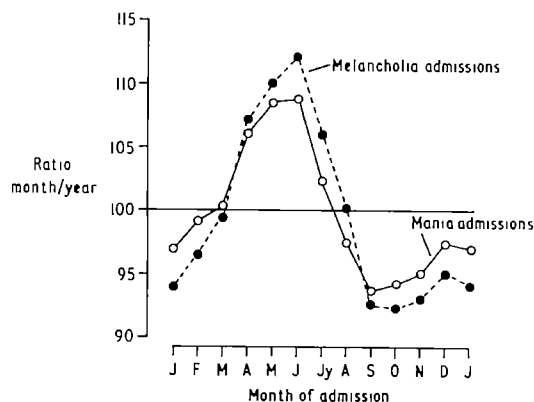


FIG. 2 Month of admission for direct admissions to asylums in England and Wales, 1895-1897. There were 26 538 admissions for mania and 15 734 for melancholia. The ratio is the mean daily number/month to the mean daily number/year, expressed as a percentage. Three-monthly running averages were used (Reproduced from Hare, 1983b).

rates for psychosis in social terms—it has occurred in too many different countries and over too long periods of time. The phenomenon would therefore seem to imply a biological factor both in the onset and the recurrences.

Seasonality of onset occurs in many medical conditions—indeed, Hippocrates thought this was the first thing to be considered in studying a disease. The most obvious examples are virus infections, e.g. influenza, poliomyelitis, and rubella, and it is worthy of note that the seasonality of onset in insulin-dependent diabetes has been taken as evidence of a viral factor in the aetiology (Gleason *et al*, 1982). However, there are many non-infectious examples, such as pellagra, accidents in the home, and lead poisoning (the Devonshire colic had a marked seasonal variation, with a peak incidence in late autumn soon after the apple harvest; however, it did not occur *only* in the autumn, and this fact was taken by Baker (1767) to imply that the colic was not merely due to the sourness of the apples but indicated a toxic substance in the cider whose effective concentration gradually diminished with time). Where no cause is apparent, seasonality of onset cannot be taken to imply any particular kind of causal agent, but it does strongly suggest an environmental agent of some kind.

Seasonality of birth

A seasonality of birth has been widely reported for schizophrenic patients (Hare, 1983b; Pulver *et al*, 1983; Watson *et al*, 1984). Moreover, there have been

very few contradictory reports, and almost all those appeared some years ago, before modern methods of data collection became widespread. The main evidence comes from country-wide or state-wide studies in Scandinavia, England and the United States, and these studies all show an over-representation of the births of schizophrenic patients during the winter or spring months (as compared with the general populations). The subject has been reviewed by Bradbury & Miller (1985). For affective disorder the reports have been conflicting, possibly because of the difficulty of distinguishing psychotic from neurotic depressions, but there has been agreement that the season-of-birth effect is not shown by patients with neurosis or personality disorder. Mania is a relatively rare condition, and adequate series of cases are not easy to collect, but data from the Mental Health Survey in England—based on 3500 first admissions during 1970-79—show the seasonal distribution of births in mania to be very similar to that of schizophrenia (Fig. 3).

The clearest instance of a disorder with seasonality of birth is congenital rubella, with its peak in November and December corresponding to maternal infection during the spring peak for adult rubella. Seasonal variation, with a winter peak, has also been a well-recognized phenomenon in stillbirths and neonatal deaths. It is a point of interest that the extent of these seasonal variations has diminished recently, and in several countries has now disappeared (Hare *et al*, 1981).

It may be asked whether a seasonal variation of 8-10% above the yearly average is enough to be

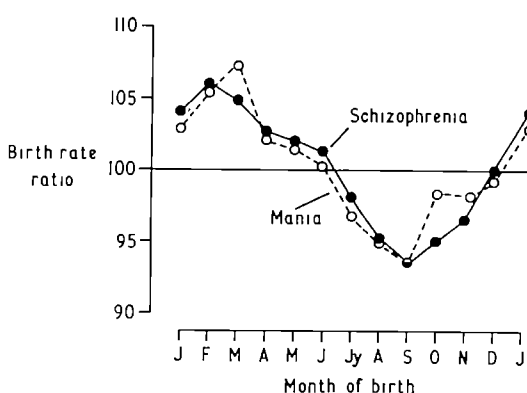


FIG. 3 Month of birth of 22 453 schizophrenic and 3490 manic patients born in England and Wales and first admitted to psychiatric hospitals and units there in 1970-1979. Observed numbers expressed as a percentage of the numbers expected from a 1% sample of the 1971 Census. Three-monthly running averages were used. (Reproduced from Hare, 1983b).

of much aetiological relevance, and whether the seasonality might be wholly or largely attributable to a small subgroup of cases. These questions are discussed in Appendix 1.

The season-of-birth effect in schizophrenia has been dismissed as a technical artefact. This argument has been countered (Watson *et al.*, 1982, Shur & Hare, 1983), however, and does not seem to have received support. It is hard to see how an artefact could produce this effect in schizophrenia and not in neurosis (where the age distribution of hospital admissions has been very similar). We may therefore consider other explanations. Perhaps the most straightforward is that the parents of schizophrenics have an abnormal seasonal pattern of procreation. If that were so, then their other children should show the same seasonality of birth as do their schizophrenic children. In principle, this makes for a simple test; in practice, it has proved hard to get a large enough series, and the evidence so far is conflicting. Relevant here are the findings on parental age (Hare & Moran, 1979; Kinnell, 1983). A number of studies have shown the parents of schizophrenic patients to be older than expected, and the most likely explanation is that constitutional traits in the parents make for a late age at marriage. In so far as this indicates a peculiarity in the parents, the finding of raised parental age adds some weight to the theory of the season-of-birth effect being due to abnormal seasonality of parental procreation. In addition, there is evidence that the normal seasonal variations in birth rate increase with increasing maternal age (Registrar-General, 1949; Rønnike, 1981).

The damage hypothesis

The main alternative explanation of the season-of-birth effect is of some seasonally varying damage to the foetus or new-born child. One difficulty of this hypothesis is the need to postulate that such damage can be causal for a disorder which is manifested only in adult life. There are few recognized examples of this, though late heart failure from congenital malformation of the heart may be one. The examples of general paralysis and the slow virus diseases, however, show that many years may elapse between an infection and the manifestation of disease. Woodrow (1983) reported that a significant proportion of people with congenital rubella develop insulin-dependent diabetes in late childhood, which implies the possibility that they acquired the viral factor during foetal life. Also, it has been suggested on purely epidemiological grounds that in familial Creutzfeldt-Jakob disease the infection (which

causes dementia in late life) occurs in infancy or early childhood (Masters *et al.*, 1981).

If foetal or neonatal damage is causal in the functional psychoses, we might expect to find evidence of brain damage long before the disorder becomes manifest. A number of studies support this idea. The brain abnormalities which have been described in schizophrenia are detectable in the earliest stages of the disease (Weinberger *et al.*, 1983), which suggests the damage was present before onset. Among schizophrenics with abnormal computerised tomography scans, a record of poor premorbid adjustment has been found (Weinberger *et al.*, 1980). In a study of monozygotic twins discordant for schizophrenia, ventricular enlargement was present in both twins, implying that it was not the manifest schizophrenia or its treatment which caused the enlargement (Reveley *et al.*, 1982).

The hypothesis of foetal or neonatal damage is at present, perhaps, the strongest contender to explain the season-of-birth effect in schizophrenia. What might be the cause of such damage? Complications of pregnancy or birth (McNeil & Kaij 1978), a dietary deficiency or a viral infection are the most obvious contenders. Considering the world-wide prevalence of schizophrenia, a dietary factor seems unlikely. Of the other two, a viral hypothesis has the advantage of being able to explain not only the seasonality of births in schizophrenia but also its seasonality of onset and recurrence.

Variations in incidence over time

The concepts of the disorders we now call schizophrenia and affective psychosis were elaborated by Kraepelin in 1899 under the names dementia praecox and manic-depressive psychosis. He distinguished these from among the various types of mental disorder previously grouped under the general term 'insanity'. A historical study of cases of insanity will therefore include all cases of what would now be called schizophrenia and affective psychosis (which together constitute the principal part of the functional psychoses). Certain historical studies of this kind have a special interest when seen in the light of the seasonality findings.

There is a considerable body of evidence that insanity—in the old sense—was rare in non-industrialized countries during the nineteenth century. This might only reflect the limited skill or thoroughness of the observers. However, a recent review of the evidence (Torrey, 1980) suggests it should not simply be dismissed. There is a curious parallel here with general paralysis, which was commonly reported during the nineteenth century to be

rare or absent in non-industrialized countries (Hare, 1959). The same explanations were offered in each case: either the observers failed to look closely enough, or the disorders were indeed absent and were later introduced by contact with 'civilization'. For general paralysis, the argument for its spread by contact with the West became more plausible once the infectious nature of its cause was known.

The possibility that a causal agent of the functional psychoses was introduced into non-industrialized countries during the past hundred years or so raises the question of whether these psychoses have always been present in the West (as has been commonly supposed) or whether there was a time when they were rare or absent. On some theories (e.g. Bleuler, 1972) schizophrenia must always have been with us. Two lines of study suggest that schizophrenia may be a disease of quite recent origin, however.

There appear to be few, if any, convincing descriptions of a schizophrenia-like disorder before the nineteenth century. (For an alternative view see Jeste *et al.*, 1985.) The book *Schizophrenia 1677* by Macalpine & Hunter (1956) springs to mind, but the case described there does not seem to me to justify their title. The lack of a description is the more remarkable in that during the second half of the eighteenth century (and probably deriving from the success of Linnaeus' system of classification), many books were written in England describing the varieties of mental disorder. Haslam, in the first edition of his *Observations on Insanity* (1798), gave no description which we could see as schizophrenia, although in *Observations on Madness* (1809) there are some intimations. The earliest unmistakable description that I have been able to find is that given by Haslam (1810) in *Illustrations of Madness Exhibiting a Singular Case of Insanity*. What he considered to be a 'singular case' we would now think a fairly typical case of paranoid schizophrenia.

This lack or rarity seems surprising in view of the striking features—delusions, auditory hallucinations and thought disorder in physically healthy young adults—which have been characteristic of acute schizophrenia in the twentieth century. The position here is as for a number of other conditions—syphilis, general paralysis, diphtheria, rheumatoid arthritis—whose antiquity has been disputed. In the course of the nineteenth century, however, the problem of insanity was forced increasingly on public attention and gave rise to what has been called 'the asylum era'. In England the prevalence rate of insanity increased by a factor of four to five. It was argued that the increase did not necessarily imply an increase in incidence; it might only reflect the inclusion of milder cases. It was also argued, however, that the statistics

meant what they seemed to imply—that insanity in England was 'fearfully on the increase'. The question was not resolved then, and recent historical studies (Hare, 1983a; Scull, 1979) have still not resolved it. Cooper & Sartorius (1977) considered that there had been no increase in the incidence of insanity but an increase in the number of chronic cases; Scull (1979) considered the increase to be due to increased recognition and to the inclusion of milder cases, and Hare (1983a) considered that there had been a real increase in incidence (and see a reply by Scull, 1984). If the psychosis rate did increase during the nineteenth century, that would support the view of schizophrenia being largely a recent disease, and due to some new environmental factor.

Conclusion

The principal reason for doubting that the diagnosis of schizophrenia is sufficiently accurate to allow a useful epidemiology is that incidence studies in general populations have been discrepant. The results of seasonality studies, based on hospital cases, have however been notably consistent; it is therefore reasonable to conclude that, for this aspect of epidemiological study, the hospital diagnosis of schizophrenia has been adequate.

Serious uncertainties remain. The seasonality of hospital admissions may not truly reflect the seasonality of onset, for example. The seasonality of births in schizophrenia (and perhaps also in mania) is a phenomenon with few parallels in medicine, so there is an understandable caution in accepting it as real; if it is real, an explanation in terms of parental abnormality has not been excluded. Nevertheless, the seasonality findings are in accord with the hypothesis that there are environmental factors of a physical kind in the cause of schizophrenia, and as Crow (1984) has pointed out, the range of possible factors is limited.

The idea of schizophrenia (or a common type of it) being a recent disease is still only tentative. It is of interest because it provides a scenario which can account for many aspects of the history of schizophrenia—such as its changing nature during the present century, and its 'persistence' in spite of the low fertility of sufferers—and also because a recent origin of schizophrenia would most simply be explained in terms of the same kind of environmental factors which would explain the seasonality findings.

Appendix 1

If a seasonal variation is of cyclic type (as those of schizophrenia appear to be), then a seasonal rate of 8–10% above the yearly average will be balanced by a similar rate below this average. The

maximum rate will therefore be some 20% higher than the minimum rate, a difference which might well be considered to have possible aetiological relevance for so common and serious a disorder as schizophrenia, for which nothing useful is yet known about the cause.

A seasonal variation in incidence rate may be due to a causal factor which acts in all (or most) cases but whose severity varies with the seasons, lead poisoning is an example (Stark *et al.*, 1980). Alternatively, a causal factor may act only (or chiefly) on a subgroup of cases, causing an increased seasonality among these, while the remaining cases show little or no seasonality; patent ductus arteriosus is an example (Rutstein *et al.*, 1952). As the degree of seasonality in such a subgroup (if there is one) cannot be foretold, its demonstration may be almost as difficult as the demonstration of seasonality was in the original group, in the case of schizophrenia, this required large numbers of cases and many replications before the findings came to command a degree of confidence.

Studies of seasonality in subgroups of schizophrenia have not so far shown much consistency. Dalen (1968), among 10 171 patients,

found that those who had been in hospital less than three years showed a more marked seasonality than the rest, though in both groups there was a winter peak. In a later study on a larger series of cases (which included those of the first study) this finding was not confirmed (Dalen, 1975). Pulver *et al.* (1983) found a significant birth seasonality (with a peak from January to April) in 1184 schizophrenic patients aged 17–23 hospitalized for less than six months, but no significant seasonality among 271 hospitalized for longer periods. Shensky & Shur (1982) found a significant difference in the birth seasonality of 377 patients with a family history of mental disorder (with a peak in May) and 598 with no such history (peak in October). Machon *et al.* (1983) found winter births to be more common among 125 high-risk urban born schizophrenic patients than among 48 low-risk non-urban born ones. Watson *et al.* (1984) found a significant excess of winter births in 1829 unmarried schizophrenic patients, but no significant seasonality in 1417 married ones. Nasrallah & McAlley-Whitters (1984) found a significant excess of winter births in 113 females with non-paranoid schizophrenia, but not in 135 with paranoid schizophrenia.

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Childhood Hyperactivity

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Severe degrees of inattentive and restless behaviour in childhood are a risk factor for later psychological disorders. They have many causes, but a pattern of severe and pervasive hyperactivity with poor concentration in the absence of affective or psychotic disorders should be recognised as a hyperkinetic syndrome. The syndrome is often associated with developmental delays in abilities such as language and motor control. Powerful short-term treatments are available, but long-term ways of promoting normal personality development need more research.

Hyperactivity means an enduring style of behaving in a restless, inattentive, and disorganised fashion. These are common complaints in children, and they have many different causes. When the problems are severe, they can handicap a child's learning and relationships with family and peers, and make him or her vulnerable to further psychiatric disability.

There has been a confusing debate about the existence of a psychiatric condition of hyperactivity. It has been argued that hyperactive behaviours are non-specific features of any psychopathology (e.g. Sandberg, 1981; Shaffer, 1980). Other authorities (e.g. Aman, 1984; Wender, 1971) recognise them as signs of a specific disorder. Most diagnostic schemes recognise a distinct category. DSM-III (American Psychiatric Association, 1980) describes a condition of Attention Deficit Disorder that may be with or without hyperactivity. The former (ADHD) is defined by the presence of specified numbers of symptoms of developmentally excessive activity, attention deficit, and impulsiveness, especially at school, and affects about 3% of schoolchildren. ICD-9 (World Health Organisation, 1980) is less precise, but describes a condition of 'hyperkinetic syndrome' that is defined by short attention span and (in younger children) by 'extreme' overactivity not attributable to any other condition (Rutter *et al.*, 1975). In practice, this has led to hyperkinesia being a rare diagnosis in the UK.

Historical sketch

Severe overactivity has been recognised as a symptom of disorder in handicapped children for at least a century (Ireland, 1877). A lecture series by the pioneer of paediatrics, Frederic Still, promoted the wider idea that organic disorder of the brain was a major cause

of behavioural problems in childhood, through the induction of "defects of moral control" (Still, 1902). His description of affected children was strikingly similar to modern definitions of hyperactivity: it included "an abnormal incapacity for sustained attention", restlessness, fidgetiness, violent outbursts, destructiveness, non-compliance, choreiform movements, and minor congenital anomalies. Still's work helped to create an intellectual climate in which hyperkinesia and brain dysfunction became almost synonymous.

The next step in recognition came with the encephalitis pandemics in the wake of the First World War. Schachar (1986) studied contemporary reports, and commented that severe behavioural sequelae of encephalitis were uncommon and diverse. The cases did, however, emphasise that brain dysfunction is a possible cause of overactive and disinhibited behaviour. In this tradition, Kahn & Cohen (1934) described three cases of "organic drivenness"; their paper was influential in arguing that hyperactivity was the central behavioural abnormality, and that it resulted from disorganisation at the level of the brain stem.

The idea of hyperactivity soon became more widely applied. It was accelerated by the chance discovery of amphetamine's powerful action in reducing hyperactive and disruptive behaviour (Bradley, 1937). By 1938, Levin was able to identify more than two hundred restless children, compare them with normally active controls, and conclude that cerebral lesions caused severe restlessness (while milder degrees resulted from upbringing problems). Strauss's writings in the 1940s and 1950s broadened the idea further (Strauss & Lehtinen, 1947), to the point where hyperactivity—in the absence of a family

history of subnormality—became sufficient evidence for a diagnosis of brain damage.

Laufer *et al* (1957) described "hyperkinetic impulse disorder" in children with emotional and conduct problems, most of whom did not have any overt evidence of brain damage. They reported a low threshold for induction of muscle jerks by metrazol and stroboscopic light; amphetamine restored the threshold to normal. The study was intended as a corrective to psychoanalytical views that strongly emphasised parents' contributions to children's problems. It was followed by an explosion of biological and pharmacological research, and by a great increase in the diagnosis of hyperactivity and the prescription of stimulants by American paediatricians.

A task force produced an influential document that regarded hyperactivity as a synonym for 'minimal brain dysfunction', and included an excessively wide range of children's psychological difficulties embracing most problems presented for psychiatric help (Clements, 1966). Hyperactivity, or ADHD, has remained the commonest psychiatric diagnosis for North American children. It has generated much good research, most notably into the cognitive deficits of affected children (reviewed by Douglas, 1983) and the nature of the action of stimulant drugs (reviewed by Cantwell & Carlson, 1978, and Rapoport, 1983).

In sharp contrast, British child psychiatry and paediatrics largely avoided the efflorescence of hyperactivity. Widely quoted papers by Ounsted (1955) and Ingram (1956) described series of severely disturbed children, all of whom were drawn from the authors' patients with overt neurological illnesses. The Isle of Wight surveys (Rutter *et al*, 1970) emphasised the infrequency of hyperkinetic syndrome in a whole population of nine year-olds. The diagnosis is still made sparingly, and around half of those receiving it are intellectually retarded or show signs of neurological illness (Thorley, 1984a). This narrow concept suffers, paradoxically, from vagueness of criteria, and has led to much less research than has stemmed from ADHD.

The attitudes of European nations have generally lain between these extremes, and rates of stimulant prescription vary enormously (World Health Organisation, 1985). Several different concepts of hyperactivity have evolved in different traditions, and taxonomic research is particularly necessary.

For clarity, I shall adopt arbitrary definitions to maintain some separation between concepts. 'Overactivity' will mean simply a quantitative excess of movement, without any necessary implication of inattention or disorganisation. 'Hyperactivity', as above, will refer to an excessively high level of

inattentive and restless behaviour. 'ADHD' will refer to the DSM-III category and also to other, similar diagnoses used by North American, Australasian, and other workers; it is a relatively broad group, not only because there are few exclusion criteria, but also because impulsiveness and school misbehaviour can contribute to the diagnosis. 'Hyperkinetic syndrome' will be taken to mean the narrower style of diagnosis, often applied to severely disturbed children; criteria for a useful definition will be suggested below.

Taxonomy

Statistical studies make it clear that several symptoms of hyperactivity covary. Short attention span, distractibility, restlessness, constant fidgeting, and off-task activity are the most constant of these. They form a separate component in many independent factor-analytic studies of rating scales (e.g. Trites *et al*, 1982; Taylor & Sandberg, 1984). This 'hyperactivity factor' is distinct from one of defiantly or aggressively antisocial conduct. Nevertheless, some behaviours—such as disruptiveness, excitability, or impulsiveness—can appear as a part of either factor, and are probably non-specific.

The emergence of a factor of hyperactivity is only one step on the road to a rational classification. Most of the children referred for help because of their disruptive behaviour show both hyperactivity and antisocial conduct. Most hyperactive children are defiant or aggressive too; most antisocial children are somewhat hyperactive (Cantwell, 1980; Stewart *et al*, 1981; Taylor, 1986b). It is therefore crucial to determine whether the idea of hyperactivity has predictive and discriminative validity: does it predict anything that is different from conduct disorder? Shaffer & Greenhill (1979) demonstrated how resolutely this question had been ignored by investigators, and how little reason there was to base research or clinical practice on a widespread diagnosis such as ADHD. Researchers must therefore identify pure cases of hyperactivity that do not show conduct disorder, and pure cases of conduct disorder for comparison; otherwise, they must separately associate the two dimensions with other clinical features.

Distinction between hyperactivity and conduct disorder

Direct observations of children in playrooms or laboratories have provisionally confirmed that inattentive-restless behaviour (judged from case histories or from research interview techniques) predicts high observed levels of off-task activity, while defiance or aggression does not (Milich *et al*,

1982; Taylor *et al.*, 1986a). Furthermore, the same groups of investigators have found that hyperactivity at home or at school correlates with the presence of developmental delays (such as motor clumsiness and low IQ) rather than with psychosocial factors. Conduct disorder, by contrast, is linked closely to indices of unsatisfactory family life, such as discord between family members, low levels of expressed warmth, and ineffective styles of coping (Loney *et al.*, 1978; Taylor *et al.*, 1986a). A comparison of pure hyperactivity with pure conduct disorder by Stewart *et al.* (1981) also suggested that hyperactive children were symptomatically distinct; on the other hand, the two groups had equally high rates of antisocial relatives (Stewart *et al.*, 1980).

These studies were based on clinical groups; McGee *et al.* (1984) reported an epidemiologically based sample. Small numbers and missing data made conclusions uncertain, but the children with the single problem of conduct disorder had higher performance IQs than those with pervasive hyperactivity solely: socioeconomic status, perinatal history, and family relationships did not distinguish between the groups.

Existing studies of the taxonomic questions are too few to allow sharp and certain definitions. They suggest (but not conclusively) a valid separation of patterns of disorder. It is therefore worth considering the lessons from some less powerful studies, that have compared hyperactive children with other kinds of disorder, but have not included the requirement that the contrast group should be quite free of hyperactive behaviour.

Koriat *et al.* (1985) have described a notable lack of differentiation between children referred to a paediatric psychiatry service in North Carolina who received different DSM-III diagnoses. Those with ADDH alone, ADDH plus other diagnoses, and other psychiatric diagnoses not combined with ADDH, were not even distinct in terms of ratings of symptoms—let alone in demographic factors or psychological test scores. Sandberg *et al.* (1978) also found that different levels of severity of hyperactivity carried few implications for aetiology within a group of boys referred to a psychiatric clinic, when 'hyperactivity' was measured by a parental questionnaire, or a teacher rating scale, or by a single period of direct observation at a clinic. Each of these measures was imperfect; when they were combined, a small group of children with pervasive hyperactivity across all measures could be separated from the others, and proved to have an earlier onset, more signs of motor clumsiness, and poorer performance on a psychometric test. Similarly, Schachar *et al.* (1981) reanalysed the Isle of Wight survey to find that pervasively hyperactive children had poorer performance on a

psychometric test, tended to come from lower social classes, and had a worse outcome than children with other kinds of disorder.

The distinction between pervasive and situational hyperactivity may not be very sharp, and some studies find the two groups to be similar (Firestone & Martin, 1979). The general point, however, is that hyperactivity can be validly distinguished from conduct disorder, but that good measures or severe degrees need to be taken if the separation is to be found.

Status of mixed cases

If the above argument is accepted, so that hyperactivity and conduct disorder are seen as separable patterns that overlap greatly in practice, what is the status of mixed cases? If they were clearly to resemble 'pure' conduct disorder or 'pure' hyperactivity, then their position in classification would be clear.

Stewart *et al.* (1981) made just such a comparison on the basis of clinical features: children with unsocialised aggression and hyperactivity (identified mainly by parental accounts) resembled those with only unsocialised aggression more than those with only hyperactivity. On the other hand, August & Stewart (1982) presented another analysis of a closely similar series of children. When hyperactivity had to have been noted in several situations, there was little difference between the children with this problem only and those who combined it with unsocialised aggression.

McGee *et al.*'s (1984) epidemiological study seemed to show that the mixed group were particularly vulnerable. Their reading was worse than that of either pure group, and their short-term outcome was worse. Such a finding is in keeping with a dimensional approach to classification: children with both problems should be predicted to have the associations of both.

A separate class of hyperkinetic syndrome?

Hyperkinesis needs to be separated not only from conduct disorder but also from emotional disorders and specific disabilities of learning. Applications of cluster analysis to simple parent questionnaire ratings have given conflicting results about the presence of a discrete group of hyperactive children (Taylor *et al.*, 1986b). Some workers find one, some do not. Halo effects and reliance on a single rater are serious problems in this research. A cluster analytical study reported by Taylor *et al.* (1986b) was based on reliable, independent measures from several sources. The most robust feature of clustering procedures was

the emergence of a small group of pervasively hyperactive and inattentive children with few emotional symptoms. This group also showed an early onset, a high rate of delays in cognitive development, and motor clumsiness. In addition, they had a much more marked response to a stimulant drug (methylphenidate).

Definition

Current evidence favours use of a rather restricted diagnostic concept of hyperkinetic syndrome, rather than the ADHD definition. Symptoms of overactivity alone are not sufficient for the diagnosis; it should be based on:

- (a) A pattern of markedly inattentive, restless behaviour (not just antisocial, impulsive, or disruptive acts) that is excessive for the child's age and IQ, and a handicap to development
- (b) presence of this pattern in two or more situations, such as home, school, and clinic
- (c) evidence of inattention, restlessness, or social disinhibition, from direct observation or testing by the diagnostician (i.e. not solely by unconfirmed reports from a child's caretakers)
- (d) absence of childhood autism, other pervasive developmental disorders, or affective disorders (including depression, anxiety states, and mania)
- (e) onset before the age of six years and duration of at least six months.

Such a diagnosis is useful, though not very common (Taylor, 1986a). It should not be missed simply because an affected child has developed antisocial conduct as well.

Hyperactivity in special subject groups

The discussion of classification above has been based on studies of normally intelligent children in the first few years of their schooling. The behaviour problems of preschool children do not yet have a stable classification (Jablensky *et al.*, 1983). In one investigation, pervasive hyperactivity in children at a day nursery predicted psychological disorder later (Campbell *et al.*, 1977); in another, even pervasive hyperactivity was not a predictive variable among nursery children (Cohen & Minde, 1983). The behaviour matters, as will be shown later, but its classification is obscure, and clinicians should be correspondingly slow to diagnose. Overactivity is also common among intellectually retarded children, but its causes are not yet clarified. Affective disorder, autism, and atypical

pervasive developmental disorder are all important causes of overactivity in this group. I have argued elsewhere (Taylor, 1986a) that some individuals should be regarded as showing a distinct hyperkinetic syndrome, but more research is required.

Prevalence

No secure prevalence rate can be given, because of disputes about what constitutes a case. Hyperactivity is continuously distributed in the population, with smaller numbers at successively higher degrees (Taylor, 1986a). No validated cut-off yet exists, so no prevalence estimate has a scientific basis.

The numbers of schoolchildren who are in practice diagnosed vary wildly: a little over 1% in two US surveys (Lambert *et al.*, 1978; Bosco & Robin, 1980), but only 0.1% in a London borough (data from the Camberwell Register)—half of the latter being intellectually retarded. By contrast, the rates derived from questionnaire surveys of teachers' ratings are much the same in the UK as in the USA and New Zealand, and range from 5% to 20% (Taylor & Sandberg, 1984). The low diagnostic rates in the UK are likely to be due to the diagnostic practice rather than the nature of the children (Prendergast *et al.*, unpublished). Chinese populations are often said to be more docile and attentive, but a survey from Beijing, based on a combination of teacher ratings and medical diagnosis of ADHD, gave comparatively high rates (3–7% in different areas) (Shen *et al.*, 1985). Hyperactivity, in some sense, evidently exists in many cultures; it remains to be seen whether it always takes the same form.

The prevalence of ADHD in boys is higher than in girls in the ratio of about 4:1 in American clinics (Ross & Ross, 1982). The ratio was similar for the narrower hyperkinetic syndrome at a London clinic, but fell to 1.6:1 for children with mental retardation (Taylor, 1986a). Girls with the diagnosis of hyperkinetic syndrome, or with the symptom of 'gross overactivity', were more likely than affected boys to show delays in cognitive development and neurological disorders such as epilepsy (Taylor, 1986a). The reason for the greater vulnerability of boys has not yet been explained. Several theories implicate damaged brain function. The greater vulnerability of male brains may well be a part of the explanation, but has not been shown to account for the whole of the enhanced risk. Adults are often more tolerant of hyperactivity in girls than in boys, at least before school age (Battle & Lacey, 1957).

Epidemiological studies in different cultures are needed to cast light on what is central to and

universal about the conditions and what is peripheral and culturally determined.

Aetiology

In spite of much research, the causes of hyperkinesis remain uncertain. Most studies have compared children referred to clinics, because of mixed behaviour problems including hyperactivity, with controls drawn from the normal population. When positive findings appear in such a design, it is hard to know whether they are associated with hyperactivity, with behaviour problems generally, with the learning disorders that are often associated, or with the factors that lead to referral to clinics.

Brain damage

Brain disease with localising neurological signs is uncommon in children with ADDH, and is specifically excluded from most research series. The narrower hyperkinetic syndrome, by contrast, is often diagnosed in children with damaged brains. Table I summarises the findings in those investigations that have used an ICD-9 style of diagnosis and reported the range of diagnoses in various samples of children. Brain damage gives rise to a high rate of virtually all the psychiatric syndromes of childhood, rather than to any pathognomonic syndrome (Rutter *et al*, 1970). Accordingly, the best index for comparing groups is not the numbers with the diagnosis of hyperkinesis, but the rate of hyperkinesis as a percentage of all diagnoses made (Table I).

The relatively high rate in neurologically handicapped groups implies that there may be a specific vulnerability to hyperkinesis in brain-damaged chil-

dren. This conclusion is not secure, for the diagnosis could in part be determined by judgements about neurological status. Explicit and independent criteria for case definition need to be developed. If there is a specific vulnerability, we do not know whether it resides in the brain damage itself or in the generalised intellectual retardation which can result. High rates of structural brain damage were reported by Reid (1980). Jenkins & Stable (1971) matched severely hyperkinetic cases (from a case register of intellectually subnormal people) with subnormal but not hyperkinetic controls: they found higher rates of cerebral palsy and other evidence of structural lesions in the hyperkinetic patients. However, the hyperkinetic patients were also more retarded on a global measure of development. Thorley (1984a), using the case records of severely hyperkinetic children, matched psychiatric controls individually for IQ level: no significant difference in rates of brain damage then appeared.

Severe hyperkinesis may have a specific neuropsychiatric predisposition; this should be investigated further. However, there is no reason to suppose that mild hyperactivity is therefore caused by mild brain dysfunction.

Perinatal trauma to the brain

This is not usually the cause of hyperactive behaviour. Case-control studies of children showing high levels of hyperactivity have usually shown little or no increase in rates of perinatal injury, retrospectively assessed (e.g. Minde *et al*, 1968; Gillberg *et al*, 1983). Cohort studies of children of low birth weight (Neligan *et al*, 1976), or of children with complicated deliveries (Nichols & Chen, 1981), make it clear that

TABLE I
Prevalence of clinically diagnosed hyperkinetic syndrome (ICD-9) as a percentage of children with psychiatric diagnoses

Study	Type of study	Patient group		
		Neurologically normal children: %	Neurological disorders or epilepsy: %	Mental subnormality: %
Rutter <i>et al</i> (1970)	Epidemiological survey	~1	12	26 ¹
Corbett (1979)	Case register	—	—	8
Reid (1980)	Clinic outpatients	—	—	15
Thorley (1984a)	Clinical records	0.9	—	2
Gillberg <i>et al</i> (1986)	Epidemiological survey	—	—	10

1. Severely handicapped children, not attending school.

hyperactivity is not particularly common in the later lives of such children. There is a small increase—as there is for other kinds of behaviour problem—but even this increase is partly attributable to coexisting family problems. Indeed, the Isle of Kauai study (Werner & Smith, 1977) suggested that perinatal damage only increased vulnerability in children with less advantaged backgrounds.

Minimal brain dysfunction

Minimal brain dysfunction has been intensively sought, so far largely in vain. Extensive reviews have concluded that there are few unequivocally positive findings of biological abnormalities in hyperactive children (Rie & Rie, 1980; Rutter, 1983b). When abnormalities have been found, their meaning is usually unclear. 'Soft' neurological signs (i.e. clumsiness and lack of coordination) are commoner in markedly hyperactive children than in other sorts of psychological disorder (Werry *et al.*, 1972; Sandberg *et al.*, 1978; Taylor *et al.*, 1986b). However, they are not yet valid as a sign of cerebral or cerebellar pathology because of uncertainty about what causes them (Taylor, 1983a).

Neurophysiological unresponsiveness

Neurophysiological unresponsiveness to stimuli is another replicated finding. Hyperactive children, by comparison with controls, probably show normal resting levels of autonomic nervous system activity. However, peripheral autonomic responses and central averaged evoked EEG responses both suggest that a new or important stimulus produces a smaller change in hyperactive children (Taylor, 1985a). This lack of reaction may not be specific to hyperactive children: diminished physiological responsiveness also characterises children with learning disorders (e.g. Maxwell *et al.*, 1974) and unsocialised aggression (Delamater & Lahey, 1983). Since these three clinical problems often coexist, which of them (or what other associated problem) is linked to unresponsiveness? We shall only know when further research untangles the various problems.

Attention deficit

Hyperactive children fail to sustain organised attention, and score poorly on psychological tests related to attention (Douglas, 1983). However, test performance can also be upset by many different sorts of psychopathology (Shaffer, 1980), and the impairment has not yet been shown to be specifically an attention problem. Although hyperactive children

behave impulsively, psychological tests of impulsiveness suggest that they respond too slowly, not too quickly (Sergeant, 1981; Sandberg *et al.*, 1978; Firestone & Martin, 1979). Although they behave distractedly, the addition of distracting information does not particularly disrupt test performance (Douglas & Peters, 1979). The deficit is present in both short tests, such as the digit span, and long ones, such as continuous performance tests. It is even manifest in a downwards shift of IQ; indeed, when IQ is allowed for, hyperactivity shows much reduced (but still present) association with tests of concentration. There is as yet no sign of a structural deficit at any one stage of processing information—rather, there is a high-level failure to allocate resources sensibly to the task in hand and to maintain self-control in organising responses.

Animal models

Animal models of hyperactivity emphasise its heterogeneity. Prefrontal damage in monkeys, ventral tegmental lesions in rats, chemical damage to dopamine and noradrenaline synthesis, foetal exposure to lead, neonatal hypoxia, and social isolation can all produce a combination of overactivity and abnormal learning that is reversible by amphetamines (Robbins & Sahakian, 1979).

Genetic inheritance

No really persuasive study has yet been made on the effects of genetic inheritance. Full siblings of hyperactive children are more likely to be affected than are half-siblings (Safer, 1973), and hyperactive children, like antisocial children, have many relatives who themselves show sociopathy or alcoholism (Stewart *et al.*, 1980). However, family interactions could explain these findings as well as genetic inheritance. The biological parents of hyperactive children living with their natural families were more likely to have had behaviour problems than the adoptive parents of adopted children (Cantwell, 1975), but this study could not compare biological with adopted parents of the same children. The adopted-away offspring of psychiatric patients were unusually likely to be hyperkinetic (Cunningham *et al.*, 1975), but a transmission of hyperactivity itself was not demonstrated. Comparisons of dizygotic with monozygotic twins suggested that the latter were more concordant with regard to several temperamental dimensions, including overactivity (Torgersen & Kringle, 1978); twin studies of children with diagnosable conditions have not yet been reported.

Chemical agents

The most controversial aspects of the aetiology of hyperactivity have come from theories implicating exogenous toxins, such as lead and food additives.

Severe lead poisoning causes brain damage, and its sequelae include hyperactivity. Lower levels of exposure can cause hyperactivity in animals, but their significance in children is still debated. High levels within the normal range are associated with small increases in problem behaviour and with lower IQ (Needleman *et al*, 1979; Yule *et al*, 1981). It is still uncertain whether these correlations reflect the damaging effect of lead at low levels of exposure, or an association with other kinds of social adversity (Rutter, 1983a). In any event, lead exposure is not the major cause of hyperactive behaviour.

In spite of the scientific uncertainties, there is a brisk trade in testing hair for lead and other minerals and in selling supposed therapies. The enterprise should not be encouraged: commercial hair measurement is unreliable (Barrett, 1985), and the proprietary treatments are unevaluated.

Food dyes and preservatives were implicated by Feingold (1975), with the suggestion that hyperactive children showed a genetically determined intolerance. Unfortunately, most trials involving elimination of food additives have given rather disappointing results: some children's behaviour improves for a while when a diet is started, but it does not usually worsen again if additives are given in a double-blind placebo-controlled fashion (Mattes & Gittelman, 1981). Occasionally, however, some children do seem to show a genuine improvement in behaviour due to the physical effect of removing additives (Weiss *et al*, 1980). The improvement is not confined to hyperactivity, but extends to 'difficult' behaviour generally. A recent trial involving exclusion of a wide range of different foods from the diet suggested that more children can be helped by a radical exclusion of many allergens; however, the trial was based on a selected sample with a high prevalence of physical allergies (Egger *et al*, 1985). Since some parents become preoccupied with diet to the exclusion of all else, more research is quite pressing.

Psychosocial factors

The psychological environment can determine the extent to which children are attentive (Taylor, 1980). It is therefore logical to seek evidence of psychosocial adversity in the hyperactive. Affected children, ascertained in a population survey of children with minimal brain dysfunction, can be distinguished from normal controls by signs of unsatisfactory family life

(Gillberg *et al*, 1983). Children who have grown up in institutions are particularly likely to be hyperactive in their classrooms, even after they have been adopted into a family home (Tizard & Hodges, 1978; Roy, unpublished). Children known to have been unwanted (because their mothers sought a termination of pregnancy unsuccessfully) are more likely to be excitable, unsociable, and hyperactive when they go to school (Matějček *et al*, 1985).

Studies such as these are vulnerable to the same criticisms that have already been levelled at investigations of physical causes: they show correlations, not causes, and point the clinician only towards the need for comprehensive assessment.

Developmental course

Hyperactivity most commonly presents during the early years of schooling. Retrospective enquiry usually finds an onset before the age of five and often before the age of two.

Infants vary greatly in their activity levels, but the individual differences are not very stable over time. Indeed, one careful study suggested an inversion in the intensity of behaviour between the age of 3-4 days and that of 2½ years (Bell *et al*, 1971): the neonates with highest magnitude, frequency, and speed of behaviours became the two year-olds with least vigour and lowest responsiveness.

In preschool children, overactivity is a common complaint made by parents, but sometimes reflects impaired parental tolerance rather than an abnormality of development. Although (as considered above) no clear hyperkinetic syndrome has been validated for this age-group, nevertheless, the complaint of overactivity should not be ignored: it predicts later antisocial disorder quite strongly (Richman *et al*, 1982).

From the age of about three years, the normal course of development involves a reduction of general level of activity in some settings but not in others (Routh, 1980). The appropriate modulation of activity can therefore be impaired by developmental delay, whatever the cause. The resulting restlessness becomes more of a problem as schooling proceeds and successively greater demands are made on children's powers of attention and self-control.

The continuities between the early years of schooling and outcome in adolescence have been examined by many follow-up studies, mostly of children referred to clinics with ADDH, contrasted with normal controls from the community (see reviews by Weiss, 1983, and Thorley, 1984b). The investigations are limited by design weaknesses (highlighted by

Shaffer & Greenhill, 1979), but taken as a whole, they suggest that the broad group of hyperactive children do not simply grow out of it. In adolescence, they tend to remain somewhat impulsive and inattentive, and to be characterised by high rates of academic failure, antisocial behaviour, and delinquency. An epidemiological study in England also found pervasive hyperactivity to be a strong predictor of persistence of psychiatric disorder between the ages of nine and 14 (Schachar *et al.*, 1981).

There is even some evidence that severe over-activity remains a risk factor for adolescents when the controls themselves show antisocial conduct. Thorley (1984b) identified children with the hyperkinetic syndrome seen at a psychiatric hospital, followed them into adult life, and compared them with matched cases of conduct disorder from the same hospital. Their adolescent outcome was characterised by more episodes of psychiatric treatment, more epileptic fits, more accidental injuries, and more placements in special schools.

In adult life, most children with ADDH do not show diagnosable illness, but the rate of antisocial and impulsive personality disorder is high (Hechtman *et al.*, 1984). It is probably no higher than that of less hyperactive children with conduct disorder (Thorley, 1984b); however, the degree of conduct disorder in childhood and the disturbance of family relationships are better predictors of an adult antisocial adjustment than is hyperactivity itself. It seems likely that pervasive hyperactivity has its major effect on development by increasing the risk that affected individuals will develop complicating problems such as educational failure or antisocial conduct disorder. If they can be protected from such complications, then a gradual improvement in adjustment in adult life can be expected.

Treatment

Multiple factors interact to cause hyperactivity; its course is largely determined by complicating adversity. Diagnosis is therefore not enough to dictate treatment; a wide assessment and a range of interventions are needed.

Drug treatment

Amphetamines and related central nervous system stimulants are the commonest treatment for ADDH in the USA, being given to more than 1 schoolchild in 100 (Bosco & Robin, 1980; Lambert *et al.*, 1978). By contrast, they are so rarely used in the UK that the

first-line drug, methylphenidate, has recently been withdrawn for lack of demand. Dogmatic guidelines are therefore inappropriate.

Scores of double-blind trials have shown that stimulants are more effective than placebo in suppressing hyperactive behaviour for children with ADDH (Taylor, 1985b). These trials are based on relatively short periods, from a few weeks to a few months; the long-term effect is less clear. Some children who have taken amphetamines for long periods are no longer getting any benefit (Charles *et al.*, 1979). Others show worsened symptoms when their drug is removed (Sleator *et al.*, 1974). Even if symptoms are controlled in the long term, it does not follow that long-term development will be improved. Follow-ups of treated and untreated groups leave it uncertain whether drug treatment is of any use over a period of years (Weiss, 1983). Accordingly, long-term treatment should only be embarked on when there is clear evidence of short-term benefit, and good supervision (including regular periods off-drug) can be provided to monitor the continuing need for therapy.

The short-term action of drugs is not paradoxical or unique to hyperkinetic children: the effects are qualitatively similar in hyperactive children, normal children (Rapoport *et al.*, 1978), normal adults (Weiss & Laties, 1962), and conduct-disordered children (Taylor, 1983b). In all these groups, they act as stimulants, not sedatives; they reduce high-frequency and off-task behaviours, and they improve performance on a variety of psychometric tests. The mechanism probably involves catecholamine neurotransmitters (Rapoport & Zametkin, 1986). Though the action is similar in kind in different groups, it varies greatly in degree.

The best and clearest indication for stimulant medication is the uncommon nuclear hyperkinetic syndrome of pervasive and severe hyperactivity and cognitive impairment in the absence of overt emotional disorder, presenting in primary school-children of normal or only mildly retarded intelligence. There are, however, other indications too. Sometimes it is desirable to induce a rapid reduction in restless, disruptive behaviour in order to bring about a change of attitude in the child's caretakers. Sometimes medication is necessary to allow a programme of learning to operate. In these circumstances, stimulants may be given to children with lesser degrees of hyperactivity. Whatever the indication, stimulants need to be planned as an adjunct to educational or psychological help.

Careful monitoring is needed, and the reader is referred to fuller accounts of drugs and their prescription (e.g. Barkley, 1981; Taylor, 1985b).

Dietary treatments

Dietary treatments are very popular with parents, and present a dilemma because of the difficulty of recognising those few children who may be helped. The diets carry the hazards of poor nutrition and the neglect of other treatments. In present knowledge, they should only be prescribed with expert help, but it is reasonable to support parents who have themselves decided on a trial.

Psychological treatments

The techniques of behaviour modification can be applied in many different ways to the treatment of hyperactivity (Yule, 1986). Operant conditioning is of demonstrated value in the short-term reduction of off-task behaviour and increase of constructive activity in the classroom. Although it may sometimes be less potent than stimulant drugs (Gittelman *et al*, 1980), it is also more widely acceptable.

Reward-based schemes of learning can also be used to reduce gross motor activity and fidgetiness (Christensen & Sprague, 1973), but the mere lessening of activity is seldom enough to help children's adjustment. They also need to learn more positive skills of learning and social interaction.

Cognitive-behavioural therapy has promise as a tool for teaching techniques of self-control and problem-solving (Bornstein & Quevillon, 1976). Systematic evaluations have indicated that it can improve performance on laboratory tests, for instance of impulsiveness (Douglas, 1983). Its value in promoting longer-term adjustment and real-life performance is not yet established.

Considerations of developmental course emphasise the importance of helping wider aspects of a child's predicament than hyperactivity alone. Adverse and coercive styles of family interaction can

readily develop, and may indicate behavioural techniques of modifying such interactions, or the training of parents in behavioural skills, or the approaches of conjoint family therapy. Educational failure is so common and important that a liaison with schools, and a consideration of special education needs, should be part of every psychiatric assessment. Many affected children acquire a very negative view of themselves as 'weird', 'rubbish', or 'stupid'; individual sessions therefore have a counselling purpose as well as that of instilling skills. If drug treatment is to be used, it needs full and repeated discussion with child and parents, lest it lead to scapegoating or to a glib alibi for bad behaviour.

Conclusions

Research continues, but provisional conclusions can be made. Severe degrees of inattentive and restless behaviour constitute a major problem for children's development, and are different from (but overlap with) conduct disorder. They probably need more recognition by psychiatrists and teachers in the UK, but may be over-diagnosed in the USA. Delays in motor and cognitive development are often associated, and may well be aetiologically important; however, they are not yet sufficiently specifically described to justify the view that affected children have a structural deficit in the processing of information from the environment. Powerful short-term treatments are available, in the shape of stimulant drugs and behaviour modification, but their long-term effect is inadequately known. Family, school, and peer relationships are usually more important than the core problem in determining eventual adult outcome. Clinical services therefore need to develop a range of treatments for affected children.

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The Prognosis of Depression in Old Age

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The initial features and progress of depressive symptomatology over 42 to 104 months are presented for 100 elderly patients admitted with severe, non-neurotic depressive states; none had experienced a previous hypomanic episode. Sixty per cent either remained well throughout or had further episodes followed by full recovery; only 7% suffered continuous depressive symptoms. Of potential prognostic factors, only male sex and poor physical health, both at presentation and developing subsequently, were associated with poorer outcomes. The view is supported that treatment with well established methods achieves worthwhile and sustained improvement for most patients.

The recent growth of psychogeriatrics has occurred mainly in relation to the major health problems posed by the dementing illnesses of late life (Arie, 1977), but while these are common, and require substantial resources for skilled assessment, management, and research, depressive states of various kinds are even more common (Kay *et al*, 1964). They produce at least as much suffering, and can be lethal, yet are potentially responsive to available treatments to a degree not yet possible with the progressive dementias. Thus, an appropriate and effective response to depressive states is a challenge to old-age psychiatric services, in that skilled intervention might be expected to produce more dramatic results than is possible with other conditions.

Although even in the elderly, depressive symptoms are more often encountered than serious cognitive decline, recent epidemiological studies (Henderson & Kay, 1984; Post & Shulman, 1985) have demonstrated a point-prevalence of only 2–3% for primary clinical depression. An additional 11–12% of the elderly in the community have been found significantly depressed, but only to a minor degree and without biological symptoms; in about half of these 'senile dysphorics', the mood disorder appeared to have been precipitated by chronic physical illness or deterioration. However, nothing is yet known about response to treatment of this important group of elderly depressives, who only rarely come to psychiatric notice. Although the prevalence of clinical depression is probably no higher than during middle age, major depression of the aged presents problems second only to those of dementia.

The clinical features of 'melancholia' were described by Lewis (1934) and Anderson (1936), while Roth (1955) reviewed the outcome for elderly patients admitted to mental hospital in the 1950s.

Post (1962, 1972) studied two cohorts treated by him as in-patients: the first in the 1950s, when electroconvulsive treatment (ECT) and leucotomy were the only effective physical treatments available, and the second in the 1960s, when tricyclic antidepressants had been added. These reports established that while permanent recovery is unusual in this group of severely ill patients, it is much more common than continuous morbidity, and that the majority of patients experience relapses that are responsive to treatment. Rather than being a gloomy prospect, this is one which encourages services to remain available for—and if possible in touch with—their elderly depressives, and prepared to identify and treat relapses when they occur.

Murphy (1983) described a cohort referred to psychogeriatric services, which chose to admit only half these patients during the period of study. Some of her findings were alarming, notably that the death rate for men in the cohort was three times that expected, and for women twice that expected. The overall outcome was summed up as 'poor' for 48% of cases, with death or dementia supervening in a further 17%, leaving only 35% with a 'good' outcome. The prognosis for those with depressive delusions at presentation was very poor indeed: only one in ten recovered by the end of the study year. An analysis of this work concluded that for the elderly depressed, "no matter what is done, a third get better, a third stay the same, and a third get worse" (Millard, 1983).

This is a very worrying situation, which might encourage pessimism and nihilism in the face of the treatable condition of depression in old age. Since it is not a conclusion which fits with clinical experience (Godber, 1983), we have examined the characteristics and outcome of patients referred to a

psychogeriatric service that manages most of its patients without recourse to admission (Jolley *et al.*, 1982), but which chose to admit these patients because they were very ill with depression.

Method

Entry criteria were: (a) age over 65 years; (b) in-patient status; (c) Feighner's (1972) criteria for depressive illness met from scrutiny of case notes; and (d) first admission under the psychogeriatric service—previous admission elsewhere might have occurred, and two patients included had been transferred from another psychiatrist. Hence, in keeping with Post's studies, cases with recent as well as remote previous depressive episodes were accepted, but those cases free from depression since the age of 60, comparable to Murphy's (1983) patients, were identified. The reliability and breadth of material in the casenotes of in-patients was likely to be greater than for patients not admitted.

Cases were recruited from the admission register from January 1976 to April 1981; the service received 2150 referrals, of whom 25% (540) were described as depressed at initial assessment. Only 511 patients had been admitted, and in 152 (30%) of these, depressed mood was considered to be a major element in the illness. There was exclusion of 52 cases for the following reasons: age under 65 years (5); organic psychosyndrome (12); secondary depression as defined by Feighner (8); neurotic depression (5) (identified where either depression was in the setting of long-standing complaints or, in two recent cases, was associated with bereavement and the Feighner criteria could not be met for even 'probable' depression); previous history of mania (6); previous history of schizophrenia or paranoid state (4); alcohol abuse (6); personality disorder (2); case notes lost (3); and diagnosis unclear (1). The 100 subjects therefore comprised roughly one in five of those described as depressed at referral.

Phase 1: index admission. Clinical and social data were recorded on a standard form. Severity of initial depressive illness was assessed by applying the Hamilton Rating Scale for Depression (HRSD) retrospectively, using all the information recorded within the first few days of admission; the clinical description of each HRSD variable by Hedlund & Vieweg (1979) was relevant here. Physical health was scored for eight body systems, according to whether there was no problem, an inactive problem (e.g. an old myocardial infarct), or an active problem (e.g. effort angina). Those discharged were classified as 'well' (free from affective symptoms), 'improved' (incomplete symptomatic recovery), or showing 'little or no change', in a manner analogous to Post (1962).

Phase 2: follow-up at one year. Clinical status one year from admission was assessed from the case notes for all but one patient, who had moved out of the area after discharge and was untraceable. Outcome was classified after Murphy (1983): (a) 'well': those recovered at one year who were

either asymptomatic or had minor symptoms insufficient to meet the Feighner criteria for depressive illness; (b) 'relapsed': those who after recovery from the index illness experienced a return of symptoms which fulfilled the Feighner criteria; (c) 'continuously ill': those who retained their depressive symptoms throughout the year, with little variation.

Phase 3: follow-up to 1984. By the autumn of 1984, 31 patients remained within the psychogeriatric service. A further 25 were dead but had been followed-up by the service from the index admission until death. Finally, 44 were no longer within the service, although 34 were known still to be alive. Re-assessment was carried out by means of personal interview in 54 cases; of the remaining 46, 35 were dead, eight were not interviewed on general practitioner's advice, or had moved too far away, and three were lost from follow-up. The personal interviews were carried out by one of us using a structured questionnaire. For all but four of the remainder, information on physical and mental status was obtained from general practitioners (GPs), social workers, officers in charge of residential homes, and others. The outcome of psychiatric and physical health was thus known for 96 of the original 100 cases from the time of admission to either death or close of study. This gave a follow-up period of 104 months for patients admitted early in 1976 and 42 months for those admitted in early 1981; the four about whom information was not complete had nevertheless been followed-up by the service for an average of 21 months.

Patients were assigned to an outcome category in terms of depressive symptoms, after Post (1972): Group 1: lasting recovery until death or close of the study; Group 2: further relapses, but with complete recovery between episodes; Group 3: some degree of residual depressive symptomatology, often with further circumscribed relapses ('depressive invalidism'); Group 4: ill with depression until death or close of the study.

In addition, the services received by these patients (inpatient, day hospital, outpatient care) and the treatments they received, from the date of first admission to exist from the study, were recorded.

Results

Characteristics of patients at entry to the study

Age, sex, and social circumstances. Women ($n=79$) outnumbered men ($n=21$) by almost 4 to 1. The age-range was 65–88 years (mean = 74.0; s.d. = 4.93), with only two patients over 85; 22% were still married, 24% were single (of whom three out of four were spinsters), 50% were widowed, and 4% were separated or divorced. Ninety-two per cent were members of a private household and 49% lived alone.

This cohort was therefore older than those described by Post, and similar to that described by Murphy; compared with Post's 1962 cohort, it included more women who were either widowed or single.

Previous psychiatric history. Seventy-nine per cent fitted Murphy's (1983) criterion for a new case, i.e. having had no previous depression since the age of 60, although 46% of the whole group had a history of depression at some time previously, and 35% had been admitted to a psychiatric hospital.

Precipitating events. Information from the case notes was judged to be adequate for 91 patients; of these, 79% had experienced a major adverse life event in the year preceding admission. The type of events were: bereavement (30%); exit of relative or close friend (13%); major health change (13%); change of home (11%); retirement (4%); and other events (8%).

Psychiatric status. All were suffering from primary depressive states as defined by Feighner, and in all but 18, the onset of symptoms had been within the previous 12 months. Most patients were severely ill. The average HRSD rating at admission for the 94 patients who could be scored was 27.5; the remainder could not be scored because of incomplete information in the notes, apart from one patient who had been stuporose: 31% were within the range 19–24, 34% 25–29, 25% 30–34, and 10% more than 35, the highest score being 40. Serious suicide attempts had contributed to the presentation in 18 cases, and depressive delusions were prominent in the mental states of 44. Thus, these patients showed similar evidence of psychotic ideation to Post's, but were more severely ill and more likely to be called psychotic than Murphy's

Physical health. Nineteen per cent were judged to have no physical pathology and a further 22% only one quiescent problem; 13% had evidence of multiple quiescent pathology but no active problems, while an active health problem was present in 46%, which in 17% of all patients affected more than one system of the body. Six major body systems contributed to most of the pathology: cardiovascular (31 inactive and 9 active problems); central nervous system (7 inactive and 12 active); musculoskeletal (4 inactive and 11 active); respiratory (7 inactive and 7 active); genitourinary (6 inactive and 8 active) and gastrointestinal (6 inactive and 4 active).

Treatment prior to admission. Antidepressants, usually initiated by the GP, had been given to 47% prior to admission.

The index admission

This was relatively short for most patients: 20 were in hospital for less than one month, 56 for between one and three months, 18 for between four and six months, three for between seven and 12 months. Only three remained for more than a year, one of whom was still there at the end of the study.

With regard to treatments received, 45% had antidepressants and 48% ECT, either alone or in conjunction with drugs. The ECT course (always bilateral) averaged nine

treatments. The antidepressants were, in order of frequency, dothiepin, amitriptyline, and imipramine, with only four patients receiving other drugs, regardless of which of these was used, the daily dose averaged just over 100 mg (range 50–150 mg). When a change of treatment occurred, the most frequent reasons were either no response to a particular antidepressant (56%) or unacceptable side-effects or risks of physical health (37%).

The numbers of patients with each status at discharge (or at the end of study if not discharged), with the figures from Post's first cohort (in brackets) for comparison were: well, 69 (22); improved, 21 (65); little or no change, 7 (12); dead, 2 (1); not discharged, 1 (0). These two sets of figures differ significantly for the first three categories ($\chi^2=46.10$, d.f.=2, $P<0.001$), and show that our patients were more likely to be well at discharge.

Status one year on from the date of first admission

One patient had been lost to follow-up by this time, but detailed information for the rest was available. This interim appraisal has been included mainly to allow a comparison with the findings of Murphy (1983). Her categories of outcome have been used, and following her decision to exclude four patients who became demented during the year, we excluded one such case from our cohort. The majority of patients had done well: 58% of the whole sample were recovered and 65% of the subsample of 'new cases' (no previous illness since the age of 60) were well at one year. Within this group of recovered patients, there were 11 from the main sample and seven from the sub-sample who had experienced relapses during the year which had been detected and successfully treated by the service; 15% (13% of the sub-sample) were in relapse, but had spent a substantial proportion of time well, and were clearly differentiated from the 18% (17% of the sub-sample) who had continued to suffer from some symptoms, not always amounting to 'illness', throughout the year. Only 8% (5% of the sub-sample) had died (Table I). This represents a substantial significant difference from Murphy's findings, with a skew towards better outcome and fewer deaths.

Further analysis explores the suggestion that severity of symptoms and the presence of delusions at the time of presentation carry a particularly poor prognosis.

Severity. Four ranges of severity were earlier described for the 94 patients scored on the HRSD; the numbers in each of the four one-year outcome categories were calculated for each range of severity. There were no differences beyond chance expectations for the whole group $\chi^2=10.15$, d.f.=9, NS) or the sub-sample of 'new cases' ($\chi^2=10.72$, d.f.=9, NS). Six patients could not be scored for severity. Four of these had delusions and one had been stuporose; it could therefore be argued that these were 'severe' cases. The sixth patient refused to answer all the interviewer's questions and so could not be scored; his status at one year was 'well'. The outcomes at one year for the other five were: two well, one relapsed, one continuously ill, and one dead. Recalculating the data with these five patients included in the most severe band still yields non-significant results for all cases and 'new cases' (two of these five).

TABLE I
Status of patients one year from index admission

Outcome	All cases ¹ (n=98)		New cases only ² (n=77)		Murphy (1983) (n=120)	
	n	%	n	%	n	%
Well	57	58.1	50	64.9	44	36.7
Relapsed	15	15.3	10	13.0	23	19.2
Residual symptoms or continuously ill	18	18.4	13	16.9	36	30.0
Dead	8	8.2	4	5.2	17	14.1

1 Compared with Murphy's sample, $\chi^2 = 10.48$, 3 d.f. = 3 $P < 0.02$

2. Compared with Murphy's sample, $\chi^2 = 15.71$, 3 d.f. = 3 $P < 0.01$

TABLE II
Relationship between initial presence of delusions and one-year outcome for all cases (n=97) and new cases only (n=76)

Delusions	Outcome	Well		Relapsed		Continuously ill or invalided		Dead		Total	
		n	%	n	%	n	%	n	%	n	%
Absent	All cases	28	28.9	7	7.2	14	14.4	4	4.1	53	54.6
	New cases	24	31.6	5	6.5	10	13.2	3	4.0	42	55.3
Present	All cases	27	27.8	8	8.3	5	5.2	4	4.1	44	45.4
	New cases	25	32.9	5	6.5	3	4.0	1	1.3	34	44.7

Delusions. The definition used was that of the Present State Examination (Wing *et al.*, 1974), and only cases where delusional ideas had been unambiguously present were accepted. Simple ideas of hopelessness, self-depreciation and guilt arising out of depressed mood were not accepted, but both mood-congruent and mood-incongruent delusions were, provided the latter had occurred in the context of severely depressed mood.

The relative frequencies with which cases who presented with delusions were to be found in each of the four one-year outcomes groups (Table II) were calculated. These did not exceed chance expectations for the whole group ($\chi^2 = 3.54$, d.f. = 3, NS) or for the sub-sample of 'new cases' ($\chi^2 = 3.99$, d.f. = 3, NS). Moreover, of those patients who were well at one year, almost equal numbers had been deluded at presentation as had been free from such phenomena for both the whole group and the sub-sample. Thus, the initial presence of depressive delusions did not presage a poor outcome after one year.

The findings of the longer-term follow-up

These are presented in a style to facilitate comparison with Post's follow-up study over six years (1962) and follow-through study over three years (1972); mortality and change in diagnosis are considered independently of the outcome

for depressive symptoms. Six patients developed dementing illnesses, two experienced manic episodes, and four experienced paranoid symptoms not related to a mood abnormality. Thirty five patients died (27 women); two deaths were due to suicide, and one woman died of renal failure secondary to dehydration, soon after admission. The remainder were bronchopneumonia (six), myocardial infarction (six), malignancy (five, including three pancreatic), heart failure (four), stroke (two), and other causes (five). The cause of death of the remaining four patients could not be traced, but all but one (who had moved away) had been known locally, and death for reasons other than natural causes was most unlikely. The death rate was thus similar to that of Post's 1950 younger cohort, over six years, with a distribution of causes that included relatively less 'cerebral' pathology.

The outcome of depressive symptoms during life (Table III) is compared with Post's 1962 findings (see especially his Table IV), and with his 1972 study. The four patients about whom information was incomplete are excluded, although they were followed-up for an average of 21 months; these included one who was well throughout, two with a relapsing course, and one with depressive invalidism. Their inclusion does not significantly alter the data. Outcome is similar for all three cohorts, although the trend identified by Post for extremes of complete recovery or unremitting illness to be less common among patients admitted since the

TABLE III
The overall outcome for depression compared with the studies of Post

Outcome	This study <i>n</i> = 96 ¹		Post (1962) ² <i>n</i> = 81		Post (1972) ³ <i>n</i> = 92	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
Lasting recovery	21	21.9	25	30.8	24	26.1
Further episodes with full recovery	37	38.5	23	28.4	34	37.0
Depressive invalidism	31	32.3	19	23.4	23	25.0
Continuously ill throughout	7	7.3	14	17.4	11	11.9

1. Four cases incompletely followed-up excluded

2. Compared with present study, $\chi^2 = 7.61$, NS

3. Compared with present study, $\chi^2 = 2.32$, NS

TABLE IV
Cross-tabulation of one-year status by overall outcome (*n* = 98)¹

One-year status	<i>n</i>	Overall outcome			
		Continuously well	Relapses with full recovery	Depressive invalidism	Continuously ill
Well	57	20	28	9	0
Relapsed	15	0	10	5	0
Residual symptoms or continuously ill	18	0	1	14	3
Dead	8	2	0	2	4

1. One-year status after Murphy (*n* = 98 to exclude one lost to follow-up and one with dementia); overall outcome after Post, i.e. status of depressed mood to end of study or death

introduction of antidepressant medication is even more apparent among our patients, although just failing to reach statistical significance (one-tailed test, $0.05 < P < 0.10$). Only seven patients experienced unremitting severe depressive symptoms, and of these four died within 12 months, and two had been referred to the psychogeriatric service as unresolved depression, despite treatment as in-patients by other psychiatrists. Of these elderly depressives, 93% were free from depressive illness for most of the follow-up period; in 22% this freedom was absolute, in 39% further episodes responded to treatment by full restitution, and 32% recovered but were less flexible and robust in mood than had been their former style, as well as sometimes experiencing circumscribed relapses of depression.

Patient characteristics and outcome

Demographic. There was no statistically significant relationship between age at admission, living alone, or civil status.

There was, however, an overall difference in the distribution of males and females over the four outcome groups ($\chi^2 = 9.42$, d.f. = 3, $P < 0.05$). This is largely accounted for by the finding that only one male patient experienced lasting recovery, in contrast to 21 of the women. Additionally, four of the seven members of the continuously ill group were men.

Past history. No association could be demonstrated between outcome and either past psychiatric history or a family history of depression. The seven patients who remained continuously ill had experienced more previous admissions than other groups, but this was not statistically significant.

Duration of illness. Only 18% had been ill for more than a year—similar to the 19% of Murphy's (1983) sample and substantially less than the 45% of Post's (1962) cohort.

Duration of illness of over 12 months did not predict a poor prognosis ($\chi^2 = 6.90$, d.f. = 3, NS).

Precipitating events. These have already been described. Neither their presence or absence nor any particular type of adverse event was associated statistically with any of the four outcome groups.

Suicide attempts. For the 18 patients who attempted suicide, this act was not associated with a poor prognosis overall.

Severity of depressive symptoms

The HRSD score at admission can be divided into two bands – 19–29 and 30–40 – to reflect two levels of severity. Excluding the six patients not ascertained for this variable, there were 62 patients with scores between 19–29 and 32 between 30–40. Of the latter, nine patients (28%) experienced lasting recoveries, ten (31%) had relapsing course, eight (25%) were left with residual features of depression, and five (16%) remained continuously ill. Compared with those with lower scores, the outcome differences are just significant ($\chi^2 = 8.36$, d.f. = 3, $P < 0.05$) and are largely accounted for by the finding that five out of six patients with group 4 outcomes (one patient from this group being unascertained) had Hamilton scores at first admission within the higher range of severity. The six excluded patients included none with 'lasting recovery', four with 'further episodes with full recovery', one with 'depressive invalidism', and one 'continuously ill throughout'. Five of these patients could reasonably be described as 'severe' (the outcome of the sixth being within the relapsing group with full recovery), but inclusion of them yields almost identical results ($\chi^2 = 8.44$, d.f. = 3, $P < 0.05$).

Delusions

The initial presence or absence of delusions can be related to each of the four overall outcome categories. As for the one-year assessment of this factor, there are no findings beyond chance expectations ($\chi^2 = 6.42$, d.f. = 3, NS). Of those with delusions at entry to the study, 25% remained well throughout, 34% subsequently had relapsing courses, 23% suffered some degree of depressive invalidism, and 18% remained continuously ill. It is interesting that six of the seven patients pursuing an unremitting course had presented with delusions, but the overall results do not suggest that the initial presence of delusions presages a poor long-term outcome for depressive illness.

Physical health

Health at admission. Statistical analysis will be confined to the six major body systems described earlier. Inactive health problems were distributed in a non-significant fashion among the four clinical outcome groups, and did not therefore predict any particular outcome. Likewise, there was no statistical association between outcome and the pathologies

of each individual body system taken separately, but the absence or presence of active health problems at presentation was not randomly distributed over the outcome groups ($\chi^2 = 12.29$, d.f. = 3, $P < 0.01$). Thus, of those without active health problems at first admission, the majority (70%) went on to clinical outcomes which were either of lasting recovery or of further relapses followed by full recovery. Only 3% of those with no active health problems at admission remained continuously ill with depression. From a different perspective, 20 of the 22 patients (91%) who experienced a lasting recovery had presented with no active physical pathology, while five of the seven (71%) who remained continuously ill with depression had at least one active health problem at first admission. The presence or absence of active health problems at entry was therefore associated with very poor and very good outcomes respectively.

Further health changes. Information concerning major health changes was available for 95 patients: 32 experienced serious changes in physical health during the follow-up period, and occurrence of these was highly associated with adverse outcome for depressive illness. Thus, 5% of group 1, 29% of group 2, 54% of group 3, and 71% of group 4 had experienced such change ($\chi^2 = 18.17$, d.f. = 3, $P < 0.01$). To assess the combined effects of initial health status together with further changes, a global health score was calculated for each case. Scores of 1 and 2 were ascribed for inactive and active health problems respectively at presentation, and a further score of 2 was added for a subsequent serious health change. A score of 0 or 1 indicated either no health problem or only an inactive one, 2 or 3 indicated moderate health problems, and 4 or more serious health problems either at entry to the study or acquired during the follow-up period. Of the 44 patients with scores of 0 or 1 ('absent or mild'), 35 (80%) had outcomes for depression within the best two groups. Of the 28 patients with scores of 4 or more ('severe'), 18 (64%) ultimately had outcomes of either depressive invalidism or unremitting depression, whereas those with 'moderate' scores experienced depressive outcomes little different from those expected by chance. Moreover, the numbers of cases with 'absent or mild', 'moderate', or 'severe' health problems represented within each of the four overall outcome categories did not conform to chance expectations. Of outcome group 1, 86% had 'absent or mild' health problems, decreasing to 42% for group 2 members, 29% for group 3, and only 14% for members of group 4. Correspondingly, only 5% of group 1 had combined health scores within the 'severe' range, compared with 24% of group 2, 46% of group 3, and 71% of group 4. These figures are significant ($\chi^2 = 27.14$, d.f. = 6, $P < 0.01$). The existence of adverse initial health and subsequent development of further physical pathology is therefore a powerful predictor of poor prognosis for depression.

Care provided after the index admission

Services provided by the psychogeriatric service. Two patients had no contact with the service after discharge. One

was discharged after persistent family interference with management plans, was subsequently readmitted under a different consultant psychiatrist, and at review, 52 months later, was clearly demented. The other patient defaulted from two out-patient appointments, and her progress until death 48 months later was traced via her GP.

Fifty-six patients either remained under supervision until their death (25) or were under supervision at the 1984 follow-up (31). Of these, one had remained in hospital throughout and 33 had one to eleven subsequent relapses; at other times, they had attended the day hospital or been seen regularly in the out-patient clinic or at home by a doctor, nurse, or social worker attached to the unit, with an average frequency of contact of three months.

The service discontinued its involvement with 42 patients. In 22, this was consequent upon an agreed plan that the GP would take on follow-up arrangements, in one case care was taken over by another specialist, and 19 simply moved away or repeatedly failed to attend the clinic, so that aftercare ended by default rather than by strategy. Nevertheless, these patients were followed up for 2-87 months (average 31), representing 46% of the potential follow-up time for this group. Thirteen of these patients (31%) had one to four admissions subsequent to the index admission and before being lost to further follow-up. In addition, outpatient or home visit contacts had been provided in the same way as described for the group successfully followed throughout.

Treatments for the mood disorder. Thirty-eight per cent were prescribed antidepressants on a prophylactic basis, either as a consequence of the index attack or of a subsequent relapse. A further 19% were prescribed antidepressants in response to relapses, but not maintained on them. Lithium was used in nine patients, but withdrawn from two because of side-effects. Thirty-six patients (including 12 from group 1) received either no psychiatric medication after the index admission or were in receipt of it only sporadically and without comprehensive supervision.

Comparison of Murphy's and Post's categories

A cross-tabulation (Table IV) shows the relationship between outcome at one year, as assessed by Murphy's method, and the longer-term 'course' outcome by Post's method. Of the 57 patients judged well after one year, 20 remained well, 28 experienced relapses (11 had occurred before the one-year cross-sectional analysis), and 9 suffered eventual chronic invalidism with or without relapses into illness. None, by definition, were continuously ill. Of those 15 in relapse at one year, ten remained characterised as 'relapsers' with recoveries to full health, and five were later chronic invalids; none, by definition, were continuously ill. Of the 18 described as 'continuously ill' up to one year, only one became so well that she could be classified as a relapser but able to regain full health; however, only three remained continuously ill in Post's terms, the remaining 14 suffering chronic invalidism but not to the extent of persistent illness. Four of the patients classified according to Post as 'continuously ill' had died within the first year, as had two

'chronically invalided' and two who had made a full recovery.

Discussion

The patients in this study were a highly selected sample of elderly depressives from the population at risk. They had been referred to a specialist service by other doctors who had made their own attempts to treat the mood disorder, and the specialist service itself felt that they were not treatable at home, whereas in general, two out of three depressives referred were managed without recourse to admission. Thus, they are likely to include more 'difficult' cases than those included by Post in his study of admissions in the 1950s, when the choice of effective drug therapy was limited and treatment might be stylised as either 'nothing' at home or admission for ECT. It is also possible that they were a more highly selected group than Post's later cohort of admissions, since his service at that time did not advertise availability of domiciliary treatment by the specialist team, although out-patient care was certainly practised and the range of physical treatments in use has not changed greatly. They represent one-third of the patients described by Murphy, who included all those referred from other services to the psychogeriatric team.

Post's cohort included younger patients and more men, but the patients in this study were very similar to those referred to the services studied by Murphy in terms of age, sex, social circumstances, duration of symptoms, and general physical health. We are less sure about social class, although the spectrum is probably similar, and we lack detailed information equivalent to Murphy's detailed enquiries about the presence or absence of a 'confidant'. Serious life events during the year before admission appear to have been more common in these patients than in those described by Murphy (1982, 1983).

It is interesting that the treatment used during their index admissions was very similar to that used by Post in the 1960s, ECT still finding a significant place and the antidepressants used being well-established drugs whose side-effects are well known and usually less limiting than is stated in the literature. On average, they spent less time in hospital than both of Post's earlier cohorts. During the index hospital stay, 21% of the group remained in hospital for longer than three months, compared with 41% of Post's 1960s cohort and 78% of his 1950s group.

The overall pattern of outcome at one year is dramatically better than that reported by Murphy: fewer patients had died and many more were well, while a particularly gloomy outcome for those who

presented with severe symptoms or delusions is not evident. The differences are so striking and the impact of Murphy's work has been so important that explanations are essential. It may be argued that our view has been rose-tinted, but this would not extend to differences in death rates, and is not in keeping with the longer-term follow-up. There can be only three explanations for our findings.

Our patients were intrinsically different and more likely to get better. This must be a possibility, though they shared many features in common and were certainly a 'more ill' group than the 'total referral' cohort in Murphy's sample. Nor can the differences be attributed solely to inpatient or outpatient status, for there was evidently no difference in outcome between those who were admitted and those who were not in East London (Murphy, 1983).

The services studied by Murphy were less effective than those in South Manchester. This too must be a possibility, although the styles of service are very similar and all are highly regarded; the range of treatments was the same, although possibly different use was made of them. It is easier to monitor response to treatment among inpatients, and it may also be easier to identify and treat relapses among patients who have been understood in depth while in hospital. Eleven such relapses that occurred to our patients during the first year after discharge were identified and successfully treated before 'one-year assessment'. No such occurrence was reported by Murphy; her comment on the 'poor' outcome cases that "remissions following treatment were often short-lived" begs the question as to whether or not those who relapsed were identified and energetically treated by the psychogeriatric service.

The only clear difference in treatment is that 48% of our patients received ECT (65% of those who were deluded), compared with only 16% (48% of the deluded) of Murphy's patients. We have remained convinced of the virtues of bilateral ECT (Benbow, 1985), but Murphy did not state which method was used. Since ECT is the most effective treatment for severely depressed patients with delusions, especially when antidepressants have failed to help them (Kantor & Glassman, 1977; Gill & Lambourn, 1979), it seems possible that some of Murphy's patients were under-treated in this respect.

There are weaknesses in the research assessment of outcome. Our own assessments must be viewed with some circumspection, as they depend on evaluation, based initially on our own records, of a situation some time in the past. However, these records are very detailed and informed by an in-depth knowledge of the patients over a prolonged period. In contrast, Murphy transposed her findings from

lengthy interviews to a number of standardised schedules; this has the apparent advantage of objectivity, but may not achieve accurate appreciation of the patients' experience. This is given credence by her comment that "The two categories 'continuously ill' and 'relapsed' appeared to be indistinguishable...". Such an observation is in keeping neither with Post's findings that many patients experience relapsing courses that do not preclude return to full health or to a status of invalidity considerably less than illness, nor with common clinical experience. An elegant research design and the utilisation of standardised schedules have much to commend them, but the data yielded may be less robust than first sight would suggest.

In contrast to the dissonance with Murphy's findings, there is considerable harmony between the pattern of outcome identified here in the follow-up study at 42-104 months and by Post at six years (1962) and three years (1972) respectively. This is despite the differences in the characteristics of the patients at entry. The shift away from the extremes of good and poor outcome for patients who were admitted in the 1960s rather than the 1950s is further accentuated in our mid 1970s to early 1980s admissions. This is understandable, since patients with the most easily treated states are likely to be excluded by treatment at home by their GP or the specialist team, thus avoiding admission. The reduction in the extreme of the poor prognosis group reflects a heartening response of many apparently difficult cases to sustained vigilance and persistence with effective treatments.

The presence of active physical illness at presentation and occurrence of new illnesses during the follow-up period are confirmed as the major factors prejudicing toward poor outcome. This is in line with previous studies (Post, 1972; Murphy, 1983) and emphasises the need for close collaboration between psychiatrists and both other specialists and GPs. However, most of the patients who suffered from unremitting mood disorder also had severe physical illnesses that were unresponsive to treatment. Carcinoma (two cases) and intractable conditions affecting the cardiac, respiratory, and haemopoietic systems were either present or subsequently developed in five out of seven such patients.

The finding that men did less well than women has not been noted previously, and it remains to be seen if this will become established as common to other groups of elderly depressives. Our men were, on average, more likely to be suffering from physical illnesses, and this may explain their excessive morbidity. Beyond these, no other factor stands out as being of prognostic significance. In particular,

delusions or severe depressive symptoms do not of themselves predict persistent morbidity, when confronted by appropriate and persistent therapeutic endeavour.

Murphy herself and Millard (1983) have suggested that her findings were similar to those of Post. However, direct comparison is not possible because the East London cases were not all admitted, and were assessed only 12 months after presentation, rather than followed through over a much longer period. In addition, the approach to assessment and the categories of outcome chosen by the two authors are quite different.

It is, however, of interest to compare the analyses of outcome for our patients using these two approaches. From the nature of the two extreme categories 'well' and 'continuously ill', it is predictable that the more time that is available, the fewer patients remain within them. Thus, some patients who were 'well' at 12 months suffered relapses from which they recovered to full health or a state of invalidism. Most patients with persistent symptoms for up to 12 months remitted to a state of invalidism, with or without episodes of illness, and one recovered entirely. The outcome categorised in this way at one year is therefore not a very helpful guide to outcome over a longer period, and it understates the importance of the intermediate outcomes of recurrent illnesses, with or without intercalated chronic invalidism, that become the rule by deterioration from full recovery and relapse from sustained illness.

Indeed, persevering with any sort of 'category' analysis of outcome has limitations. Post (1962) described a number of alternative approaches to this problem, but eventually returned to the original scheme (re-used later) which we have employed here to facilitate comparisons. The difficulty that categories designated 'continuous' are vulnerable to losing cases with the passage of time has been mentioned. A patient need only relapse for one month in a six-year follow-up to be placed in the same category as another patient who experiences 12 relapses with return to good health each time, and another who has a period of three months' good health followed by five years of unremitting illness. We encountered no such patient in actual fact, but there were some for whom the pattern of symptomatology changed so markedly that their inclusion in a particular category of outcome appeared unrepresentative of the real situation. For ten patients the change was quantitative, e.g. one man had five relapses in 30 months, but then remained well for the next 36 months. For another ten patients the change was qualitative, eight deteriorating from relapses with return to their former selves or to relapses

intercalated with chronic invalidism, while one made the opposite shift, regaining good health after a period of depressive invalidism. One woman, having been continuously ill from the date of admission for almost a year, began to improve and remained entirely well at follow-up – thus achieving a shift from continuously ill to continuously well (from the time of first recovery). Two patients who had experienced relapses and recovery to good health or invalidism over a period of more than three years then fell into psychotic illnesses that persisted for more than a year – to death for one and to the end of the study for the other. They had been well for large parts of the follow-up period, and thus remained classified among the relapsers with good outcome or invalidism.

These changes in the pattern of symptoms are clearly important, and it would be helpful to devise a classification capable of reflecting them. Interestingly, eight of the nine patients demonstrating a shift for the worse had experienced a new major physical illness. Of the eleven experiencing improved patterns of symptomatology, five achieved more supportive living conditions (one to live with a daughter, four to local authority residential homes), and three had been established on prophylactic medication (lithium for two and an antidepressant for the other).

While for the present purpose of comparison Post's scheme for the classification of long-term outcome is vindicated and supported, it must also be seen as inadequate in the case of many individual patients. A definitive classification of outcome could be achieved only by a prospective study of patients from inception to death, quantifying for this period the proportions of time during which patients had been completely well, intermittently well, depressively invalidated, or ill. Over the total further lifetime, numerous unpredictable factors will appear which affect prognosis, and prognosis of outcome may then be compared with other characteristics over the course of one, two, three, or more years to death.

Conclusion

It is clear that the prognosis for elderly patients suffering from depressive states which are severe enough to demand admission is far from gloomy. Most patients are helped by the exhibition of well tried and tested methods of treatment, offered in the context of a community-orientated psychogeriatric service that does not keep them in hospital for long periods of time, but is able to achieve responsible and responsive follow-up arrangements. Physical ill-health and male sex appear to make recovery from depressive symptoms less complete, but even the

most severely depressed, including those with delusional ideas, can regain normal mood. Collaboration with primary health care, other hospital specialties, and social services is essential in working with these patients, and is repaid by their response to treatment.

Much thought needs to be given to research methodology in this field; in particular, the prognosis for elderly depressives not admitted to hospital requires urgent evaluation. A modification of Post's

method of describing outcome would be helpful, and might be extended to facilitate comparisons with outcome for younger patients.

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Exposure to Parental Alcoholism During Childhood and Outcome in Adulthood: A Prospective Longitudinal Study

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The effects of children's exposure to parental alcoholism was assessed using records from an existing prospective 40-year longitudinal study of working-class families: 176 men who had grown up with an alcoholic parent or parents were compared with 230 men without such exposure. Degree of exposure to alcoholism in the childhood family environment was highly correlated in later life with alcohol use, alcoholism, time in jail, sociopathy, and death, but not with increased rates of unemployment, poor physical health, or measures of adult ego functioning. Most of the impairments observed occurred in those subjects who actually developed alcoholism. Exposure to alcoholism in the family environment and family history of alcoholism independently contributed to the later development of alcoholism.

Alcoholism is a disorder that occurs in families (Cotton, 1979). The importance of genetic factors in this relationship has been well established, but the role of stressful environmental factors remains unclear. There is increasing concern that the children of alcoholics may be at risk for a variety of psychosocial impairments as a result of direct exposure to the parents' drinking behaviour. However, little attention has been directed at the quantitative examination of the degree of exposure of the child to the alcoholic parent or parents in the family environment during childhood. If environmental psychosocial factors do play a role in the occurrence of disorders in the offspring of alcoholics, then variability in exposure to such factors would be expected to affect offspring differentially; this paper examines the degree of exposure to parental alcoholism during childhood and its outcome during adulthood.

Previous studies

Investigators employing cross-sectional study designs have examined samples of children with parents who have presented for treatment for alcoholism. These studies demonstrated more deviant behaviour (Fine *et al.*, 1976), higher rates of psychiatric diagnoses (Herjanic *et al.*, 1977), more social problems (Chafez *et al.*, 1971), and more medical and psychosomatic symptoms (Nylander & Rydelius, 1982) in the children of alcoholics than in comparison subjects at the time of assessment. In terms of psychological functioning, children of alcoholics have been found to have poorer self-concept (Prewett *et al.*, 1981) and a lower sense of control on a locus of

control measure (Kern *et al.*, 1981) than comparison subjects. Various patterns of disrupted family functioning have also been observed (Wolin *et al.*, 1979; Wilson & Orford, 1978). The effects of such psychological factors on adult outcome is unknown.

Long-term impairments are evident in the children of serious alcoholics who have been followed over time. For example, in a recent follow-up study of men whose fathers had been diagnosed as alcoholic 20 years earlier (Rydelius, 1981), major impairments were evident in the areas of need for social assistance, alcoholism, social problems, sick leave from work, somatic complaints, and visits to a prepaid health clinic. Similar correlations were not found among the controls.

Genetic studies using adopted-away designs concur in finding a significant genetic component in the transmission of alcoholism. In several of the studies (Cadoret *et al.*, 1980; Goodwin *et al.*, 1973, 1974, 1977a, 1977b), data indicated that environmental factors did not play a large role. In others (Cloninger *et al.*, 1981; Bohman *et al.*, 1981), two distinct types of inheritance were postulated: one purely determined by genetic factors, and the other determined by an interaction of the milieu and a genetic susceptibility to alcoholism (termed 'milieu limited'). At the present time, it is not known which of these two is correct.

Longitudinal studies of subjects not identified initially because of alcoholism, but involving some subjects who develop alcoholism, present an opportunity to examine the antecedents of adult alcoholism. Studies of youngsters already identified in childhood as deviant have been followed by McCord

& McCord (1960) and by Robins (1966). They demonstrate that such antisocial youths, coming from homes where parents abuse alcohol and have a number of problems associated with social disadvantage, develop both alcoholism and sociopathy more frequently than comparison subjects. McCord & McCord postulated personality factors and family variables as the key to understanding later alcoholism; Robins placed more emphasis on genetic factors.

Longitudinal studies of subjects selected for health at the time on entry have particularly examined the issue of whether there is a specific personality trait, or cluster of personality traits, that predisposes to alcoholism, following the suggestion (McCord & McCord, 1960; Jellinek, 1960) that such personality factors exist. The weight of the evidence from longitudinal studies is against this (Beardslee & Vaillant, 1984; Vaillant, 1980; Kammeier *et al.*, 1973).

A sample of men recruited by Glueck (1950, 1968) as comparison subjects for the study of delinquents have proved particularly valuable in understanding both the course and the aetiological components of alcoholism, because of its size, the length of the prospective longitudinal follow-up, and the relatively high proportion of alcoholics. The natural history of this disorder has been described (Vaillant, 1983), and the process of recovery was carefully delineated by Vaillant & Milofsky (1982a). Investigators have separated the antecedents of several overlapping adult outcomes in this sample: poor mental health, sociopathy, and adult alcoholism (Vaillant & Milofsky, 1982b). Using multiple regression techniques, four childhood variables (childhood environmental strengths, boyhood competence, childhood emotional problems, and IQ) significantly predicted adult ratings of mental health 33 years later. Four other variables predicted sociopathy: delinquent parents, multi-problem families (families with lack of cohesion and lack of affection or supervision by mother or father), poor infant health, and premorbid antisocial personality. Finally, four more variables were significantly associated with adult alcoholism, but not with sociopathy or poor mental health; alcoholism in parents, alcoholism in ancestors (excluding parents), alcoholism in heredity (a combined scale of the two previous categories), and ethnic background.

Our current study is a natural extension of previous explorations of Glueck & Glueck's sample, and examines the effect of the child's degree of exposure to the parents' alcoholism. We predicted that varying degrees of exposure to parental alcoholism would affect blindly related data of adult outcomes. Exposure to parental alcoholism includes

both the amount of drinking and the disruption of family functioning due to alcoholism, in such areas as discord, unavailability, lack of financial support, and family violence.

We specifically examined the following hypotheses:

1. Within the entire sample, increasing amounts of exposure to alcoholism and its concomitants in the environment of the developing child will be correlated with higher rates of general impairment in adulthood.

2. Increased exposure to alcoholism in the environment will lead to increased alcohol use, increased abuse of alcohol, and increased sociopathy in adulthood.

We examined the effects of the degree of exposure to parental alcoholism on the age of death in the two groups; the number of men who had died was expected to be too small to be significant, and thus no specific hypothesis was formulated.

In addition, an exploratory analysis was undertaken to examine the relative contribution of an extensive family history of alcoholism and of exposure to environmental alcoholism in terms of the outcomes in adulthood.

Method

Sample

The subject sample (Vaillant, 1983; Glueck & Glueck, 1968; Glueck & Glueck, 1950) consisted of 456 inner city youths chosen by the Gluecks as control subjects in their prospective study of juvenile delinquents. Between 1940 and 1945, these men were studied in junior high schools as non-delinquents. They had been carefully matched with respect to IQ, ethnicity, and residence in high-crime neighbourhoods with 500 Boston youths who had been remanded to reform school. Almost all of the men were from blue collar homes, and 31% of their parents met the Hollingshead-Redlich Social Class V criteria (unskilled workers in derelict housing with nine or less grades of education). Of the boys' parents, 61% had been born in a foreign country. All the subject families were Caucasian, except for one Oriental family.

At the time of the original study, the boys and their parents and teachers were individually interviewed. For all first-degree relatives, public records were searched for an excess of alcohol or criminal behaviour and mental illness. Attrition over the years has been modest. The majority of subjects were re-interviewed at ages 25 (circa 1955), 32 (circa 1962), and 90% of them at 47 (circa 1977).

Of the 456 men, 176 had had some exposure to alcoholism. For the purpose of this investigation, these 176 men were defined as a sample exposed to alcoholism and then rated using the Exposure in Childhood to Alcoholism in the Family Environment scale (ECAFE), while the remaining men served as non-exposed comparison subjects.

Independent variables assessed

Clinicians blind to all information after adolescence reviewed social service records and interviews with each boy, his parents, and teacher, and rated each subject on the following premorbid variables:

IQ: Each subject was administered the Wechsler-Bellevue Intelligence Test on entrance to the study.

Social class: Using the five-point classification scale devised by Hollingshead & Redlich (1958), each participant was rated for social class on the basis of his parent's education, father's occupation, and familial residence.

Alcohol abuse in relatives. This scale (Vaillant & Milofsky, 1982b) reflected alcoholism in the child's heredity (in known ancestors prior to 1945). Parents and siblings were included as well as other first- and second-degree relatives, if known. If reliable judgement was not possible, due to ambiguous or absent familial data, a rating was not made. The scale points employed were: 1 = no evidence of alcoholism in the family; 2 = one relative with minor evidence of alcoholism (two convictions for alcoholism, mention of alcoholism in official records, or strong suspicion in the case record that was compiled from family interview and multiple social service agency reports); 3 = two relatives with minor evidence of alcoholism or one relative with major evidence (defined as two or more criteria for minor evidence and evidence for chronicity); 4 = three relatives with minor evidence or two relatives with major evidence.

The ECAFE scale

The data base for rating the Exposure in Childhood to Alcoholism in the Family Environment (ECAFE) scale included all material collected on the child and his family until age 17 in the existing Glueck records. It included direct interview material, family interviews, school records, and psychological tests. The scale and a manual defining the scale points were constructed by detailed review of a series of records to identify variables that could be reliably rated, were expected to be harmful to the developing child, and were likely to be associated with alcoholism. Greatest weight was given to extent of parents' drinking, with less weight for associated problems.

For father, mother, stepfather, and stepmother, separate ratings were made of the extent of alcoholism in the family environment to which the child was exposed. Ratings of 0 were made when there was no evidence of alcohol abuse whatsoever, or if the subject had suffered from alcoholism but not during the child's lifetime. Ratings of 2 were made when there was some evidence of alcohol abuse (possibly involving bootlegging charges, or arrest for violation of liquor laws) but such evidence was not extensive in the records. Ratings of 4 were reserved for situations in which alcoholism was severe and long standing, having occurred during at least several years of the child's life. Criteria included references to chronic alcoholism in the material from the raters, indications that home and work life were severely affected by the subject's drinking, and a record of more than five arrests for drinking behaviour.

In addition, individual ratings of either 0 or 1 were made for six types of family behaviour harmful to children: fre-

quent absence of the parent (not due to employment or military service); fiscal irresponsibility of the parent; physical abuse of the spouse or the child by the alcoholic parent; and two ratings of disorder – general discord and specific parental marital discord. It is not possible to say that every episode of these six types of behaviour occurred because of a specific episode of drinking, the intent was to code them when they occurred in association with overall drinking behaviour. They were chosen as six clear behaviours which were known from general review of the literature of alcoholism in families to be associated with alcoholism, were also known to be harmful to children during their development, and which could be rated on the basis of 40-year old records.

Finally, a senior clinician reviewed all the records in order to weight the time interval of exposure of the child to the parents' alcoholism. This variable was given a score of 0, 1 or 2 and was used primarily to give added weight to families in which there were long periods of exposure to alcoholism (greater than five years), in comparison with families in which alcoholism had been limited only to a relatively short period of the child's life.

Reliability was examined by means of a blind paired rater trial, using 25 rated records before the ratings were made, and was found to be above 0.60 for all except 'drinking parent financially irresponsible', for which it was 0.50.

Using this rating process, scores on the ECAFE scale ranged from 0-15. The median score for the exposed group was 7, the mean score 7.1, and the standard deviation 3.06.

Outcome variables: overall adult functioning

On the basis of two-hour interviews at age 31 and 47, and of a review of recent mental health and arrest records, clinicians blind to all data gathered before age 31 rated the men at age 47 (± 2 years) on the following variables.

Overall adult functioning

Percentage of time unemployed. This was taken as the percentage of time from entry into the study until the time of measurement unemployed.

This was rated using the overall health Health Sickness Rating Scale (HSRS) (Luborsky, 1962). On the basis of graded ratings, HSRS places individuals along a 100-point continuum that ranges from total institutional dependency (0-10) to exhibition of multiple manifestations of positive mental health (90-100).

Social competence. This 25-point scale (Vaillant & Milofsky, 1980) measures the capacity for human relations; ratings reflect relative success in accomplishing eight difficult tasks of adult object relations. One task each reflected enjoyment of children, family of origin, and workmates; three tasks reflected friendship networks, and two tasks reflected participation in group activities. Marriage is excluded.

Mood disturbance. This was rated a measure of mental health at time of interview. Mood ratings were made on a four-point scale. 1 = excellent; 2 = good; 3 = fair, moody; 4 = depressed, blocked.

Alcoholic/sociopathic variables

Alcohol dependence (DSM-III). A three-point scale defined by DSM-III (American Psychiatric Association, 1980) and reflecting the medical model for diagnosing alcoholism was used: 1=no alcohol abuse; 2=alcohol abuse; 3=alcohol dependence (evidence for alcohol abuse and tolerance or withdrawal symptoms from alcoholism).

Time in jail. This was expressed as the percentage of time the subject had spent incarcerated since age 18.

Sociopathy. This rating was based on the 19 criteria used by Robins (1966) for the diagnosis of sociopathic personalities. Presence of five or more criteria led to the diagnosis of sociopathic personality.

Problem drinking scale (PDS). This scale (Vaillant, 1980) was an equally weighted 16-point scale devised to combine the emphasis of DSM-III (American Psychiatric Association, 1980) on the physiological dependence and the emphasis of the Cahalan scale (Cahalan, 1970) on social deviance. The scale depends on objective alcohol-related problems (e.g. blackouts, arrests, medical complications of alcohol abuse) and gave double weights for alcohol problems that occurred frequently. Men with one or no symptoms on the scale were by definition social drinkers; men with four or more symptoms were defined as alcohol abusers.

Statistics

In order to examine the effects of increasing amounts of environmental exposure to parental alcoholism using the ECAFE scale, Kendall's tau was used, as both the outcome variables and the dependent variables contained ordered categories. In order to examine the inter-relationship of alcoholism in heredity and the family environment, two different statistical techniques were used. Firstly, the analyses of the ECAFE scores compared with adult outcome were repeated specifically for those subjects who had

high scores (3 or 4) on the 'alcohol abuse in relatives' scale, in order to separate, as much as possible, the effects of family history of alcoholism from environmental exposure. Secondly, stepwise multiple regression techniques were used to explore further the relationship between the 'alcohol abuse in relatives' and ECAFE scales. IQ and social class were included as covariates. The regression analyses were carried out without ordering the variables before entry.

Results

Although attrition had been modest, not all men had received all measures in the project. Samples sizes thus varied between categories of outcome

Overall functioning

For four measures of overall functioning in adult life (percentage of life unemployed, HSRS rating, social competence, and mood disturbance), increasing amounts of exposure to alcoholism did not result in increased disorder, when compared with the non-exposed group of men at follow-up. A summary of the mean group scores for four levels of ECAFE exposure is given in Table II. There was a difference between the extremes of exposure for only one variable: years unemployed.

Alcoholic and sociopathic outcomes

A strikingly different picture emerged when the outcomes of DSM-III diagnosable alcoholism, time in jail, sociopathy, and problem drinking were analysed (Table I). In each area, significant associations were found between increasing levels of exposure to alcoholism in the child's family environment and poor outcome.

A diagnosis of DSM-III alcohol dependence was received by 71 men in this sample. Of these, 26 came from the sample

TABLE I
Relationship of childhood exposure to alcoholism and adult outcome: mean score and statistical association

ECAFE range	Time unemployed: %	HSRS	Social competence	Mood disturbance	DSM-III Alcohol dependence	Time spent in jail	Sociopathy	Problem drinking
0	8.80	74.82	5.99	2.46	1.36	1.32	2.06	2.84
1-4	4.81	75.77	5.91	2.32	1.60	1.45	2.50	3.90
5-7	8.86	73.37	6.16	2.44	1.70	1.60	3.00	4.72
8-9	11.54	74.17	5.82	2.38	1.81	3.00	2.76	5.54
10+	16.00	73.57	6.48	2.43	1.91	1.98	3.51	5.49
Kendall's tau	0.028	-0.34	0.028	-0.03	0.207	0.08	0.169	0.220
P	0.235	0.194	0.248	0.26	0.00001	0.03	0.00001	0.00001
Total n	419	377	383	390	398	430	429	441

with no exposure ($n=280$, i.e. 9%), while 45 came from the sample with an exposure to alcoholism in the family environment ($n=176$, i.e. 26%). In terms of sociopathy, 19 (7%) men received a definite diagnosis in the non-exposed group, while 31 (18%) did in the exposed group.

Age of death

Although the numbers were modest, there was a higher death rate in those exposed to alcoholism in childhood (Table II). Causes of death present no clear pattern. Most of this effect occurred in the older age-group: 10 men had died before age 35 (4%) in the non-exposed sample, and four (3%) in the exposed sample. After age 35, 17 (7%) had died in the non-exposed sample, while in the exposed sample 25 (15%) had died.

Nature of individuals seriously affected

The effects of parental environmental exposure to alcoholism on outcome were then examined, excluding the cases with a DSM-III diagnosis of alcohol dependence (71 cases), and the cases where no diagnosis was possible (57). It was then possible to examine whether the main effects in this sample occurred in those individuals with severe alcoholism, or whether such effects were more evenly distributed throughout the entire exposed sample. When the cases of diagnosable alcoholism are removed in this way, the results for the overall functioning measures are not significant, as before. For the alcoholic and sociopathic outcomes, significant effects are not present for any of the variables except problem drinking behaviour (Table III). Most of the effects of environmental exposure were thus due to the relatively few individuals who were seriously affected.

Relative contributions of family history and childhood environmental exposure

For the sample with high (3 or 4) scores for family history of alcoholism, more exposure to parental environmental alcoholism resulted in more pathology. There is a significant association of ECAFE score with sociopathy, DSM-III diagnosis of alcoholism, problem drinking, the overall health rating, cumulative unemployment, and time in jail, but not mood disorder, defences, or object relations (Table IV). This is similar to the previous analysis (Table I),

although some variables of overall adult functioning now reach significance.

These findings were explored using multiple regression techniques (Table V). Three main outcomes were examined: DSM-III alcoholism, sociopathy, and problem drinking.

TABLE II
Age of death of men in sample ($n=196$)

	ECAFE range				
	0	1-4	5-7	8-9	10+
Deceased	27	2	8	7	8
Living	252	38	47	35	31
Percentage*	9%	5%	17%	20%	26%

*Kendall's tau = 0.178, $P=0.05$

TABLE III
Relationship of childhood exposure and adult outcome in subjects, omitting those who received the DSM-III diagnosis of alcohol abuse mean scores and statistical association for alcoholic and sociopathic outcomes

ECAFE range	DSM-III alcohol dependence	Time spent in jail	Sociopathy	Problem drinking
0	1.17	1.17	1.82	2.04
1-4	1.25	1.00	1.89	2.37
5-7	1.19	1.06	1.94	2.49
8-9	1.21	1.79	1.38	2.13
10+	1.20	1.00	2.60	2.68
Kendall's tau	0.04439	-0.05814	0.05140	0.08312
P	0.2953	0.1272	0.4532	0.0405
Total n	327	327	327	327

TABLE IV
Analysis of exposure in childhood for the sample with a large number of relatives with alcoholism

	Time unemployed	HSRS	Social competence	Mood disturbance	DSM-III alcohol dependence	Time spent in jail	Sociopathy	Problem drinking
Kendall's tau	0.15	-0.14	0.08	-0.08	0.28	0.15	0.25	0.22
P	0.007	0.01	0.12	0.23	0.0003	0.015	0.00001	0.0003

TABLE V
Relative weight of heredity and environmental exposure, by multiple regression

<i>Dependent variable</i>	<i>Independent variable¹</i>	<i>Beta weight</i>	<i>t</i>	<i>p</i>
DSM-III alcohol dependence	Heredity	0.28	4.7	0.00001
	ECAFE score	0.21	4.2	0.00001
Sociopathy	IQ	-0.96	-2.0	0.04
	ECAFE score	0.21	4.6	0.00001
Problem drinking	Heredity	0.19	4.2	0.00001
	ECAFE score	0.23	5.0	0.00001

¹ IQ, heredity, ECAFE score and social class of origin were all tested for each dependent variable; only significant results are shown

Two variables shown to be important in long-term outcome from the overall longitudinal follow-up work were included: social class of origin and IQ. For all three dependent variables (alcoholism, sociopathy, and alcohol use), childhood familial environmental alcoholism made a significant independent contribution. Family history of alcoholism contributed to DSM-III alcoholism and to problem drinking, but not to sociopathy. IQ is related to sociopathic outcome but not to the other outcomes, while social class of origin has no effect.

Discussion

The persistent major effects of increasing degrees of exposure to alcoholism in the family environment on the developing child are in the areas of adult alcoholism and sociopathy. The main hypotheses of our study are strongly confirmed for the outcomes of problem drinking, DSM-III diagnosis of alcoholism, time in jail, and sociopathy, but not for the general indices of overall adult psychological functioning. Multiple regression analyses, and analyses of a sub-set of the sample including all individuals with extensive family histories of alcoholism, demonstrate that both alcoholism in the environment of the developing child and a family history of alcoholism make independent contributions to poor adult outcome.

The main effects of exposure to parental alcoholism in childhood occurred largely in the relatively small group of individuals who develop alcohol abuse; when alcoholic subjects were eliminated from the analysis, the differences between subjects exposed to alcoholism and those not exposed disappears. This, in combination with the lack of effect on major variables of adult functioning (such as overall physical and mental health, years unemployed, and mood), suggests a considerable resiliency in terms of

overall functioning in the children of alcoholics who do not develop alcohol abuse, although such a thesis must be advanced with caution. The most important caveat is that our follow-up sample was 47 years-old at the most recent assessment, and had been initially selected as a control group. The affliction of parental alcoholism may have affected the individuals' lives in ways not evident in these objective outcome variables. Moreover, a common result of parental alcoholism is delinquent adolescents, but this sample was specifically selected for non-delinquency.

The importance of a longitudinal perspective is emphasised by findings from the examination of the age of death. Men whose parents were alcoholic and who were exposed to alcoholism in growing up have significantly higher death rates, but this pattern emerges only in men over 35. No conclusion can be reached at this point about why there is a higher death rate: it is known that the death rate of alcoholics is two to three times that of non-alcoholics, but only 18% of these deaths occurred in men who were alcoholic.

A central issue of interest is the relative weights of genetic and environmental influences in the aetiology of alcoholism in this or other samples. This study was not designed to separate genetic from environmental influences; nor is it possible to do so within the limits of the data, as the two effects occurred together. It may be possible to do it in future years, using techniques of mathematical modelling and results from studies under way.

The relationship of these data to the findings of wide ranging impairments in children of alcoholics in many of the cross-sectional studies deserves comment. Most such reports involve youngsters whose parents presented for treatment of alcoholism in clinics. These samples were assessed in adolescence, not mid-life. Moreover, some children in

the cross-sectional studies who were already delinquent would not have been included in our study. Most importantly, it must be emphasised that we have used blindly rated, objective behavioural outcomes 30 years after the period of parental exposure. This means that many other factors influencing overall mental health functioning, such as quality of marriage, employment, and physical health since childhood, have intervened. We also did not specifically examine such psychosocial factors as self-esteem, family rituals, or other psychodynamic factors related to alcoholism in the cross-sectional examination in childhood.

Limitations

It was not possible to follow all individuals over this long time period with all measures. The tables give the number of individuals for each calculation, and hence give some idea of the number of missing values.

Attrition was more common in those individuals with high ECAFE scores. There was little difference between the exposed and non-exposed groups in terms of missing data on the dimensions of sociopathy, alcoholism, alcohol use, or years unemployed. In 34 cases, attrition limited information on overall mental health rating (HSRS) and social competence, with more data missing from the high-exposure group. If anything, the effects of environmental exposure would be likely to increase in these dimensions, had the additional individuals been present.

It was not possible with this data set to characterise accurately the total length of alcoholic episodes or the exact timing in relation to the child's life, as the initial assessments were conducted in the men's adolescence, and such information in detached diagnostic terms was not elicited. In this sense, the ECAFE scale represents a compromise. In the interval between assessment and follow-up, other variables may have played a role in alcoholism. These factors, such as peer relationships (Jessor & Jessor, 1975) and occupation (Plant, 1979), were not examined as causes of alcoholism in adulthood.

It could be argued that the family environmental alcoholism scale really taps two dimensions: pure drinking behaviour; and family discord and parental irresponsibility. However, since the first is so often highly confounded with the second in the actual experience of children in these families, and the focus

of this investigation is the child's experience, we did not separate them.

The study focused on relatively discrete outcomes in the offspring and the quantification of a single major parental variable (familial exposure to alcoholism and its effects.) We did not attempt to examine various factors within the child which might lead to increasing amounts of alcoholism. Such factors as adaptational capacity, resiliency, early and persistent use of alcohol, drugs, or smoking, and early aggressive behaviour (Kellam *et al*, 1980), all present intriguing areas for study.

Implications

This study emphasises specifically the importance of consideration and further study of the child's contact with and exposure to parental alcoholism in combination with genetic factors. Those families in which parental alcoholism has been long standing and in which there is an association between alcoholism and various kinds of family disorganisation and discord are those in which the youngsters are at highest risk in the long term. There is a need for intervention as soon as possible in the youngsters' lives, and in the parents' course of disorder, because the greater the degree of exposure, the greater the effect.

Conclusion

In spite of the negative effects of growing up in a family with alcoholic parents, the overall conclusion is hopeful. The majority of youngsters in this sample functioned similarly to those in the non-exposed group during adulthood, in spite of the severe alcoholism and discord in the families in which they grew up.

In contrast to the frequency with which poor outcomes such as chronic depression or 'borderline personality' appear to be associated with a history of parental alcoholism, in this study parental alcoholism did not reflect an increase at age 47 of non-alcohol related psychiatric disability.

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Marital Adjustment, Intimacy and Needs in Female Agoraphobics and Their Partners: A Controlled Study

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Female agoraphobics and their partners were compared with three groups of control couples (non-phobic female psychiatric patients and their husbands, maritally distressed couples, and happily married couples) on measures relating to marital adjustment, intimacy, and needs. Neither agoraphobics nor their partners rated their marriages as more maladjusted or unpleasant than non-phobic psychiatric patients or their partner controls. Instead, agoraphobics and their spouses were found to be more comparable to happily-married couples than to maritally-distressed controls. Non-phobic psychiatric patients and their partners were generally found to rate their marriages as being as distressing and unpleasant as those of maritally distressed controls.

It has been suggested that the relationship of agoraphobic patients with their marital partner may be of critical importance in the precipitation, maintenance, and perpetuation of the agoraphobic symptoms. A number of authors (Webster, 1953; Fry, 1962; Lazarus, 1966; Hand & Lamontagne, 1976; Goodstein & Swift, 1977; Chambless & Goldstein, 1980; Barlow *et al*, 1981; Goodstein, 1981; Hafner, 1977, 1982; O'Brien *et al*, 1982) have pointed out that agoraphobia may more suitably be described in terms of both an interpersonal, particularly marital, conflict and an intra-psycho conflict. However, in comparison with the emphasis that has been put on the possible role of marital difficulties in the aetiology of agoraphobia, hardly any methodologically sound studies have been conducted with the aim of scrutinising whether and to what extent the marriages of agoraphobics differ from those of controls.

Goldstein (1973) concluded, on the basis of case-history reports, that agoraphobics can be typified at the time of symptom formation by four characteristics: (a) female; (b) in a close relationship, usually married; (c) feeling a strong urge to escape this relationship; and (d) feeling trapped in the sense that the patient is afraid of or intimidated by possible alternatives. He argued that conflict created by an unhappy marriage is a constant situation from which escape can only be through psychological avoidance responses, and proposed that "agoraphobic symptoms are often the result of such psychological avoidance behaviour in conflict situations that seem unsolvable". Thus, in his view and that of Chambless & Goldstein (1980), agoraphobics wish to flee the marriage but cannot do so because of their dependency and fears of being alone.

Most of the available studies emphasising the importance of quality of marriage in the aetiology of agoraphobia (e.g. Webster, 1953; Fry, 1962; Goldstein, 1973; Symonds, 1973; Hudson, 1974; Shafar, 1976; Melville, 1977; Hafner, 1981; Holmes, 1982) are based on confusing methodologies, characterised by any of the following aspects: (a) lack of adequate control groups; (b) unspecified or inadequate operationalisation of the criterion measures (e.g. marital adjustment, intimacy, or quality); (c) the use of interview techniques of unknown reliability and validity; (d) retrospective description of clinical anecdotal material; (e) use of samples that are biased, inadequately specified as to diagnosis and outcome, and whose patients have been selected and treated by the authors themselves; and (f) lack of distinction between the sexes. Despite these shortcomings, these studies have provided an abundance of comments concerning the quality of marriage in agoraphobics and their partners.

On the basis of data obtained from an unspecified number of female, mainly agoraphobic patients, Fry (1962) suggested that the husbands' statements indicate doubt about their commitment to the marriage and that they were also characterised by their jealousy and occasional impotence. Usually, the marriage was miserable and the couple distant and discontented, the symptoms functioning to keep the couple united in a compulsory marriage. Though the discontented patient may at times indicate some desire for an extramarital relationship, her phobia prevents her from getting involved with other men.

Symonds (1973) selected three female patients suffering from intense fear of travelling and of being alone, from records of a larger group of patients with

phobias ($n=12$) gathered over a period of 20 years. A picture emerged of the patients as being helpless and dependent, and unable to handle even the slightest friction that occurs between two people who live together. Symonds argued that the many psychosomatic symptoms observed were an expression of repressed anger towards the partner, and that the phobias are one of the ways of dealing with their repressed feelings.

Hitherto there has only been one controlled study examining the quality of agoraphobic marriages: Buglass *et al.* (1977) compared agoraphobic housewives and their spouses with normal control couples in terms of domestic activities (i.e. the execution of specified family tasks), decision-making in a number of areas, and manifest interaction ratings (assertion-compliance and affection-dislike). The most striking feature of the comparison between the two groups of couples was their similarity with respect to these measures.

The aim of the present study was to investigate the quality of the marital relationship of agoraphobic patients and their partners by comparing their marriages with those of three groups of controls: non-phobic psychiatric patients and their partners; maritally distressed couples; and non-distressed (happily married) couples.

Clinical anecdotal studies suggested that agoraphobics as a group would: (a) report their marriages as more distressing or maladjusted, both maritally and sexually; (b) indicate greater needs for interpersonal contacts and intimacy (not necessarily sexual or erotic) outside their marriages; (c) appear to be emotionally more dependent on their marriage partners and show greater tendencies towards centredness on their partners; and (d) exhibit greater avoidance of communication and greater lack of intimate communication than non-phobic psychiatric and maritally non-distressed controls (e.g. Webster, 1953; Fry, 1962; Goldstein, 1973; Symonds, 1973; Melville, 1977; Holmes, 1982). As a consequence, agoraphobics should appear to be more comparable to maritally distressed subjects than to either non-distressed or non-phobic psychiatric controls on measures of marital adjustment and intimacy. Furthermore, as compared with controls, the partners of agoraphobics should show evidence of greater degrees of felt distress, due to the possible intention of the patients to perform extramarital acts with a sexual component (e.g. Fry, 1962). In view of the general picture of the spouses of agoraphobics as being persons who are not dissatisfied with their marriages—i.e. the spouses might have vested interests in maintaining the phobia (e.g. Fry, 1962; Hudson, 1974)—no further specific differences were

hypothesised for comparisons involving the partners and controls.

The above hypotheses suggest that an examination of similarity of marital ratings within and across couples might also prove illuminating.

To control for the possible confounding effect of gender on the self-report of the quality of marriage (e.g. Schumm *et al.*, 1981), only female psychiatric patients were considered here. Surveys from the literature on background factors affecting the self-report of marital quality (Lewis & Spanier, 1979; Dominian, 1982; Markman *et al.*, 1982; Birtchnell & Kennard, 1984; Filsinger & Wilson, 1984) suggested a number of other variables which may be of importance: age at the time of marriage, education, occupation, race, religiosity, and rate of conception of children. In our analysis of data, the influence of these factors was accounted for. Finally, since no study investigating the present matter would be complete without considering the issue of desirable responding (Hansen, 1981), two measures were taken to reduce its impact as much as possible.

Method

Subjects and procedures

Thirty agoraphobic, 11 non-phobic psychiatric, 14 maritally distressed, and 38 non-distressed (happily married) females and their partners took part. Demographic data for husbands and wives are given in Table I.

The agoraphobic out-patients participating in the present study were females who were referred to our department for treatment between October 1980 and May 1983. Their selection was based on three main criteria. Firstly, the subjects had to meet the DSM-III (American Psychiatric Association, 1980) criteria for agoraphobia as a major disorder. Secondly, the agoraphobia had to be persistent, severe, and result in consistent avoidance of the phobic situation. To determine this, 30-minute clinical interviews were conducted to obtain clear evidence of whether work or social life-style were significantly affected by the agoraphobia. Thirdly, subjects had to score at least 17 on the Agoraphobia sub-scale of the Marks & Mathews (1979) Fear Questionnaire (FQ) (mean = 30; range = 17–40).

Three control groups of couples were used. The first consisted of female non-phobic psychiatric patients who were awaiting treatment, on an out-patient basis, after their initial contact with the clinic. Their husbands also participated in the study. The patients were selected on the basis of the following criteria: the subject was judged both by the clinical team and by the researcher not to be suffering from a clinical phobia, and the subject had to score below 17 on the FQ Agoraphobia sub-scale. The second group of controls were randomly drawn waiting-list maritally distressed or conflict couples who were self-referred for marital difficulties to a non-university community mental health centre. To ensure that there would be no overlap with other couple groups, an additional selection criterion was that

TABLE I
Demographic, neuroticism and social desirability data describing each subject group for husbands (H) and wives (W) separately

		Agoraphobic patients and spouses (n=30)		Non-phobic psychiatric patients and spouses (n=11)		Maritally distressed couples (n=14)		Maritally non-distressed (n=38)		Overall χ^2 or F
		Mean	s.d.	Mean	s.d.	Mean	s.d.	Mean	s.d.	
Age: years	H	34.27	8.26	40.45	10.34	37.15	6.72	37.29	13.28	1.02
	W	31.87	8.34	37.45	12.17	34.64	7.23	35.24	13.08	<1
Education: scale of 1 to 5	H	2.87	1.33	2.82	1.60	3.50	0.94	3.87	1.09	4.60*
	W	2.59	0.93	3.18	1.33	2.43	1.16	3.70	1.09	8.03**
Occupation ¹ : scale of 6 to 1	H	3.59	1.00	2.89	1.37	3.29	0.91	2.64	1.14	4.45*
	W	5.50	0.85	3.00	1.41	4.57	0.98	3.05	1.27	11.61**
Length of marriage: years	H	10.81	7.95	14.82	11.05	13.79	7.35	12.29	10.46	<1
	W									
Number of children	H	1.50	1.27	1.73	1.10	2.07	1.38	2.00	2.27	<1
	W									
Religiosity										
High	H		10%		36.4%		14.3%		28.9%	5.47
Low			90%		63.6%		85.7%		71.1%	
High	W		10%		45.5%		21.4%		13.2%	7.87
Low			90%		54.5%		78.6%		86.8%	
Trait-neuroticism	H	11.62	7.51	14.00	7.62	19.86	4.94	11.44	4.82	
	W	21.04	6.93	22.70	6.81	17.46	6.83	12.11	5.98	
Marlowe-Crowne Scale										
Assert-good	H	5.30	1.93	5.90	2.21	5.00	1.71	5.39	1.90	
	W	5.55	2.13	5.73	2.24	6.31	2.20	5.37	2.20	
Deny-bad	H	6.31	2.41	6.18	2.56	4.54	2.06	6.16	2.12	
	W	5.93	2.20	5.70	2.15	5.43	2.17	5.38	2.42	

1. Due to missing data (because of some wives being unemployed or housewives) the *ns* on which the calculations are based differ from those given above

* $P \leq 0.01$, ** $P \leq 0.001$

both husband and wife be judged by the clinical team and by the researcher not to be suffering from a clinical phobia. They were also found to score less than 9 on the FQ Agoraphobia sub-scale. Subjects from the third control group (maritally non-distressed couples) were individuals considering themselves to be happily married, and were solicited from newspaper advertisement for participation in a project on 'communication in marital relationships'. Inclusion criteria for this group, applied to both partners, were: (a) no prior history of in- or outpatient treatment for psychiatric disorders; (b) judged not to be in need of treatment for psychiatric difficulties; (c) scores below 17 on the FQ Agoraphobia sub-scale.

The following criteria were used across all groups: (a) minimum age of 18 years; (b) married and cohabitating with the spouse for at least one year; (c) Dutch-born Caucasian subjects (i.e. ethnically/racially non-mixed couples).

Clinical interviews were conducted in our department after completion of the questionnaire set described below, no volunteering couples were excluded from the study on the basis of these interviews. Since none of the eligible agoraphobic subjects failed to volunteer, they can be considered representative of the female married patients treated in our department in terms of demographic variables and the degree of severity of the agoraphobic complaints presented.

For all subjects awaiting treatment, it was required that they participated prior to its start.

All subjects were tested individually, to prevent the couples from completing the questionnaires together and discussing the items. Standard instructions for the questionnaire set were given to each subject. All questionnaires were completed anonymously, and confidentiality of the subjects' responses was emphasised. To further minimise 'socially desirable' responding, honesty in responses was emphasised, which Eysenck *et al* (1974) have shown to significantly reduce dissimulation. In addition, a measure of social desirability was appended to the questionnaire set, to have some means of evaluating its impact.

Measures

The following scales were utilised.

(a) The *Maudsley Marital Questionnaire (MMQ)* (Crowe, 1978; Cobb *et al*, 1980) in a version adapted for use in the Netherlands by Arrindell *et al*, (1983a). The MMQ is a short 20-item scale relating to marital, sexual, and general life maladjustment (function and interaction). The scale is also known as the Modified MMQ (Cobb *et al*, 1984).

(b) A *General Marital Dissatisfaction* and a *Sexual Dissatisfaction* scale: satisfaction is defined in the sense of learning theory, as the frequency with which one experiences the interaction with one's partner as pleasant (positively reinforcing).

(c) A number of *Communication* scales, referring to intimate communication, destructive communication, *discongruent* communication, and avoidance of communication; these deal with the quality of perceived communicative behaviour in the marital relationship.

(d) The *Need for Interpersonal Stimulation* scale, as a

measure of the degree of an individual's need for regularly experiencing new personal contacts.

(e) The *Need for Intimacy* scale, as a measure of one's need for intimate personal (but not necessarily sexual or erotic) contacts with people other than the marital partner.

(f) An *Emotional Dependency* scale, as a measure of the degree to which one feels oneself emotionally dependent on one's partner.

(g) An *Exclusive Partner-Centredness* scale, as a measure of the degree to which one experiences an exclusive directedness or focus on the marital partner as positively reinforcing.

(h) An *Intentional Extra-Marital Involvement* scale, as a measure of the degree of felt distress from possible intents, ideated by oneself, of one's partner performing extra-marital acts with a sexual component.

(i) A trait-neuroticism scale, the *Delfse Vragenlijst (DV)* (Appels, 1975), as a measure of obtaining a general description of each sample in terms of neuroticism.

(j) The *Marlowe-Crowne Social Desirability (MC-SD)* scale (Crowne & Marlowe, 1964). Both of its sub-scales (Assert-good and Deny-bad), in a version adapted for use in the Netherlands by van Rooijen & Smoor-van Son (1978), were used. Higher scores on these scales indicate the greater tendency of subjects to attribute to themselves desirable but highly unlikely characteristics, and to deny about themselves undesirable but highly likely behaviour.

Data in support of reliability and validity of the MMQ have been reported by Arrindell *et al*, (1983a,b) and Arrindell & Schaap (1985). Construction of scales (b)-(h) is based on work by Buunk & Bosman (1980) and Buunk & Nijsskens (1980), who also report data supporting scale reliability and validity, examples of items captured by these scales are given in Arrindell *et al*, (1983b). Data pointing to the utility, reliability, and validity of the remaining scales (a, i and j) are given in the cited studies. The scales were randomly distributed within each questionnaire set, the only constraint being that the personality measures (i and j) were preceded by the remaining scales (a to h).

Statistical analyses

Data were compared by means of *t*-tests. Although it is widely believed that such parametric statistics should not be used with data which are not normally distributed, such tests are in fact robust in the face of departures from normality, and may actually be preferred to non-parametric statistics (Everitt, 1979).

In making between-group comparisons, *a priori* contrast tests were utilised. The following contrasts were considered: female agoraphobic patients versus (a) female non-phobic psychiatric patients; (b) maritally distressed female subjects; and (c) maritally non-distressed females. Similarly, these three contrasts were conducted involving the spouses of the above subjects. Thus, these comparisons were made for males and females separately on a relatively large number of variables (14), thereby increasing the risk of obtaining positive results on the basis of chance alone. To counteract this risk, the Bonferroni inequality (Grove & Andreasen, 1982) was employed. This involves choosing an

TABLE II
Descriptive statistics on each measure for each group of couples, for wives (W) and husbands (H) separately¹

	Agoraphobic patients and spouses		Non-phobic psychiatric patients and spouses		Maritally distressed couples		Maritally non-distressed couples	
	Mean	s.d.	Mean	s.d.	Mean	s.d.	Mean	s.d.
<i>Variable set 1</i>								
MMQ Marital Maladjustment	W 11.77	9.58	18.45	10.27	23.07	11.10	6.50	5.00
	H 12.67	8.46	15.27	12.67	21.64	14.56	7.18	5.75
MMQ Sexual Maladjustment	W 8.57	9.13	14.64	10.30	12.79	8.85	4.61	4.51
	H 6.70	6.89	14.27	11.26	14.21	10.30	4.16	4.57
General Marital Dissatisfaction	W 11.27	3.42	14.00	3.97	15.50	4.77	9.87	1.77
	H 11.63	3.38	13.73	3.77	13.71	2.67	10.26	2.94
Sexual Dissatisfaction	W 6.50	2.70	9.27	2.53	8.14	2.41	5.70	1.93
	H 6.37	2.19	9.18	2.64	7.93	1.82	5.87	2.04
Intimate Communication	W 33.33	4.86	31.00	4.58	30.14	5.93	38.86	3.42
	H 33.77	4.16	31.91	4.39	30.57	4.59	38.24	3.31
Destructive Communication	W 28.90	7.60	32.00	5.92	33.14	8.92	26.68	5.13
	H 30.79	7.12	38.45	8.52	35.86	6.54	28.33	5.60
Discongruent Communication	W 11.04	3.60	14.09	3.45	12.57	4.05	9.97	2.47
	H 9.73	2.70	12.09	4.95	10.50	3.32	9.11	2.23
Avoidance of Communication	W 7.23	2.67	8.45	1.29	7.14	2.41	6.00	1.89
	H 7.57	2.30	9.18	2.56	9.43	2.24	6.22	2.02
<i>Variable set 2</i>								
Need for Interpersonal Stimulation	W 19.40	8.63	21.09	6.72	22.21	7.40	26.32	6.39
	H 22.17	6.64	23.45	5.66	23.21	5.73	24.32	6.13
Need for Intimacy	W 12.90	5.18	11.45	4.23	11.00	4.61	10.76	4.95
	H 13.50	4.43	13.73	4.24	12.00	3.46	13.47	4.40
Emotional Dependency	W 25.40	3.56	23.30	4.61	21.21	4.76	22.49	4.57
	H 24.50	4.88	22.64	3.07	22.29	3.45	23.63	4.60
Exclusive Partner-Centredness	W 13.77	1.55	13.18	1.89	11.57	3.61	12.61	2.75
	H 13.07	2.30	13.45	1.37	12.50	1.87	11.66	3.73
Intentional Extra-Marital Involvement	W 42.67	2.32	41.91	5.11	37.86	7.00	39.47	5.55
	H 40.87	4.70	42.73	2.80	40.36	3.00	38.32	5.18
MMQ General Life Maladjustment	W 13.33	7.45	15.09	5.05	12.43	5.47	6.84	4.22
	H 10.33	6.59	8.27	4.36	9.36	2.59	6.71	3.86

¹ A high score for a variable indicates a high degree of that behaviour

overall α (0.20 in this study—that is, a relatively large risk of Type I error—in order to find relationships which could be investigated further) and dividing this by the number of tests (k) to be conducted, to result in an individual level of significance (α_i) for each test. In cases where the data fell into small sub-sets, each of which could be used to study a separate group of hypotheses, the Bonferroni inequality was applied separately to each group.

Similarly, in carrying out within-couple comparisons across groups, the Bonferroni inequality was used in an

analogous fashion. These comparisons were conducted by means of multivariate analysis of variance with a mixed three-factor (Couple $\times$ Sex $\times$ Group) design (Hull & Nie, 1981)

The following subsets of variables were considered.

- (1) the MMQ Marital and Sexual Maladjustment scales, the General Marital and Sexual Dissatisfaction scales, and all four communication scales ($k=8$); and
- (2) the MMQ General Life Maladjustment scale, scales referring to the need for interpersonal stimulation, for

TABLE III

Results of planned comparisons between groups (female patients (P) versus female controls, and husbands (H) of patients versus husband controls), and indices of Effect Size (Cohen's d) for each measure¹

		<i>Agoraphobics and partners versus*</i>					
		<i>Non-phobic patients and partners</i>		<i>Maritally distressed couples</i>		<i>Maritally non-distressed couples</i>	
		<i>t</i>	<i> d </i>	<i>t</i>	<i> d </i>	<i>t</i>	<i> d </i>
<i>Variable set 1</i>							
MMQ Marital Maladjustment	P	-2.27**	0.67	-4.17***	1.09	2.57**	0.69
	H ²	-0.80		-2.99**	0.75	2.42*	0.76
MMQ Sexual Maladjustment	P	-2.24**	0.62	-1.70		2.11*	0.55
	H ²	-2.93**	0.81	-3.16**	0.86	1.42	
General Marital Dissatisfaction	P	-2.43**	0.74	-4.09***	1.02	1.79	
	H ²	-1.88		-2.04		1.78	
Sexual Dissatisfaction	P	-3.36***	1.06	-2.17*	0.64	1.39	
	H ²	-3.74***	1.16	-2.26*	0.78	0.96	
Intimate Communication	P	1.48		2.20*	0.59	-5.06***	1.32
	H ²	1.34		2.51**	0.73	-4.67***	1.19
Destructive Communication	P	-1.31		-1.95*	0.51	1.35	
	H ²	-3.28***	0.98	-2.36*	0.74	1.52	
Discongruent Communication	P	-2.68**	0.86	-1.47		1.34	
	H ²	-2.25*	0.59	-0.80		0.86	
Avoidance of Communication	P	-1.57		0.13		2.29**	0.53
	H ²	-2.07		-2.60**	0.82	2.50**	0.63
<i>Variable set 2</i>							
Need for Interpersonal Stimulation	P	-0.65		-1.18		-3.85***	0.91
	H ³						
Need for Intimacy	P	0.84		1.20		1.78	
	H ³						
Emotional Dependency	P	1.39		3.01**	1.00	2.77**	0.71
	H ³						
Exclusive Partner-Centredness	P	0.67		2.72**	0.79	1.91*	0.52
	H ³						
Intentional Extra-Marital Involvement	P	0.43		2.99**	0.92	2.63**	0.75
	H ⁴	-1.16		0.35		2.30**	0.52
MMQ General Life Maladjustment	P	-0.87		0.49		4.64***	1.07
	H ²	1.21		0.62		3.06**	0.67

1 For purposes of interpretation Cohen (1977) considers a d of 0.20 small, a d of 0.50 medium, and a d of 0.80 large

2 Overall F significant at $P \leq 0.05$

3 Overall F not significant

4 One-tailed t -test (overall F -test not performed)

* $P \leq 0.025$ (Variable set 1) or $P \leq 0.033$ (Variable set 2), ** $P \leq 0.01$; *** $P \leq 0.001$ (one-tailed tests for comparisons involving female patients versus female controls; two-tailed tests for those involving husbands of patients versus husband controls, unless otherwise specified).

intimacy, emotional dependency, exclusive partner-centredness, and intentional extra-marital involvement ($k=6$).

In the case of unplanned comparisons, *a posteriori* contrast tests (two-tailed, $\alpha=0.05$) were performed, the overall F being significant at $P \leq 0.05$.

Results

Employing overall χ^2 or F -tests ($k=6$, $\alpha=0.033$), comparisons between agoraphobics and their female control counterparts, and between partners of agoraphobics and male controls, on the demographic variables mentioned earlier showed systematic between-group differences on education and occupation only ($P \leq 0.01$).

In view of the relatively small size of the non-phobic psychiatric and maritally-distressed samples, the associations of education, occupation (including employment status), religiosity, age at the time of marriage, and child density (the latter two variables were derived from the demographic variables given earlier) with the criterion measures were carefully examined. Employing the Bonferroni inequality, none of these variables were found to be significantly associated with the criterion variables. Analyses were conducted for the separate sexes, and the above conclusions held for both (observed P s > 0.05).

Corroborating previous findings (Arrindell *et al.*, 1983a,b), none of the criterion measures were found to be meaningfully associated with the sub-scales of the MC-SD scale. In addition, no systematic between-group differences were noted in the subjects' tendency towards socially-desirable responding for comparisons involving the separate sexes across groups (see Table I for means); nor were meaningful differences found in the degree of associations which were denoted by the correlations of the MC-SD sub-scales with the criterion variables across samples by sex.

In line with findings from previous studies (Buglass *et al.*, 1977, Arrindell & Emmelkamp, 1985), partners of female agoraphobics were not found to be more neurotic than male controls. On neuroticism, the partners scored as high as partners of other patients and as high as maritally non-distressed males ($P < 0.0001$), who as a group had the highest mean score (Table I). Female agoraphobics were significantly more neurotic than females from both non-distressed ($P < 0.0001$) and distressed ($P = 0.05$) couple groups, and equally as neurotic as their non-phobic psychiatric counterparts (all tests one-tailed).

In the light of the generally moderate correlations between spouses' scores on the different sub-scales used here, the individuals' scores, rather than the couples' joint score, was used (see also Snyder *et al.*, 1981).

Table II sets out the means and standard deviations for each scale for both husbands and wives for each of the samples. Table III gives the results of *t*-test comparisons; indices of effect size (Cohen, 1977) as a means of evaluating the degree of psychological relevancy of the obtained differences are also given.

Agoraphobic patients versus controls

For the second set of variables ($\alpha=0.033$), it was found that agoraphobics as a group could not be distinguished from other patients, although occasionally they differed from either maritally distressed or maritally non-distressed females. However, except for General Life maladjustment, the means of the three control groups overlapped for each variable of this set. On the General Life scale, maritally non-distressed wives scored significantly lower ($P < 0.01$) than each of the other three groups. For the first set ($\alpha=0.025$), the following results were found.

Marital maladjustment and marital dissatisfaction. Agoraphobics as a group rated their marriages as significantly more maladjusted than did non-distressed females, but significantly less maladjusted than did either maritally-distressed or non-phobic psychiatric females. Non-phobic psychiatric subjects rated theirs as being as maladjusted as maritally-distressed females did; that is, the *a posteriori* Newman-Keuls procedure showed that the groups of non-phobic psychiatric and maritally-distressed subjects formed a homogeneous sub-set (i.e. a sub-set of one or more groups, whose highest and lowest means do not differ by more than the shortest significant range for a sub-set of that size).

Agoraphobics experienced their marriages as being as positively reinforcing as maritally non-distressed subjects did, while females from both the maritally-distressed and non-phobic psychiatric couples group described their marriages as more unpleasant than did the agoraphobic patients. Non-phobic psychiatric females reported their marriages as being as unpleasant as maritally distressed females indicated theirs to be (Newman-Keuls test).

Sexual maladjustment and sexual dissatisfaction. Agoraphobics were found to rate their sexual relationships with their marital partners as less adjusted than maritally non-distressed females did, but as significantly more adjusted than those of female non-phobic psychiatric subjects, and as 'adjusted' as the sexual relationships of maritally distressed female psychiatric subjects. The latter two groups had similar mean scores. Furthermore, agoraphobics described their sexual relationships with their partners as being as satisfying as happily-married females described theirs; non-phobic psychiatric and maritally-distressed females rated their sexual relationships with their spouses as significantly more unpleasant than did female agoraphobics with the former two groups forming a homogeneous sub-set.

Communication. Despite significantly greater self-reported intensities of intimate communication by females from the happily-married couples group, as compared with agoraphobic patients, the latter scored significantly higher than maritally distressed females, and as high as non-phobic psychiatric females. Compared with maritally non-distressed females, the marriages of agoraphobics cannot be characterised by greater patterns of destructive communication. Such patterns are more typical of disturbed marriages, as

reported by the female spouses, than of marriages of female agoraphobics. Furthermore, agoraphobics were found to be comparable to their non-phobic psychiatric counterparts in this respect.

Patterns of 'discongruence in communication, as expressed by the spouses, are not significantly more present in the marriages of agoraphobics than in those of either happily married or maritally distressed wives. In fact, agoraphobics reported the presence of such communication patterns in their marriages significantly less often than did non-phobic psychiatric females. The Newman-Keuls procedure further indicated that non-phobic psychiatric subjects differed markedly (Cohen's $d > 1$) from maritally non-distressed wives in this respect.

Agoraphobics reported avoiding communication with their partners significantly more often than did maritally non-distressed wives, although they did not avoid doing so more often than either maritally distressed or non-phobic psychiatric females. The groups contained by the two homogeneous sub-sets which emerged indicated that the difference between happily married and non-phobic psychiatric groups was largest; agoraphobics as a group lay closer to the happily-married females ($d = 0.50$) than did the non-phobic psychiatric group of females ($d = 1.52$).

Partners of agoraphobic patients versus controls

Systematic (overall) between-group differences were observed on all variables, except on those referring to the Need for Interpersonal Stimulation, the Need for Intimacy, Emotional Dependency, and Exclusive Partner-Centredness. All subsequently conducted t -tests were examined two-sidedly, except those involving the variable Extra-Marital Involvement, which were studied one-sidedly.

For variable set 2 ($\alpha = 0.033$), partners of agoraphobics could not be distinguished from those of non-phobic psychiatric patients on Intentional Extra-Marital Involvement or General Life Maladjustment. Despite differences between the partners of agoraphobics and maritally non-distressed males on these variables, the overlap between the latter group and maritally distressed males rendered interpretation of the results somewhat ambiguous. For variable set 1 ($\alpha = 0.025$), the results were as follows.

Marital maladjustment and marital dissatisfaction. Despite the higher scores of husbands of agoraphobics, compared with maritally non-distressed husbands, the former scored significantly lower than maritally-distressed husbands and as high as partners of non-phobic psychiatric patients on marital maladjustment. In terms of the positively reinforcing qualities of the marriage, no differences emerged in comparisons involving the husbands of agoraphobic patients on the one hand and any of the remaining groups on the other. The Newman-Keuls procedure, however, revealed a sizeable difference ($d > 0.80$) between husbands of non-phobic psychiatric patients and those of happily-married females in terms of marital dissatisfaction (the former group scoring significantly higher), and also similar mean scores on marital dissatisfaction for spouses of non-

phobic psychiatric patients and of maritally-distressed females.

Sexual maladjustment and sexual dissatisfaction. While rating their sexual relationships with their marital partners as being as adjusted and as positively reinforcing as happily married husbands did, the partners of agoraphobic patients scored significantly lower than husbands of both maritally distressed and of non-phobic psychiatric subjects. In fact, the Newman-Keuls procedure indicated that two homogeneous sub-sets could be distinguished, the first comprising happily married husbands and partners of agoraphobic patients and the second comprising husbands of maritally distressed and of non-phobic psychiatric subjects.

Communication. The partners of agoraphobics scored significantly lower on intimate communication than the maritally non-distressed males did, but as a group, they scored significantly higher than the group of maritally distressed males and as high as the group of partners of non-phobic psychiatric patients. Again, the latter two groups could not be distinguished from each other on this variable (Newman-Keuls test).

Patterns of destructive communication are not more characteristic of the marriages of partners of agoraphobics than they are of those of maritally non-distressed male subjects. As compared with the reports of partners of agoraphobic patients, such pathological patterns of communication are more clearly present in the marriages of husbands of both maritally distressed wives and of non-phobic psychiatric patients, with the latter group having the highest mean score. Two homogeneous sub-sets were produced: happily married males and partners of agoraphobic patients; and maritally distressed males and partners of non-phobic psychiatric patients.

The partners of agoraphobics scored as high on discongruence of communication as maritally non-distressed husbands did, but also as high as maritally distressed males. By contrast, the former subjects rated discongruence, as expressed by their agoraphobic partners, as being significantly less present in their marriages than did the husbands of non-phobic psychiatric patients.

Compared with maritally non-distressed husbands, partners of agoraphobics had significantly higher mean scores on avoidance of communication, but significantly lower ones than maritally distressed husbands, and equally high mean scores as partners of non-phobic psychiatric patients. The Newman-Keuls procedure indicated the presence of three homogeneous sub-sets: maritally non-distressed husbands; partners of agoraphobics, and partners of non-phobic psychiatric patients and of maritally distressed females. Hence, the latter groups were very similar to each other on these terms.

Within-couple analyses

No significant sex-by-group interaction effects were noted for the sets of variables used in this study (observed $P_s > 0.20$), so that further inspection of differences within

couples by group was not warranted. Since no significant main effects of sex were noted on sub-scales relating to marital and sexual maladjustment and dissatisfaction, it could be concluded that, in each of the groups of couples, spouses were very much like each other in terms of their ratings on these measures.

Discussion

At odds with the views of Webster (1953), Fry (1962), Goldstein (1973), Melville (1977), Holmes (1982) and others, the present findings indicate that female agoraphobics as a group do not: (a) report their marriages as more maladjusted or unpleasant; (b) indicate greater needs for interpersonal contacts and intimacy outside their marriages; (c) appear to be emotionally more dependent on their partners; (d) show greater tendencies towards centredness on their marital partners; or (e) express greater presence of disturbed patterns of communication in their marriages than their non-phobic psychiatric counterparts. Furthermore, at variance with clinical observations by Fry (1962), the partners of agoraphobics as a group do not show evidence of greater degrees of felt distress as a result of their ideated perceptions of the patients having probable intents of performing sexual acts outside their marital relationships than do the partners of non-phobic psychiatric patients. It could be concluded that agoraphobics and their partners were similar to each other in terms of their ratings on marital and sexual adjustment and satisfaction; the same picture emerged for members of the remaining groups of couples.

When compared with non-phobic psychiatric subjects and their partners, there were no special dimensions which characterised the marriages of the agoraphobic patients or their partners in a more disturbed way. On the contrary, both non-phobic psychiatric patients and their spouses were found to score even higher than agoraphobics and their partners on certain measures. On a number of measures (Marital and Sexual satisfaction), no distinctions could be made between agoraphobics (or their partners) and spouses from the maritally non-distressed couples group. By contrast, on certain measures (Marital and Sexual dissatisfaction), no distinctions could be made between non-phobic psychiatric patients (or their partners) and their maritally distressed counterparts, whereas on the same measures, such distinction could clearly be made in comparisons involving maritally distressed husbands and wives on the one hand and agoraphobics and their partners on the other.

The present findings indicate that agoraphobics and their spouses tend to be comparable to happily-

married subjects in terms of some intimacy aspects (marital and sexual adjustment and satisfaction, and certain communication dimensions), while non-phobic psychiatric patients and their partners are comparable to maritally distressed subjects (e.g. compare the indices of Effect Size (Table III), which were generally of moderate to large magnitude).

Thus, in keeping with previous findings by Buglass *et al.* (1977), not one of the initial hypotheses was supported by the present study. While underscoring the need to incorporate non-phobic psychiatric subjects as controls in the study of marital factors in phobic individuals, the present findings also, in conjunction with those of Buglass *et al.* (1977), Cobb *et al.* (1984), and Arrindell & Emmelkamp (1985) on the psychological profile of the spouse of the agoraphobic patient, underline Symonds' (1973) view that it is necessary to analyse the patient's psychological make-up rather than the marriage or the husband, "since the problem lies not in the marriage, nor in the husband, but within the patient herself." This is not to say that there may not be individual cases in which marital problems serve to complicate agoraphobic suffering, but although important in clinical practice, such individual observations cannot be used as a basis for making general statements concerning the quality of the marital relationships of agoraphobics as a group.

Reviews of psychosomatic illness and family function have suggested that marital pathology may play a role in the selection and perpetuation of symptoms (e.g. Waring, 1977). Research evaluating the marriages of patients who suffer from hypochondriasis, chronic pain, obesity, and myocardial infarction demonstrates marital maladjustment, sexual dysfunction, and emotional illness in the spouse (Waring, 1983). While our results support comparable marital factors in both non-phobic psychiatric patients (marital maladjustment, marital dissatisfaction, and sexual dissatisfaction) and their partners (marital and sexual maladjustment and dissatisfaction, lack of intimate communication, destructive communication, and avoidance of communication), no such support could be found in agoraphobics or their partners. This lack of support in agoraphobia, found here and in the Buglass *et al.* (1977) study, may lie in the fact that on the whole, clinicians have relied on a strikingly conservative working method (the unstandardised one-to-one therapeutic or diagnostic interview), compounded by a concentration on fairly narrow (biased) segments of the population of agoraphobics. In Hafner's (1982) view, "the great majority of agoraphobic patients discussed by therapists in clinical and anecdotal studies have entered and persevered with

some form of psychotherapy, often as private patients, and may therefore be unrepresentative with regard to personality and socioeconomic factors." In this area, sample representativeness with respect to crucial variables is often puzzling.

A problem related to most of the studies pointing to a marital factor in agoraphobia is that the subjects comprising the samples described were chosen and treated by the authors themselves. The experimenter as therapist is a serious methodological problem in marital treatment research (O'Leary & Turkewitz, 1978); the emotional investment and enthusiasm of the author/therapist may not only increase placebo effects and spuriously inflate the success of treatment under evaluation, but may also lead to selective sampling and description of patients. Unfortunately, such selective procedures may lead to serious prejudices and myths about human behaviour that may be difficult to erase (Adelson & Doehrmann, 1980; Hare-Mustin, 1983).

The similarity found here between spouses—particularly those in the maritally distressed and

happily married couple groups—in terms of their ratings on marital adjustment and satisfaction, supports the internal validity of the present study. Marital satisfaction studies tend to demonstrate serious discrepancies between the husband's and the wife's description of the same relationship (Waring, 1982).

It should be borne in mind that only female agoraphobics participated in the present study. It would thus seem advisable to examine this issue employing female partners of male agoraphobics as subjects, since the findings do not necessarily apply to such a group. Nor would they apply to agoraphobics in same-sex relationships.

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Prevalence of Schizophrenia in an Urban Population

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The treated one-year prevalence and point-prevalence of schizophrenia, schizoaffective psychosis, and paranoid state in the city of Salford were derived from the Salford Psychiatric Case Register. The high rates found are attributed primarily to the effects of selective migration from the area. A breakdown of the use of health services in the area by patients with these diagnoses is given; hospital care was found to be the major form of treatment.

Scientific investigation of schizophrenia, as of other forms of major psychiatric disorder, has been much concerned with its epidemiology, yet great uncertainty remains about this aspect of the condition. Variations in diagnostic practices have been responsible for much of the lack of agreement on rates of morbidity, although the introduction of instruments of assessment and diagnosis which can reliably be used cross-nationally, such as the Present State Examination (PSE) (Wing *et al*, 1974) has achieved much more consistency. Other problems, however, have resulted from the use of non-standard populations, e.g. the inclusion of those aged under 15, who are not significantly at risk, or from confusion between point-prevalence and that calculated for varying periods.

The legitimacy of comparing rates from societies at different stages of industrial development has also been questioned. For instance, in poorly developed countries, those affected by psychosis may die more quickly than elsewhere, thus appearing to reduce the rate, or there may be more organic psychoses, which are confused with schizophrenia (Leff, 1981). Both Torrey (1980) and Warner (1985) claim to have elicited striking differences in the data on prevalence of schizophrenia between highly industrialised and other countries, with much lower rates in the latter, so that 'civilisation' is then identified as an important aetiological factor. However, the WHO cross-national study of incidence (Sartorius *et al*, 1986) confirms the previous consensus view that the rates at which new cases appear in any population are very similar world-wide; what happens subsequently to these cases, though, would be expected to vary – at least to some extent – with cultural, economic, and other local conditions. Finally, the effects of migration are often complex (Giggs, 1985), but might perhaps account for substantial differences between areas in their prevalence of schizophrenia.

In industrialised countries, prevalence rates are especially important due to their implications for the

provision of services, since schizophrenia makes long and heavy demands on these, being a mainly chronic disorder of intermittent and variable duration. Hence, rates need to be calculated for geographically defined populations, which has the advantage of ordering the data along a single axis, regardless of other characteristics of the study design (Jablensky, 1986). Although 'true' prevalence could only be determined by examining every member of a defined population with a standard diagnostic instrument (and then only for one point in time), 'treated' prevalence might give a reasonable approximation to it in certain situations. The value of a treated prevalence figure should be enhanced if special attention has been paid in that place to record keeping, and particularly if data have been consistently collected for some years from service facilities, with a view to providing material for research; that was the case in the study reported here.

Background

The historical and social background relevant to this study has already been reported (Freeman, 1984). Salford, in the north-west of England, was essentially a small market town until the arrival of industrialisation in the late eighteenth century. It then experienced an astonishing growth of population, mainly from migration, but with both birth and death rates at high levels, for the rest of the nineteenth century. With an area of less than eight square miles, its population continued to rise to a peak of almost a quarter of a million, in the late 1920s; since then, the figure has fallen steadily to under 100 000 in 1981, and continues to fall. This reduction has also resulted mainly from migration (both voluntary and enforced through redevelopment). Birth and death rates have been much lower in recent years than in the earlier part of this century. The migration has been of a differential nature, consisting mainly of complete families and leaving behind relatively more of the old, of single-parent families, of one-person households, and of the physically or mentally disabled. In terms of socio-economic status, the population is overwhelmingly working class, and since the greatest part of the housing stock is owned by the

City Council, their administrative policies can be very influential on the kinds of people who occupy different homes. For instance, since high-rise flats are hard to let, they may be made available to previously long-stay patients who are being discharged from hospital.

The Psychiatric Case Register for Salford was established in 1968, with the financial support of the Department of Health & Social Security (Fryers *et al.*, 1970; Wooff *et al.*, 1983); it has been particularly useful as a database, from which studies such as the present one can be derived. In 1974, the Salford County Borough (CB) was approximately doubled in size, to form a new Metropolitan District, but data which referred to the former city area could still be identified separately, and these have been used in the study reported here.

Method

The treated one-year prevalence of schizophrenia was derived from the following population, as provided by the Salford Psychiatric Case Register:

- (a) all persons with a home address in Salford CB in contact with the mental health services on 31 December 1973 (point-prevalence)
- (b) all such persons who came into contact with these services during 1974.

Since 1 January 1968, the Salford Case Register has collected psychiatric and social service data on all persons with a home address in Salford treated by any of the mental health agencies, but excluding treatment from general practitioners (GPs). To establish the number of people with identifiable schizophrenic illness, the Syndrome Check List (SCL) was utilised (Wing *et al.*, 1974). This involved reviewing psychiatric case histories for all patients within the population base who had certain specified clinical diagnoses, as recorded on the Register for 1974. The symptomatology obtained from each case history was rated, coded according to the SCL criteria, and then processed using the CATEGO computer program. Since this was an analysis of patients who had become ill over many years, and since the majority were on maintenance medication, it would not have been feasible to use the PSE on the whole sample at that time. Frank psychotic symptoms would be expected in only a minority of patients (Cheadle *et al.*, 1978); also, since the study was carried out retrospectively, it would not in any case have been possible to obtain PSE data for all patients for 1974. However, PSE data deriving from an earlier investigation were available for some of the patients in the study group, and relevant information from this source was incorporated into the study.

Base data

Statistical data on patients in contact with the services at 31.12.73 were provided by the Register's annual listing of point-prevalence for 1973 ('Census 1973'). This embraces the following categories of contact with the psychiatric services:

- (a) long-stay hospital in-patients: one year or more of continuous stay (including transfers between hospitals, within the same episode of treatment)
- (b) medium and short-stay hospital in-patients: 1-364 days' continuous stay
- (c) day hospital and other day centre patients (currently registered at 31.12.73 and actually attending)
- (d) out-patients with attendances both before and after the census point, with not more than three months between attendances
- (e) patients receiving domiciliary visits from psychiatrists, or visits in general hospital wards on 31.12.73, or both, before and after that date, with not more than three months between visits
- (f) patients currently in contact with social workers at 31.12.73 and defined as having mental health problems, according to the Register criteria
- (g) patients on the books of community psychiatric nurses (CPNs) at 31.12.73.

A second annual listing ('New Episodes 1974') provided equivalent data for psychiatric patients other than those already included in Census 1973 who came into contact with the services during 1974. The New Episodes listing included both patients making a 'first ever' contact with the psychiatric services during 1974, and patients who had had previous contact with the services, but who were not eligible for inclusion in Census 1973.

These two listings provided the base for the initial identification of patients to be included in the initial study group.

Diagnostic categories included in the initial study group

The initial aim of the study was to identify patients with the clinical diagnoses of schizophrenia, schizoaffective psychosis, or paranoid state who were in contact with the psychiatric services at 31.12.73 or during 1974, and to check these by a standardised procedure, in an attempt to establish how many of these diagnoses could be regarded as valid. At an early stage, it was decided that some attempt should also be made, using the same standardised technique, to identify the incidence of schizophrenic illness among patients with other diagnoses, recorded on the same two Register listings. It would have been impracticable to have reviewed the case histories of all patients within the Register base population. However, the diagnosis recorded on the Register is that given by the senior clinician in the Salford psychiatric service who is responsible for the patient's initial episode of care, and in practice, these diagnoses were made by a small number of psychiatrists who had worked together for some time and shared similar orientations and training. Since these diagnoses were made for clinical purposes, however, they could not be expected to have the degree of rigour required for a research exercise. The Register has a mechanism for altering diagnosis, if this seems to be necessary when the patient makes a subsequent service contact.

An attempt was therefore made to identify those categories considered most likely to be used in difficult

borderline cases, or when mis-diagnosis occurred, or when patients previously diagnosed as schizophrenic subsequently presented with non-schizophrenic symptoms. Initially, these other diagnostic categories consisted of certain functional psychoses, as well as paranoid and schizoid personality disorders. As the study progressed, however, it became apparent that further groups should be incorporated; the original list was therefore expanded to include all other personality disorders and functional psychoses, and the final list of diagnostic categories covered was: schizophrenia (including schizophrenia, schizoaffective psychosis, paranoid state, and morbid jealousy); mania (including hypomania); severe depression; depressive delusions and/or hallucinations; history of mania and depression, history of mania and depression and/or hallucinations; psychosis, not otherwise specified; personality disorders.

The two Register lists comprised 924 names, a few of which were later found to be ineligible because they did not have a home address in Salford CB during 1974. The final list of the initial study group consisted of 909 patients, of whom 553 were in contact with the psychiatric services at 31.12.73 and the remaining 356 came into contact during 1974. This method makes the assumption that a patient's diagnosis did not change during the study period, however, it should not be taken to mean that every subject was suffering from schizophrenia *throughout* the year.

Expansion of the study group

It became apparent that in any one year, a few known schizophrenics might have been in contact with the psychiatric services but, being free of psychotic symptoms, be given a Register diagnosis (e.g. depression) other than schizophrenia or one of the other diagnostic groups listed above. At that time, the diagnostic hierarchy of the Register was not always strictly observed (although this was corrected shortly afterwards). To ensure that such patients would not be missed, it was decided to include in the study any individuals who had appeared on the Register's annual prevalence listings before 31.12.73 with a diagnosis of schizophrenia, schizoaffective psychosis, or paranoid state, and who were in contact with the defined psychiatric services in 1974, but with a diagnosis for that year which did not fall into any of the groups listed above. It was also decided to include any patients who appeared on the Register's subsequent annual prevalence counts (up to 31.12.79) with a diagnosis of schizophrenia, schizoaffective psychosis, or paranoid state, but who at 31.12.73 or during 1974 were recorded with a diagnosis other than those listed above, the number of these was very small. However, no patient data from later than 31.12.74 were included in the assessment.

At the same time, a comparison of lists of patients receiving depot neuroleptic injections from CPNs in 1973-74 with the Register's annual prevalence listings for 1974 revealed that some patients known to be receiving injections at this time were not included in Census 1973 or New Episodes 1974. The CPN service was then in its early stages in Salford, and Register recording of CNP contacts at that

time was known to be incomplete. In an attempt to identify these and any other eligible patients who may not have been included in the Register's annual prevalence for 1974, listings were made of all patients with diagnoses of schizophrenia, schizoaffective psychosis, or paranoid state from all annual point-prevalence and new episodes lists from 1.1.68 to 31.12.79 (the latest list available at that time). Those patients who appeared on annual prevalence listings both before and after 1974, but not in 1974, were identified; they numbered 134. These procedures also revealed that a further 260 patients with a diagnosis of schizophrenia, schizoaffective psychosis, or paranoid state appeared on Register annual prevalence listings before 31.12.73 but not subsequently, and 149 appeared after 31.12.74 (up to 31.12.79), but not previously. The Register manual record cards for all these patients were therefore examined, to ascertain whether any were, in fact, in contact with the psychiatric services during 1974, and all found to be eligible were incorporated into the study group.

These procedures resulted in the identification of an additional study group of 150 patients; together with the initial study group, this gave a total of 1059. Of the 150, 53 were at this stage known to have been in contact with the mental health services in 1974. For the remaining 97 patients, the following data sources were examined, to establish whether or not they had had contacts in 1974: hospital and social services case notes; information about CPN injections not yet transferred to the Register; and injection record cards at the hospitals.

Those remaining patients who were not positively identified as having contacts with the psychiatric or social services during 1974 could have been receiving maintenance treatment from GPs in 1974; it was decided to retain them within the study, but to identify them separately. Investigation of case notes had already established that some of these patients had in fact received such treatment (initiated by the specialist psychiatric services). Further information was then sought from GPs about the remaining 42 patients; those for whom there was no positive evidence of such treatment were then eliminated from the study.

Coded information on a large number of the patients was then cross-checked with the results of PSE interviews on the same patients, carried out for a previous study (Cheadle *et al.*, 1978). Additional information from the interviews was incorporated into the present study, and in a number of cases confirmed the presence of symptoms for which there was insufficient positive evidence in the case notes, and which were necessary to give a CATEGO classification in one of the schizophrenia groups. The opportunity was also taken to make further checks on the data collection and coding, using any additional information which had come to light; e.g. through enquiries at other hospitals, or from data collected over a number of years on patients treated by depot neuroleptics.

Attempts were made to examine and summarise all available hospital and social service case notes for the 1059 patients in the study group. Notes for 18 patients had been lost or destroyed, but only three of these had a diagnosis of schizophrenia or paranoid state recorded on the Register, and none had had an extensive history of contacts with the psychiatric services.

Results of classification

The following CATEGO classifications (Wing *et al.*, 1974) were regarded as likely indicators of schizophrenic or paranoid illness:

- (a) schizophrenic psychoses: S+, S? (subclasses NS+, NS?, DS+, NSMN/DSMN, NSPD, DS?)
- (b) paranoid psychoses: P+ (subclasses DP+, DPMN, DPPD)
- (c) borderline or doubtful psychoses: O+ (subclasses CS+, CSMN, RSMN, RSPD, CSPD).

For all cases which received one of these classifications, the CATEGO result was compared with the clinical diagnosis, as recorded on the Register. In discrepant cases, where the Register diagnosis was other than schizophrenia or paranoid state, the case note summaries and codings were re-examined, and if necessary the ratings amended and reprocessed. At this stage, all cases ($n=16$) recorded as having organic aetiologies, or where alcohol or drug involvement were considered to be the primary feature of the illness, were excluded from the 'schizophrenia' group.

Where the CATEGO processing resulted in classifications of P? (subclasses DP?, DP?/AP?) or O? (subclasses RS+, RS?, SS, UP+, UP?, XP), the case note summaries were also re-examined, and if possible additional symptom information was sought. Cases which were then still considered as doubtful were not included in the 'schizophrenia' group.

A further investigation of clinical evidence was also carried out for those patients with a Register diagnosis of schizophrenia, schizoaffective psychosis, or paranoid state, but who did not emerge from the computer processing with one of the S, P, or O classifications listed above. Again, if necessary, symptom ratings were revised and reprocessed.

Finally, a group of 588 patients was identified as forming a 'main schizophrenia group' (including paranoid states), and an additional 36 patients formed a 'borderline group'; 538 patients in the main group and 32 in the borderline group had had a Register diagnosis of schizophrenia, schizoaffective psychosis, or paranoid state in 1974. A further 100 patients in the study group had Register diagnoses in the schizophrenia/paranoid groups, but were not confirmed as satisfying our criteria for those categories. Fifty patients in the main group and four in the borderline group did not have a Register diagnosis of schizophrenia, schizoaffective psychosis, or paranoid state relevant to 1974. However, further examination of these patients' records revealed that almost all of them had either at some time other than in 1974 been recorded on the Register as having diagnosis of schizophrenia or paranoid state, or had a clinical diagnosis in these groups which had not been recorded on the Register. They were therefore included.

All case-note assessments were carried out by the same person.

Results

Final allocation of cases to the main and borderline groups.

Analysis by the CATEGO major classes and by Register diagnosis respectively showed a considerable consistency of diagnosis, when the two methods were compared (Table I).

Out of 588 patients in the main group in the study, 50 had received other diagnoses on the Register; 100 patients who had received a diagnosis of schizophrenia, schizoaffective psychosis, paranoid state, or morbid jealousy in the Register did not have that diagnosis confirmed by this study.

The results given below are for the main and borderline groups combined.

Nature of contact with services. For the one-year period, there were a total of 624 subjects, of whom 546 were in contact with specialised psychiatric services, as defined by the Register, 52 were in contact with their GP only, and 26 were in contact with social services only. The point-prevalence numbers for 31.12.73. were 404 for the specialist services and 14 for social services only; no figure could be obtained for GP contact only.

The same figures have been calculated in terms of rates per thousand adult population (aged over 15) and per thousand total population, the addition of the borderline group made a difference of 0.4 per thousand to the rates for total annual period prevalence. Thus, all forms of care account for 6.8 per thousand adult population, over one year.

In-patients The point-prevalence data (31.12.73) showed that patients resident for 24–44 years were the largest subgroup, representing 42% of the in-patients and 15% of the total one-year prevalence. The next largest group had been resident for 10–24 years, representing 24% of all in-patients and 8.5% of all patients. The two groups resident for 1–4 years and 5–9 years each contributed about 10% of the total in-patients, while the groups of under one year and of 45 years or more were each about 7%.

Nine long-stay in-patients died during 1974; eight of these had been in hospital for 15 years or more, and five were aged 75 or over. Thirteen long-stay in-patients were discharged during 1974; all except three were aged 45 or over, and four had been in hospital for at least 35 years. Only one long-stay in-patient was aged under 25, whereas 38.5% were aged 65 and over. In terms of population rates, all in-patients represented 2.45 per thousand of the adult population and 1.86 per thousand of the total population.

The total number of in-patient days for patients with a confirmed diagnosis of schizophrenia was 73 909, compared with 64 106 for all other patients. The average length of stay for the former was 137.4 days, and for the latter 42.6 days.

New cases in 1974. Thirty-five cases had their first contact on the Register in 1974. Of these, 19 were known to have had some previous psychiatric care, nine cases were definitely known not to have had such contact before, and in seven cases this information could not be determined.

Age and sex distribution. The male rate (7.02 per thousand adult population) is somewhat higher than the female

TABLE I
Treated prevalence of schizophrenia in Salford CB, 1974: final allocation of cases to main and borderline groups, analysed by CATEGO major classes and by case register diagnoses

Catego major class	Description	Schizophrenia, Schizoaffective psychosis, paranoid state				All other diagnoses				Total			
		Main group	Borderline group	Excluded	Total	Main group	Borderline group	Excluded	Total	Main group	Borderline group	Excluded	Total
S+	Schizophrenic psychoses	379	8	10	397	35	2	3	40	414	10	13	437
S?		22	1	—	23	—	1	—	1	22	2	—	24
P+	Paranoid psychoses	41	7	4	52	7	1	3	11	48	8	7	63
P?		30	8	11	49	4	—	21	25	34	8	32	74
P/R+		—	—	2	2	—	—	1	1	—	—	3	3
O+		47	4	4	55	4	—	2	6	51	4	6	61
O?		8	—	2	10	—	—	7	7	8	—	9	17
O/N+	Borderline and	1	1	1	3	—	—	7	7	1	1	8	10
O/N?		1	1	1	3	—	—	—	—	1	1	1	3
O/R+	doubtful psychoses	4	—	3	7	—	—	11	11	4	—	14	18
O/R?		4	—	1	5	—	—	—	—	4	—	1	5
O/B+		1	—	—	1	—	—	1	1	1	—	1	2
O/XP		—	—	1	1	—	—	—	—	—	—	1	1
M+	Manic and mixed affective psychoses	—	—	14	14	—	—	20	20	—	—	34	34
M?		—	—	9	9	—	—	7	7	—	—	16	16
D+	Depressive psychoses	—	1	20	21	—	—	32	32	—	1	52	53
D?		—	—	1	1	—	—	5	5	—	—	6	6
R+	Retarded depression	—	1	9	10	—	—	34	34	—	1	43	44
N+	Neurotic depression	—	—	4	4	—	—	90	90	—	—	94	94
N?		—	—	1	1	—	—	8	8	—	—	9	9
H	Hysterical conditions	—	—	—	—	—	—	2	2	—	—	2	2
X	Other conditions	—	—	—	—	—	—	16	16	—	—	16	16
NO	No abnormalities	—	—	2	2	—	—	8	8	—	—	10	10
Total		538	32	100	670	50	4	278	332	588	36	378	1002

TABLE II
Age and sex of patients (main and borderline groups) for annual prevalence, 1974

Age group	Age and sex-specific rates ¹ per 1000 population		
	Males	Females	Total
5-24 years	2.17	1.14	1.67
25-44 years	8.33	5.77	7.08
45-64 years	9.50	10.52	10.03
65 years & over	6.47	6.98	6.80
Total annual treated prevalence	7.02	6.61	6.80

¹Including patients with paranoid states

(6.61), but females had a slightly higher rate in the age-group 45 and over. Among age-groups, the highest rate was for those aged 45-64, with almost identical rates in the 25-44 and 65-and-over groups; the rate for those aged 15-24 was very low (Table II).

Comparison with general population. The estimated population, aged 15 and over, of Salford CB in 1974 was 91 700. Those aged 65 and over comprised 18%, almost exactly the same as their proportion in the total one-year period prevalence of schizophrenia, but the age-group 15-24 represented 21.5% of the adult population, compared with only 5.5% of the one-year prevalence. The 24-44 age-group was 28.5% of the general population, which was almost the same as its proportion of the prevalence (29.5%), but the 45-64 age-group was only 32% of the population, compared with 47.5% of the one-year prevalence.

TABLE III
Treated prevalence of schizophrenia¹ in Salford CB. statistics on service usage for 1974

Service usage	Main group only	Main and borderline group
In-patients at 31.12.73		
<i>n</i>	211	225
% of group total	35.9%	36.1%
Other patients admitted in 1974		
<i>n</i>	78	84
% of group total	13.3%	13.5%
Total in-patients		
<i>n</i>	289	309
% of group total	49.1%	49.5%
In-patient admissions (including readmissions)	98	107
Total in-patient days	73 909	78 740
Day patient days	7 500	8 231
Patients with day patient attendances	40	43
Out-patient attendances	484	520
Patients with out-patient attendances	179	192
Domiciliary visits	10	13
Patients receiving domiciliary visits	10	12
Patients receiving out-patient depot injections	171	180
Patients on the books of social workers (mental illness referrals) ²	113	123
Total days on the books of social workers (mental illness referrals) ³	16 174	17 214
Patients who died	12	13
Patients known to have moved from the area	7	7
Patients with GP contact only	50	52
Patients in contact with the psychiatric services	538	572
Total patients	588	624

¹Including paranoid states

²Including 6 with number of days not known

³Excluding those mentioned in note 2

Marital status Single patients represented 54% of the total annual period prevalence, but 69% of the long-stay in-patients, and 72% of those in contact with social services only. The exact rate of unmarried adults in the general population of Salford for 1974 is not known, but is much less than any of these percentages.

Domestic unit. At the time of inclusion in the study, 33.5% of subjects were in hospital, 24.5% with family of marriage or cohabitee, 21.5% with parents, siblings or children, 10% living alone, 6% in hostels, groups homes, or elderly person's homes, and 5.5% not known or in other situations. The percentage of males living with parents (24%) was much greater than that of females (9.5%). Only 14% of males were living with a spouse or cohabitee, compared with 35% of females. Sex differences in the other categories were negligible.

Service usage. Table III summarises the use of different categories of service

Comparison of service usage There were 538 schizophrenic patients in contact with the psychiatric services during 1974, compared with 1503 patients with all other diagnoses. Mean hospital in-patient days per schizophrenic patient (137.4) were more than three times those for all other diagnoses (42.6%), while total in-patient days for schizophrenics (73 909) were substantially more than those for all other diagnoses (64 106). Day-patient days (7500) were about one-third those for all other diagnoses (22 300). Combined out-patient attendances and domiciliary visits (494) were less than one-fifth of the total for all other diagnoses (2754). Schizophrenic patients had 16 174 days on the case-books of social workers, compared with 43 724 for patients with all other diagnoses.

Discussion

The study measured morbidity through service contacts—a method proposed by Odegaard (1946), on the assumption that when a psychiatric service is readily available to a whole population, any member suffering from a severe mental disorder will make contact with the facilities sooner or later. This view was supported by data from the Danish island of Samsø, where the service has been free and accessible over a number of years; referral to the specialist facilities was taken as the criterion of 'caseness' in the epidemiological studies there (Stromgren, 1968; Nielsen, 1976). Jablensky (1986) states that in spite of the well documented presence of unrecognised psychiatric morbidity in the community, the importance of epidemiological investigations of treated (or 'declared') cases has not diminished: "surveys based on service contacts can answer questions about the

pathways leading to treatment and management, about the social and personal consequences of being diagnosed as mentally ill, and also, in areas of sufficient density of treatment facilities, questions about incidence and prevalence. A carefully designed survey of service contacts can be a reasonable approximation of a door-to-door survey". Salford readily satisfies Jablensky's requirement of density of treatment facilities (Freeman, 1975).

The rates of 'declared' schizophrenia found in this survey are high, relative to most published figures. Cooper (1978) summarised the results of 15 investigations from 12 countries, carried out between 1929 and 1972, which gave one-year prevalence rates between 1.5 and 4.2 per thousand total population, but most were in the range 2.0–3.5 per thousand. Jablensky (1986) recorded 19 surveys in Europe based on service contacts (although one dealt only with depressive disorders); his conclusion, from this and other forms of epidemiological enquiry, is that "the most striking finding concerning schizophrenia is the relatively good agreement between the rates of prevalence, incidence and morbidity risk obtained in different surveys". This relative concordance of results is despite much lack of unanimity about the operational definition of schizophrenia.

The association of these relatively high rates with the predominantly working-class population of Salford CB is consistent with previous studies showing an excess of schizophrenia in lower social classes; however, this is unlikely to be an aetiological association, as Kohn (1974) and Warner (1985) have claimed. The data on marital status are also in agreement with previous findings; however, in most industrialised countries there are now significant numbers of couples living in stable unions, although unmarried. No data were available on cohabitations, but the number in this sample is believed to have been very small.

The results of the present study emphasise that large samples of schizophrenics, derived from whole populations, are predominantly middle-aged. This is consistent with the condition being one of relatively low incidence (mainly at a young age), but of high prevalence due to its low mortality and its tendency towards a chronic course.

Very high prevalence rates, similar to those reported here, have also emerged from Ireland: the point-prevalence figure for three mainly rural counties was 8.3 per thousand of the adult population (Walsh *et al*, 1980). In addition to possible effects of differential migration, other suggested influences there were "historic-cultural characteristics such as an over-generous provision of in-patient facilities, a passive-submissive tendency on the part of patients

to accept hospitalisation, a paternalistic attitude and practice on the part of psychiatric personnel, and a lack of involvement of primary health care personnel". Murphy & Vega (1982) reported an excess of first admissions for schizophrenia among Catholic males (both in Northern Ireland and Eire), which declines along the geographical axis from south-west to north-east; the percentage of unmarried males in the western Irish counties is double that in the eastern. There is no reliable evidence, however, of a higher-than-average incidence of schizophrenia in Ireland (Ni Nuallain *et al*, 1984).

Jablensky (1986) states that no clear-cut support has been found for the hypothesis that geographical rates of schizophrenic morbidity are affected by negative selection, through the out-migration of healthier individuals. However, it is noteworthy that County Roscommon showed a 50% fall in population during an earlier period of 50 years (O'Hare & McHugh, 1982), which was the same as that for Salford CB in the 50 years from 1928. One statistical effect of such population decline is that the long-stay in-patient rate will be artificially high, because those patients who have been in hospital for many years derived from a much larger population than the current one. Although people migrate for many reasons, both voluntary and involuntary, it would seem likely that areas which have become economically and culturally demoralised (as both western Ireland and inner city Salford could be regarded as being) will lose relatively more of their healthier people. Those left behind represent what Strömberg has described as a 'residual population', in which many kinds of disability have a high prevalence; the opposite situation is found in suburban areas or new towns.

The results of this survey give totals for the use of all categories of psychiatric care by every person suffering from schizophrenia in the defined population, over a period of one year. The heavy demands made by schizophrenia in the hospital care of psychiatric disorder from this particular population are manifest

in the data. The part played by day care in the treatment of schizophrenia is surprisingly small, considering the value widely placed on this for chronic and recurrent psychiatric disorders. Less than one-twelfth of the total identified cases had no contact with any specialist service during the year (i.e. they received GP care only). The contribution of social services was also relatively modest: about one-fifth of the total were in contact with a social worker during the year, and a small number of patients received day care at a local authority centre. A study of chronic schizophrenic patients living outside hospital in Salford in 1976 (Freeman *et al*, 1979) confirmed this picture, and showed that the extent of social service care for any patient tended to be in the same proportion as care from the hospital-based services. At the time of the present study, therefore, the care of schizophrenia in this population was largely the responsibility of the National Health Service psychiatric facilities. Subsequently, the CPN service has been substantially developed, but this was largely in addition to the pre-existing services (Wooff *et al*, 1983).

By pricing each form of care, it would be possible to calculate the total cost to public funds of this condition for a total population – something which has not been done up to now. Results have also been calculated separately for electoral wards in Salford CB, and these small-area variations will be published elsewhere. On the basis of this study, it seems reasonable to propose that predominantly working-class industrial communities that have suffered considerable population loss will show prevalence rates of schizophrenia that are substantially higher than accepted averages for developed countries.

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HLA Antigens in Schizophrenia: A Family Study

R. CHADDA, P. KULHARA, T. SINGH AND S. SEHGAL

HLA haplotypes A and B were studied in eight families with two or more schizophrenic members. All index cases and all but two sick relatives satisfied Research Diagnostic Criteria for the diagnosis of schizophrenia; 45 subjects were studied. Data were analysed by sequential tests for the detection of linkage and by the affected sib-pair method. The results do not support a linkage between HLA and schizophrenia.

There is considerable evidence that genetic factors contribute significantly to the aetiology of schizophrenia. Though various putative modes of transmission, ranging from Mendelian dominant/recessive inheritance to polygenic inheritance, have been suggested, the exact mode of transmission remains an enigma (Slater & Cowie, 1971; Gottesman & Shields, 1982).

The possibility of locating a genetic marker in schizophrenia offers an opportunity of opening up the entire field of genetic research to further scientific enquiry. A genetic marker is an inherited characteristic which is polymorphic in nature, has a known mode of transmission, and can be assigned or potentially assigned, a precise chromosomal location. It should be specific, readily detectable, and unaffected by the state of illness or recovery (Watt, 1982; McGuffin *et al*, 1983). A trait is 'polymorphic' when it exists in two or more forms, with each genetic variant existing at a frequency of 1% or more in the general population.

Recently, human leucocyte antigens (HLA) have been demonstrated to be associated with various disorders, mostly of a chronic nature – e.g. ankylosing spondylitis, juvenile diabetes, and multiple sclerosis (Svejgaard *et al*, 1975; Thomson, 1981) – all of which have an uncertain aetiology and pathogenesis. Since there are certain similarities in this respect between these disorders and schizophrenia, HLA has been studied in relation to that condition.

Two approaches have been used: in some studies, the incidence of various HLA antigens in the general population has been compared with that in schizophrenic patients (Cazzullo *et al*, 1974; Smeraldi *et al*, 1976; Crowe *et al*, 1979; Miyanaga *et al*, 1984). The results are diverse and conflicting, but there appears to be greater agreement when the disorder is divided into sub-types (McGuffin *et al*, 1981; Ivanyi *et al*, 1983). The other approach, utilising a pedigree paradigm, has attempted to demonstrate linkage

between HLA and schizophrenia (Turner, 1979; McGuffin *et al*, 1983). Here again, the results are conflicting, with Turner (1979) finding evidence for, and McGuffin *et al* (1983) against linkage.

The present study was undertaken with the aim of exploring the relationship between HLA and schizophrenia.

Method

Selection of families

To select suitable families, case records of patients diagnosed between January 1980 and December 1983 as schizophrenic were scrutinised, and those patients who had a positive family history of schizophrenia in one or more living parents or siblings were identified. In addition, colleagues were requested to refer any such patients.

Families were selected for linkage analysis if they contained at least one living parent and two offspring. To study only familial psychosis and avoid inclusion of 'sporadic' forms of the disorder, attempts were made to select large families with two or more schizophrenic members. Eight families were thus identified and found suitable for the study. Many other families, though suitable for linkage analysis, were not included because they were not approachable, or because not enough consenting members were available.

Diagnosis

Probands and all the available relatives were interviewed. All index cases satisfied Research Diagnostic Criteria (RDC) for the diagnosis of definite schizophrenia (Spitzer *et al*, 1978). Relatives who fulfilled RDC for definite schizophrenia or matched the description of schizophrenia spectrum disorder of Kety *et al* (1968) were designated as 'sick', while all others were considered to be psychiatrically healthy for the purpose of the investigation.

Of the available total of 64 subjects in the various families, HLA typing was carried out in 45; of these, 16 were classified as RDC schizophrenia and two as schizophrenia spectrum disorder.

HLA typing

All subjects were called to the Department of Immunopathology, and HLA typing A and B was carried out on the same day, using fresh venous blood. Antigens C and DR were not typed, due to lack of facilities. A standard method of micro-lymphotoxicity test, described by Terasaki *et al* (1974), was employed.

Linkage analysis

Sequential tests for detection of linkage (Morton, 1955). This method consists of determining the probability of an observed pattern of segregation of two characters in a family for a given range of possible recombination fractions (θ), where θ varies from 0 to 0.5. Dividing the probability for a particular value of θ by the probability for a value of 0.5 gives the relative probability or odds ratio at the value of θ . The log of this ratio gives the 'lod' score (log of the odds). Linkage may be tight or loose; tight if the recombination fraction is low, loose if it is high. A value of 0.5 indicates no linkage. The recombination fraction that gives the highest probability or lod score is also the best estimate of the degree of linkage between two loci.

The lod score method, as devised by Morton, is meant to be applied to regular Mendelian phenotypes, whereas for any plausible single gene model of schizophrenia, the notion of incomplete penetrance needs to be invoked. Incomplete penetrance causes a systematic bias against detection of linkage (McGuffin *et al*, 1983).

Affected sib-pair method for detecting HLA-linked genes in disease (Green & Woodrow, 1977). This test requires no assumption about the mode of inheritance of the disorder, and is not affected by errors due to variation in age of onset, as only the individuals already affected with the disease are studied. It can be used only for families in which there are two or more siblings suffering from the illness.

If, in a sibship of two, a particular HLA haplotype is inherited from a parent by both sibs, this is scored as one repeat. Thus, a sibship of two individuals may show 0, 1 or 2 repeats. For sibships of more than two, where an HLA haplotype is inherited by r siblings, the score is $r-1$ repeats. The expected mean score for a sibship of size s , assuming no linkage, is $2s-4+2^{-s+2}$, and the expected variance is $2^{-s+2}(1-2^{-s+1})$ (Green & Woodrow, 1977).

A total score S can be obtained by summing the scores derived from various families. Similarly, the sum of the means and variances gives total expected mean (μ) and total expected variance (σ^2). The test statistic T is defined by

$$T = \frac{(S - \frac{1}{2} - \mu)}{\sigma}$$

Comparison with a standard normal integral table is then made.

Results

The details of all the families studied are shown in Fig. 1. All the family members, whether tested or not, have been in-

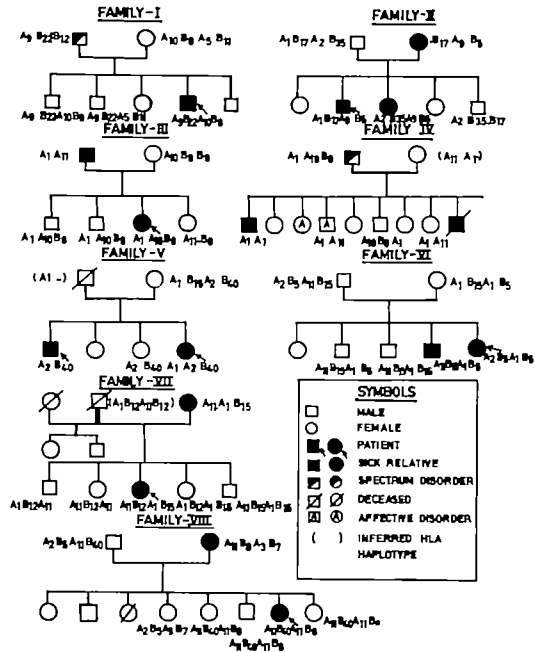


Fig. 1 The distribution of psychiatric diagnosis and HLA haplotypes of the families

TABLE I
Family lod scores*

Family	Recombination fraction (θ)				
	0.05	0.1	0.2	0.3	0.4
I	-0.721	-0.444	-0.194	-0.076	-0.018
II	0.535	0.465	0.318	-0.170	-0.049
III	-1.443	-0.887	-0.388	-0.151	-0.036
IV	-1.443	-0.887	-0.388	-0.151	-0.036
V	-0.721	-0.444	-0.194	-0.076	-0.018
VI	-0.464	-0.229	-0.060	-0.011	-0.001
VII	-1.443	-0.887	-0.388	-0.151	-0.036
VIII	-1.443	-0.887	-0.388	-0.151	-0.036
Total	-7.151	-4.201	-1.781	-0.598	-0.129

*Presumed mode of inheritance, dominant with total penetrance

cluded, and the HLA haplotypes identified as mentioned. Table I shows the combined lod scores of the families at different recombination fraction values, presuming the mode of transmission in schizophrenia to be autosomal dominant with complete penetrance. Following Morton (1955), the convention is to regard a lod score of 3 or more as firm evidence for, and a score of -2 or less as firm evidence against any linkage. Any score in between can be

TABLE II
Comparison of present work with earlier studies

Study	Recombination fraction (θ)				
	0.05	0.1	0.2	0.3	0.4
Present	-7.151	-4.201	-1.781	-0.598	-0.129
Turner (1979)	1.351	2.350	2.445	1.688	0.649
McGuffin (1983)	-8.088	-4.187	-1.072	-0.084	0.104

considered as equivocal. The total of lod scores at recombination fractions 0.05 and 0.1 are firmly against the probability of any linkage between HLA and schizophrenia (Table I).

The combined lod scores of the present work were compared with the earlier investigations of Turner (1979) and of McGuffin *et al* (1983) (Table II). Our results are most similar to those of McGuffin *et al*.

An analysis of the data by the affected sib-pair method was made. The scores of repeats (S) was 5, the expected mean (μ) 4, and the expected variance (σ^2) was 2. This gave a T value of 0.354, which at d.f. = 3 is not significant. The result does not favour a linkage between HLA and schizophrenia.

Discussion

The present study fails to support the notion of a linkage between HLA and schizophrenia. Our findings are in agreement with McGuffin *et al* (1983), but not with Turner (1979), though the same sequential method of linkage analysis has been carried out on all three data sets.

Analysis of our data by the affected sib-pair method of Green & Woodrow (1977) also does not indicate linkage between HLA and schizophrenia; this is in agreement with McGuffin *et al*. Turner did not use the affected sib-pair method, but his data were subjected to analysis by this method by McGuffin *et al*, and that too did not favour linkage. However, the small number of sibships is a limitation which must be kept in mind when interpreting the results.

In any linkage study in schizophrenia, there are numerous practical and methodological problems. Finding an adequate number of consenting informative families suitable for the purposes of such studies is a major problem. Despite our identifying a large number of potentially suitable families, only eight could be tested. These difficulties have been highlighted by Watt (1982) and by McGuffin *et al*.

A potential source of error in such studies is the uncertainty of diagnosis of schizophrenia. The

present study has employed RDC (Spitzer *et al*, 1978), and in two subjects (both relatives), the diagnosis of schizophrenia spectrum disorder (Kety *et al*, 1968) was entertained. All the index cases were admitted to hospital at some time, and the sick relatives, with two exceptions, were registered at a psychiatric out-patient clinic. Turner (1979) employed the criteria of Feighner *et al* (1972) for the diagnosis of schizophrenia, but relied quite heavily on the spectrum disorder concept of Kety *et al* (1968). McGuffin *et al* (1983) used the CATEGO system (Wing *et al*, 1974) for the diagnosis of schizophrenia and an objective schedule devised by Khouri *et al* (1980) for identifying schizophrenia spectrum disorder. These three studies thus used strict criteria for the diagnosis of schizophrenia, and the concept of schizophrenia spectrum disorder is common to all. Therefore, our failure to demonstrate a linkage between HLA and schizophrenia lends more support to the view that differences in diagnosis alone cannot account for the discrepant results obtained by Turner and by McGuffin *et al*. There is a strong possibility of a genuine absence of linkage between HLA and schizophrenia.

The status of schizophrenia spectrum disorder remains controversial, and it is debatable whether cases diagnosed as such should be included in a linkage study. It has been demonstrated that a number of non-schizophrenic disorders have a tendency to concentrate in the families of patients with schizophrenia (Heston, 1966; Kety *et al*, 1968; Rosenthal *et al*, 1971; Stephens *et al*, 1975). However, Lidz *et al* (1981) have been critical of this concept, and have challenged its validity. Lowing *et al* (1983), in a reanalysis of the original data of Rosenthal *et al* (1971), found a higher occurrence of spectrum disorders in the families of index cases, compared with controls. In view of the evidence supporting the concept of schizophrenia spectrum disorder, we have included subjects falling in this category in our analysis.

Genetic heterogeneity of schizophrenia is a factor which could lead to failure to establish any linkage between the marker studied and the disease. McGuffin *et al* (1983), in an effort to increase the homogeneity of the sample, analysed the data after excluding families with an admixture of diagnosis, but even then were unable to demonstrate linkage. By using strict diagnostic criteria, we have endeavoured to reduce heterogeneity; our findings do not support the existence of two types of schizophrenia—one linked to HLA and the other not.

Finally, it is important in linkage studies that the mode of transmission of the disorder should be known, which is not the case in schizophrenia. However, we have presented the results of our data

analysis presuming the mode of inheritance to be autosomal dominant with total penetrance. In addition, we have used the affected sib-pair method of linkage analysis which, though less powerful than sequential tests, makes assumptions about genetic modes of transmission unnecessary. Our data are being analysed further, using the assumption that the

mode of inheritance is recessive and autosomal dominant with incomplete penetrance.

Our results do not indicate support for linkage between HLA and schizophrenia. Perhaps studies with more homogeneous samples of schizophrenic families and better methods of linkage analysis are needed to unravel the genetics of schizophrenia.

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Late Onset Involuntary Movements in Chronic Schizophrenia: Relationship of 'Tardive' Dyskinesia to Intellectual Impairment and Negative Symptoms

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Intellectual impairment, negative symptoms, and medication history were assessed in chronic schizophrenic patients with and without abnormal involuntary movements (tardive dyskinesia). Patients with involuntary movements had received neither longer nor more intensive treatment with neuroleptics or anticholinergics. However, the presence or absence of involuntary movements was prominently associated with the presence or absence of intellectual impairment/negative symptoms; these features are characteristic of the defect state/type II syndrome of schizophrenia, in which structural abnormalities of the brain may be over-represented. The role of subtle organic changes in conferring vulnerability to the emergence of such involuntary movements should be re-evaluated.

More than 20 years of research has failed to identify the essential factors which determine whether late-onset involuntary movements (tardive dyskinesia) occur in individuals receiving long-term treatment with neuroleptic drugs. The extensive literature on this topic (Jeste & Wyatt, 1981; Kane & Smith, 1982; Waddington, 1984; Waddington *et al*, 1986) indicates that affected individuals have had neither longer nor more intensive treatment with neuroleptics, but suggests that older age increases the likelihood of developing involuntary movements. Vulnerability to their emergence resides more within the central nervous system of the individual patient, usually diagnosed as schizophrenic, than with the treatment.

It has been proposed that patients with pre-existing forms of organic brain dysfunction may be more likely to develop involuntary movements. However, previous studies on heterogeneous patient groups with varying indices of organicity are inconclusive on this issue (Kane & Smith, 1982). Increasing attention is now being paid to chronic schizophrenic patients who show intellectual impairment and whose clinical picture includes prominent negative symptoms—the 'defect state' or type II syndrome (Crow, 1980). These characteristics are over-represented in the subgroup of schizophrenic patients who show structural abnormalities of the brain on computerised tomography (CT) scan, particularly ventricular dilatation (see Crow, 1982; Andreasen & Olsen, 1982; Seidman, 1983; Waddington *et al*, 1986). The present study specifically investigated associations between the presence of involuntary movements and of intellec-

tual impairment and negative symptoms in chronic schizophrenia, in relation to exposure to neuroleptic and anticholinergic medication.

Method

Our study population comprised 68 long-stay in-patients, aged 30-90 years, who satisfied the criteria of Feighner *et al* (1972) for a diagnosis of schizophrenia and who consented to assessment.

Movement disorder was evaluated using the Abnormal Involuntary Movement Scale (NIMH, 1976); this was followed by neuropsychological assessment of intellectual/cognitive function using an abbreviated form of the Roth-Hopkins test, similar to that described by Qureshi & Hodkinson (1974). The investigator was blind to clinical and medication histories until after this index evaluation.

There is the possibility that objective evaluation of negative symptoms (e.g. poverty of speech and blunting of affect) in relation to involuntary movements will be compromised by the essentially self-revealing nature of such movements. Therefore, they were indexed as follows: muteness was taken as an extreme of poverty of speech, and considered present when verbal production was absent during motor assessment and during attempts at neuropsychological evaluation; in the presence of muteness, no estimate of intellectual function was made. Blunted affect was considered present when review of those periodic case-note reports made prior to the index evaluation, by the consultant in charge of each patient, made specific reference to the presence of this symptom. While these reports appeared thorough, practice in recording blunted affect would undoubtedly vary between the several consultants involved. Thus, both of these estimates of the presence or absence of negative symptoms were conservative, by virtue of their

sensitivity only to the more prominent signs, but had the clear advantage of objectivity

During the subsequent case-note review, demographic details and history of drug treatment were obtained. Doses of neuroleptics were converted to chlorpromazine equivalents according to standard tables (Davis, 1976) and length, average, and current daily dose and total lifetime intake of neuroleptics were determined. Data were expressed as means $\pm$ SEM or as percentage prevalences, and were analysed using Student's *t*-test or the chi-squared test.

Results

As noted in the majority of reports in patients with long-term exposure to neuroleptics (e.g. Owens *et al*, 1982; McCreadie *et al*, 1982; Karson *et al*, 1983), involuntary movements of the limbs and trunk were rare in comparison with those of the orofacial region. Since several recent studies additionally suggest that limb and trunk movements may constitute a pathophysiologically distinct syndrome (Kidger *et al*, 1980; Karson *et al*, 1983), all further references to abnormal involuntary movements (AIM) relate to these hallmark characteristics of the tongue, jaw, lips, and muscles of facial expression.

Out of our population of 68 chronic schizophrenic patients, 28 exhibited at least mild but definite AIM of one or more orofacial areas; in the remaining 40 patients, either no or minimal movements were present. As recommended by Schooler & Kane (1982), movement examination was repeated within one week of the index evaluation in a random group of 12 patients with AIM, particularly to estimate the reliability of assessments made at the 'mild' level of

abnormality. Of these 12 reassessed patients, abnormality was confirmed in 11; the remaining individual showed minimal movements on that occasion, and was included in the group of 40 patients without abnormality. There were no significant sex differences in the prevalence of AIM, which were present in 8 of 22 males (36%) and in 20 of 46 females (43%).

Medication

Patients with AIM were significantly older than those without AIM (Table I). There was a non-significant trend for AIM patients to have a longer duration of illness and to have been older when first given neuroleptics. The two groups had received neuroleptic drugs for indistinguishable periods of time. However, those with AIM had received significantly smaller average daily doses of neuroleptics, and had significantly smaller total lifetime intakes of neuroleptics. The two groups were not distinguished by the presence or absence of current neuroleptic treatment at the index evaluation.

For those individuals not currently receiving treatment, the period that had elapsed since drug withdrawal did not differ between the two groups (without AIM: 4.9 ± 1.3 years; with AIM, 4.5 ± 1.1 years). Patients with AIM were somewhat more likely to have been drug-free for at least one month (72% were drug-free, compared with 46% of those without AIM) and tended to have spent a longer total time drug-free (3.5 ± 0.8 years drug-free, compared with 2.4 ± 0.6 years for those without AIM) since initiation of neuroleptic treatment; these trends approached, but did not attain statistical significance.

TABLE I
General characteristics and medication histories of schizophrenic patients with and without AIM (mean $\pm$ SEM, unless otherwise indicated)

	Without AIM (n=40)	With AIM (n=28)
<i>General</i>		
Age: years	61.6 $\pm$ 2.1	68.5 $\pm$ 2.7*
Duration of illness: years	33.4 $\pm$ 1.8	38.5 $\pm$ 2.5
<i>Neuroleptic treatment</i>		
Age at start: years	44.4 $\pm$ 2.2	50.6 $\pm$ 2.6
Duration: years	13.8 $\pm$ 1.0	13.0 $\pm$ 1.3
Daily dose: mg chlorpromazine	449 $\pm$ 69	259 $\pm$ 49*
Lifetime intake: g chlorpromazine	2233 $\pm$ 386	1180 $\pm$ 231*
Current dosage: n		
0	9 (23%)	10 (35%)
25-1000 mg chlorpromazine/day	19 (47%)	17 (61%)
> 1000 mg chlorpromazine/day	12 (30%)	1 (4%)
<i>Anticholinergic treatment</i>		
Duration: years	5.9 $\pm$ 0.8	4.9 $\pm$ 0.9
Currently receiving treatment: n	10 (25%)	6 (21%)

**P* < 0.05, Student's *t*-test

TABLE II
Intellectual impairment and negative symptoms in schizophrenic patients with and without AIM

	Without AIM (n=40)	With AIM (n=28)
<i>Intellectual impairment</i> ¹		
Mean score ($\pm$ SEM)	6.7 $\pm$ 0.3	5.3 $\pm$ 0.6*
Score < 50%: n	2 (6%)	6 (38%)**
<i>Negative symptoms</i>		
Muteness: n	6 (15%)	11 (39%)**
Blunted affect n	7 (17%)	11 (39%)
<i>Defect state/type II signs</i>		
One or more of muteness, blunted affect, or intellectual impairment	14 (35%)	25 (89%)***

¹ n = 32 (without AIM), n = 16 (with AIM)

*P < 0.05, Student's *t*-test

**P < 0.05, chi-squared

***P < 0.001, chi-squared

The two groups were not distinguished by any measure of the anticholinergic treatment which almost all patients had received at some stage during neuroleptic therapy, i.e. current dosage and total duration of treatment with anticholinergics (Table I). Comparisons of anticholinergic doses did not help to distinguish the two groups, because of the consistent use of only a small number of drugs over equivalent daily dose ranges (usually benztropine, 2–4 mg).

Intellectual impairment and negative symptoms

Intellectual impairment was assessed in 48 of the patients; of the remainder, 17 were mute and three refused to co-operate with neuropsychological evaluation. Mean scores were significantly lower in the group with AIM, indicating greater intellectual impairment in comparison with those patients without AIM (Table II). Prominent intellectual impairment was defined by a score of less than 50% on neuropsychological assessment, and occurred with significantly greater frequency in patients with AIM. Negative symptoms, defined as above, also occurred more frequently in patients with AIM; for muteness, this excess was statistically significant, while for blunted affect it just fell short ($P < 0.1$) of significance. Table II also shows the number of patients in each group who showed at least one of these three features, while only 35% of patients without AIM showed one or more of these signs, 89% of those with AIM were characterised by these features ($P < 0.001$). This difference could not be accounted for by the slightly but significantly greater average age of the group with AIM. In two age-matched groups, derived by elimination of data from individuals at the extremes of age, this clinical difference was essentially unaltered; 40% of those without AIM (age = 64.2 ± 1.8 years, $n = 35$), compared with 87% of those with AIM (age = 65.6 ± 2.8 years, $n = 24$), showed one

or more of the characteristics of prominent intellectual impairment, muteness, or blunted affect ($P < 0.001$).

Discussion

The prevalence of AIM (41%) in this group of schizophrenic patients was typical. Patients with AIM had a slightly but significantly higher mean age, consistent with the majority of studies. The phenomenon appears to reflect an increased vulnerability with ageing and chronicity, rather than increased exposure to neuroleptics with increasing age (Kane & Smith, 1982; Waddington & Youssef, 1985). In further agreement with the majority of studies (Kane & Smith, 1982), our schizophrenic patients with AIM had received neither longer nor more intensive therapy with neuroleptics. Rather, patients with AIM had received significantly smaller average daily doses and total lifetime intakes of neuroleptics over their period of exposure. This finding is consistent with the report by Smith *et al* (1978) of a similar inverse relationship between involuntary movements and total neuroleptic intake during hospital treatment. It might suggest that patients with AIM were less severely ill than their counterparts, or that the form of their illness was different in some way.

Over the past few years, there have been a number of reports suggesting that a greater extent of drug-free periods during long-term neuroleptic treatment

may in fact be associated with an increased risk of AIM (Kane & Smith, 1982; Branchey & Branchey, 1984). Our patients with AIM tended to have had more discontinuous treatment, but the data were inconclusive. We also found patients with AIM not to be distinguished by greater exposure to anticholinergic medication. The literature as a whole (Kane & Smith, 1982) supports this finding in relation to vulnerability to AIM.

Although we had greater difficulty in distinguishing our two groups of patients in terms of medication history, the presence or absence of certain symptoms was a powerful discriminator of the presence or absence of AIM: intellectual impairment, poverty of speech, and blunted affect. These are prominent features of the defect state or type II syndrome of schizophrenia (Crow, 1980). Even using the present conservative indices, almost 90% of our patients with AIM had one or more of these symptoms, while only about one-third of those without AIM were characterised in this way. The strength of this discrimination ($P < 0.001$) suggests an intimate relationship between features of the defect state and AIM in schizophrenia.

Other studies support this notion. Regarding intellectual impairment, Famuyiwa *et al* (1979) found schizophrenic patients with AIM to be more impaired on a paired-associate learning test; they did not show greater impairment on the Withers & Hinton test of the sensorium, but were some 15 years younger than our own patients. More recently, Wolf *et al* (1983) found no difference in cognitive function between schizophrenic patients with and without AIM respectively, using the indices of Wechsler and of Rey, in patients a decade younger than our own. However, in younger patients, Struve & Wilner (1983) found those with AIM to be more impaired on the Conceptual Level Analogy Test. Regarding negative symptoms, McCreddie *et al* (1982) noted that older schizophrenic patients with AIM more commonly showed flattening of affect, and older schizophrenic patients with these features of intellectual impairment and negative symptoms may show AIM even in the absence of a history of exposure to neuroleptics (Owens & Johnstone, 1980; Owens *et al*, 1982). Thus, neuroleptics might not cause such movements, but rather, might enhance a usually covert, but sometimes overt predisposition towards AIM (Waddington *et al*, 1983, 1986).

This over-representation of intellectual impairment and negative symptoms in our AIM group might be the basis of their reduced exposure to neuroleptics in the present study. A clinical picture in which these features predominate would require less intense treatment with neuroleptics. The case-notes

revealed that the psychiatrists caring for our patients had attempted to reduce neuroleptic dosage to match the extent of positive symptoms, and also to minimise medication of patients with the defect state, where least benefit is likely to be derived (Crow, 1982). This interpretation is supported by the study of Opler *et al* (1984), who found that schizophrenic in-patients with negative symptoms had been less heavily medicated than patients with positive symptoms. Similarly, the trend towards more discontinuous medication in our patients with AIM may be explicable in these terms: case notes indicate that drug-free periods were not a result of non-compliance, but rather a clinical decision that in the absence of positive symptoms, neuroleptic treatment was not warranted. However, patients with AIM were slightly older, and tended to have had a greater duration of illness when neuroleptics were first introduced; this may in itself have contributed to a less enthusiastic approach to neuroleptic treatment.

Involuntary movements and intellectual impairment/negative symptoms may be two separate manifestations of a common pathophysiological process that both underlays deterioration to the defect state and confers vulnerability to the emergence of AIM during long-term neuroleptic treatment. Our point-prevalence study cannot indicate whether AIM and these features of type II schizophrenia tend to arise together, or whether either one might precede the other. This would only become clear from prospective studies in schizophrenia.

Our data indicate that signs of type II schizophrenia are a much better predictor of the presence or absence of AIM than any variable related to medication. Such features of the type II syndrome have been associated with a variable excess of structural abnormalities of the brain by CT scan, particularly ventricular dilatation (Crow, 1982; Andreasen & Olsen, 1982; Seidman, 1983). While CT scan studies in relation to the presence or absence of AIM are not consistent, this may reflect heterogeneous groups which include non-schizophrenic patients with other potential structural or organic changes (Waddington *et al*, 1986). In the light of the present evidence, we suggest that a role for various subtle organic factors in rendering a patient vulnerable to the emergence of AIM should be reconsidered, not just in schizophrenia but also in patients with other diagnoses who are given neuroleptics and in whom AIM are demonstrable.

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The Nithsdale Schizophrenia Survey V. Follow-up of Tardive Dyskinesia at 3½ years

A. D. T. ROBINSON and R. G. McCREADIE

The point-prevalence of tardive dyskinesia in schizophrenics from a discrete geographical area (Nithsdale, in Dumfries and Galloway Region) in 1981, 1982, and 1984 was 31%, 27%, and 30% respectively. This suggests that the prevalence of tardive dyskinesia in a community of schizophrenics has reached a plateau. In 12% of patients there was persistent dyskinesia, i.e. abnormal involuntary movements were present at all three assessments. Persistent dyskinesia was more common in older patients. The severity of tardive dyskinesia fluctuated between assessments in 41% of patients, indicating that it is only a transient feature in some cases.

A previous study of tardive dyskinesia (TD) in all known schizophrenics in Nithsdale, a historically ancient and geographically discrete part of Dumfries and Galloway Region, found a point-prevalence of 31% (McCreadie *et al.*, 1982); an interim review one year later found a prevalence of 27% (Barron & McCreadie, 1983). It was suggested that as a generation of schizophrenics has now been exposed to neuroleptics, which are thought to be the main aetiological factor in TD (*The Lancet*, 1979), the community prevalence might have reached a plateau. To determine whether this may be so, the present study examined Nithsdale schizophrenics 3½ years after the initial census. It also identified and described patients with persistent dyskinesia.

Method

The 1981 census

The original census, carried out in March 1981, identified all known schizophrenics in Nithsdale. Included in the survey were all in-patients, day-patients, and out-patients of Crichton Royal Hospital who on 1 March 1981 had a firm case-record diagnosis of schizophrenia and whose home address was in Nithsdale. In addition, all the general practitioners in the area replied to our questionnaire and identified schizophrenics known to the practice and receiving treatment. The Feighner criteria for schizophrenia (Feighner *et al.*, 1972) were applied to each patient. The census identified 133 schizophrenics, 117 of whom were examined for tardive dyskinesia.

3½-year follow-up

The repeat census was carried out 3½ years later (20 August 1984) in a similar way to the initial survey. It identified 145 schizophrenics, of whom 70% fulfilled the Feighner criteria for definite or probable schizophrenia, and 88 (61%) were

members of the original cohort: 17% were in-patients, 24% day-patients, 39% out-patients or supervised by a community psychiatric nurse (CPN), and 20% were attending only their family doctor.

We examined 130 of the schizophrenics (90%) for evidence of tardive dyskinesia—all the in-patients, all the day-patients except one, all the out-patients except one, all the CPN patients and 57% of the general-practice patients. To assess TD, we used the Abnormal Involuntary Movements Scale (AIMS) (US Department of Health, Education and Welfare, 1976), in which individual areas of the body are examined and then a global assessment is made: this ranges from 0 (no movements) through 1 (minimal), 2 (mild), and 3 (moderate), to 4, 'severe movements'. The assessments exclude tremor, akathisia, and dystonia. Our assessments were made during a 6-week period by two psychiatrists, and 12% of patients were examined independently by both psychiatrists to assess inter-rater reliability.

Of those examined, 16% were not currently receiving neuroleptics, but only 5 patients (4%) had no record of such drugs having been prescribed in the past; 27% were receiving anti-parkinsonian drugs. The length of time for which patients had been receiving neuroleptics since the initial census and the route of drug administration were determined; no attempt was made to estimate the total quantity of medication prescribed, as such information would probably have been inaccurate, especially for out-patients and general-practice patients.

Differences between groups were tested by the chi-squared test.

Results

Inter-rater reliability on the 5-point global AIMS scale was high (Spearman rank correlation coefficient $r = +0.82$, $P < 0.01$).

1984 prevalence of abnormal movements

If patients with a rating of at least 'mild' on the global AIMS

TABLE I
Point prevalence of abnormal involuntary movements among schizophrenics in Nithsdale (1981, 1982, 1984)

Abnormal movements (AIMS score)	1981		1982		1984	
	All patients (N = 117)	Subgroup* (n = 88)	All patients (N = 122)	Subgroup* (n = 88)	All patients (N = 130)	Subgroup* (n = 88)
Mild/moderate/severe	31%	35%	27%	32%	30%	30%
Nil/minimal	69%	65%	73%	68%	70%	70%

*Subgroup: patients rated on all three occasions

scale are considered to have definite TD, then the prevalence is 30% (Table I): 18% with only oro-facial movements, 3% with trunk and/or limb movements, and 9% with both oro-facial and distal movements.

Persistent dyskinesia

Of the 145 patients, 88 (61%) had been examined on all three occasions. The prevalence figures for definite TD in this subgroup in 1981, 1982, and 1984 were 35%, 32%, and 30% respectively (Table I). Almost half (47%) of the subgroup had never shown signs of dyskinesia; 12% had dyskinesia on all three occasions ('persistent dyskinesia'); and 41% had dyskinesia on at least one but not three occasions ('fluctuating dyskinesia'). In the 'fluctuating' group, the severity of dyskinesia increased over time in 11% of cases, decreased in 17%, and showed no trend in 13%. The details of these changes are presented in Table II.

Patients with persistent dyskinesia were compared with those who had never had abnormal movements, on the basis of sex, age, hospitalisation status. Feighner categorisation, and medication (current and previous, antipsychotic and

anti-parkinsonian). There were no statistically significant differences between the two groups, although the trend was: that patients in the persistent dyskinesia group were older (mean age 51 years vs 45 years) and more often in-patient: or day-patients (73% vs 44%).

In the group with fluctuating dyskinesia, when those with worsening TD were compared with patients showing decreasing TD, there were no statistically significant differences in the clinical, social, and demographic characteristics outlined above. In particular, there was no clear relationship between the starting or stopping of anti-psychotic or anti-parkinsonian medication and fluctuation in dyskinesia. However, the trend was that patients with worsening TD were younger (mean age 51 years vs 61 years).

Of those with persistent dyskinesia, 9% had only oro-facial movements and 91% both oro-facial and distal movements.

Discussion

The point-prevalence of TD in all known schizophrenics from a given geographical area, assessed on three separate occasions spread over 3½ years, has remained fairly constant between 27% and 31%. It is probable, therefore, that the community prevalence has indeed reached a plateau, and is not likely to increase further with time. As new patients develop schizophrenia and are given neuroleptics, some will develop dyskinesia. These will be balanced in numbers by others dying or recovering from the illness; in the latter group, medication will probably be stopped. The plateau in the present series of studies contrasts sharply with the reported increase in prevalence during the 1970s. As anti-psychotic medication was widely prescribed at that time, the supposed increase in prevalence probably reflect clinicians' increasing awareness of the condition: the true prevalence may well have been essentially unchanged throughout.

Interpretation of our results is complicated by the finding that although about a third of the patient

TABLE II
Patterns of presence/absence of abnormal involuntary movements in a group of 88 schizophrenics all of whom were assessed in 1981, 1982 and 1984

1981	1982	1984	n (%)
Absent	Absent	Absent	41 (47%)
Absent	Absent	Present	4 (4%)
Absent	Present	Present	6 (7%)
Absent	Present	Absent	6 (7%)
Present	Absent	Present	5 (6%)
Present	Absent	Absent	10 (11%)
Present	Present	Absent	5 (6%)
Present	Present	Present	11 (12%)
			88

had TD at any given time, only 12% of those patients rated on all three occasions had persistent dyskinesia. This latter figure is probably a more accurate assessment of the severity of the problem: point-prevalence figures include mild and reversible TD, and paint too gloomy a picture. Schizophrenics with persistent dyskinesia tended to be older than those with no TD: age is the only factor consistently found to be associated with TD (Smith *et al.*, 1979; Seeman, 1981; Kane & Smith, 1982; Barnes *et al.*, 1983).

41% of patients examined on all three occasions had TD on one or two, but not all three occasions. There are no doubt several reasons for fluctuating dyskinesia (reviewed by Bergen *et al.*, 1984). Deficiencies in assessment, such as within-rater variability (Bergen *et al.*, 1984), lack of sensitivity of the rating scale (Barnes, 1984), or lack of standard-

isation of rating schedules in relation to time of depot neuroleptic administration (Barnes & Wiles, 1983), may be important. It is unlikely, however, that these methodological problems can entirely explain fluctuating dyskinesia: the present findings therefore emphasise the transient nature of TD in some patients. In the present study, worsening dyskinesia over time was more common in young patients. Such patients had probably had a shorter exposure to neuroleptics, which had not yet produced their maximum effect; as the years go by, dyskinesia may be expected to worsen in them.

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Deliberate Self-harm: How Do Patients View Their Treatment?

D. PIERCE

The opinions of patients who had caused deliberate self-harm about the attitudes of doctors, nurses, families and others towards their acts were studied. Their views of the attitudes of hospital staff were found to be markedly different from attitudes reported elsewhere by staff themselves. There were no differences between the patients' views of their handling by nurses, families, and others towards their acts were studied. Their views of the attitudes of families were unsympathetic. A significant association was found between repetition of an act of deliberate self-harm and the perception of an unsympathetic attitude within the family.

While we have information about the attitudes of doctors and nurses (Patel, 1975; Ramon *et al*, 1975) and of people close to the patient (James & Hawton, 1985) towards cases of deliberate self-harm (DSH), there is little about how the patients themselves view the treatment they are given after their acts of self-harm. It is known for instance that hospital staff have a different attitude towards cases of this kind from that towards patients presenting with physical illnesses (Patel, 1975), but we do not know how much of this is apparent to the patients. There is evidence that nursing and medical staff differ significantly between themselves in their feelings of sympathy towards parasuicidal patients (Patel, 1975; Ramon *et al*, 1975); if patients are aware of these differences, there may well be implications for their assessment and management in hospital.

Significant other persons tend on the whole to be sympathetic in their reactions (James & Hawton, 1985), although Morgan (1979) has described expressed hostility and a definite lack of sympathy in some cases. It is of practical importance to know whether such responses are evident to the patients, and if so, whether these perceived attitudes have any effect on their subsequent behaviour. One could argue that a reaction perceived as hostile might discourage a person from repeating his self-harm, while an over-sympathetic response might even promote such repetition. What advice, therefore, should we give to relatives who find themselves in this dilemma?

This study was designed

- (a) to find out how patients view the treatment they have received on general hospital wards after admission for DSH
- (b) to relate these views to the actual attitudes of doctors and nurses towards DSH patients, as previously reported

- (c) to compare the patients' views of the attitudes of professional staff towards them with their views of the attitudes of their families
- (d) to establish whether the patient's view of his treatment by professional staff or by his family is related to the likelihood that he will repeat his DSH.

Method

Patients admitted consecutively to East Glamorgan Hospital for treatment of the effects of DSH were considered for the study, and interviewed as soon as their clinical condition allowed. The interview was a standardised one, used at the hospital for several years, and leads to the measurement of suicidal intent, to a psychiatric evaluation, and to a decision about further management. Patients who were confused or suffering from a psychotic illness were subsequently excluded, as were those unable to provide a significant other person likely to be affected by their action.

At the end of the interview, each patient was asked to give his opinion of the treatment he had received in hospital from the medical and nursing staff in the casualty department and on the ward. He was asked to categorise it as:

- (a) definitely sympathetic and helpful
- (b) neutral, i.e. neither particularly sympathetic or unsympathetic
- (c) definitely unsympathetic and unhelpful.

It was made clear that this opinion was not to be relayed to the staff concerned, and the patient was not asked to complete a form. It was considered that this might be intimidating and thus discourage patients from giving an honest opinion.

He was then asked to provide his opinion of the attitude of his close family or a suitable significant other person to his behaviour, categorised in the same way. If several members of the family were felt to hold varying attitudes, he was asked to report his view either of the overall family attitude or of the attitude of the family member most important to him.

TABLE I
Attitudes of hospital staff: perceived and self-reported

Staff group	Unsympathetic	Attitude: % Neutral	Sympathetic
Senior doctors, self-reported ¹	25	33	42
Patients' view of doctors attitude	12	31	57
Junior doctors, self-reported ¹	44	44	12
Patients' view of nurses' attitude	14	31	55
Nurses, self-reported ¹	40	47	13

1. Figures from Patel (1975)

The patients were followed-up for a year, and a note made of further episodes of DSH leading to hospital treatment. It was not possible to obtain accurate information about episodes treated at home.

The results were analysed using the Spearman rank correlation to measure the degree of agreement between the patient's ratings of doctors, nurses, and families. The Wilcoxon matched pairs test and the Mann-Whitney U test were used to explore the relationships between various subgroups of patients.

Results

One hundred patients were included in the study (37 male, 63 female), and the mean age was 34 years; they were therefore comparable with other reported samples of DSH patients (Birtchnell & Alarcon, 1971; Silver *et al*, 1971). All had been admitted to the hospital via the accident and emergency department, and their mean length of stay in the hospital before interview was a little under 24 hours. All but two were nursed on general medical wards.

Of the total, 55% thought that the nurses had been definitely sympathetic towards them; this was almost exactly the same as the figure of 57% for their view of their treatment by the doctors. Fourteen percent felt that the nurses had handled them unsympathetically, compared with 12% who held the same opinion about the doctors. For the most part, it was the same patients who were unhappy about the attitudes of the different hospital professionals.

The patients' perceptions of the attitudes of both doctors and nurses towards them were markedly different from the attitudes previously reported by hospital staff themselves (Table I).

One-third of the patients believed that their families had been unsympathetic to their action, while 42% felt that they had been helpful; this indicated that the families were regarded as significantly less sympathetic than either nurses or doctors ($P < 0.01$, Wilcoxon matched pairs test). There was no tendency for the same patients to think that both the professionals and their families were unsympathetic. A strong relationship emerged between perceived lack of sympathy on the part of the family and a history of earlier acts of DSH: half of those patients who had harmed themselves previously thought their families unhelpful, and this was

TABLE II
Repetition and perceived attitude of significant other persons

	Perceived attitude: n	
	Unsympathetic	Sympathetic or neutral
Repeaters	8	4
Non-repeaters*	25	63
Total	33	67

* $P < 0.02$ (Fisher's Exact test) for repeaters compared with non-repeaters

significantly different ($P < 0.01$) from the attitude reported by the other patients.

No relationship was found linking the patient's view of his hospital management with the likelihood of repetition, but a connection did emerge between repetition and his view of the family's feeling towards his behaviour. Among those patients who repeated DSH within the follow-up period, as many as two-thirds had regarded their families as unsympathetic after the index episode (Table II). This is a significantly higher proportion than among the non-repeaters ($P < 0.02$). Furthermore, when the 11 patients who had an earlier history of DSH and who considered their families to be unsympathetic were looked at separately, seven of them repeated DSH during follow-up (64%), compared with 6% for other patients ($P < 0.01$). As expected, there was strong tendency for patients with a history of earlier episodes to repeat during follow-up ($P < 0.001$).

Discussion

The substantial differences between the patients' perceptions of nurses' and doctors' attitudes towards them and the attitudes earlier reported by staff themselves suggested that any lack of sympathy felt by staff treating DSH cases is not readily apparent to their patients. If this is so, the finding must be a

welcome one from the ethical point of view, unless one subscribes to the opinion that some degree of unsympathetic handling may discourage repetition. There is a possibility, however, that the apparent differences in perceptions of patients and staff can be explained by a reduction in the antipathy of doctors and nurses towards self-harming patients brought about by improvements in education and training during the years between Patel's study and ours. An alternative explanation could lie in sampling differences between the two studies, but this seems unlikely, as they were carried out in similar settings, involving unselected cases.

It remained true, however, that one in six patients felt that nurses had not been helpful or sympathetic – perhaps not a surprising finding in the light of the percentage of DSH cases reported to have histories of antisocial behaviour (Morgan *et al.*, 1975), who are likely to provoke hostility in all those around them. Yet there was no trend for the same patients to see both families and professional staff as not sympathetic, so that the perceived attitudes may be a true reflection of their handling, rather than simply a mirror of their own personalities.

James & Hawton (1985) have recently shown that 50% of significant other persons express much sympathy after parasuicidal acts, while 3% feel no concern, and 18% show 'little sympathy'. The present study categorised the patients' perceived attitudes of significant others in a different way, but 42% thought that the other person had been definitely sympathetic – a comparable figure to James & Hawton's, suggesting a close correspondence between actual and perceived favourable attitudes of those near to the patient. This is of some interest in view of earlier authors' comments (e.g. Bancroft *et al.*, 1977) on faulty or disturbed communications between DSH patients and those important to them.

The finding that one-third of the patients felt

that their families had been unsympathetic has implications for the management of DSH cases, but this cannot be divorced from the question of the possible effects of this lack of sympathy. The results show clearly that the perception of a lack of sympathy in the family is associated with repetition. If this link is causal, then efforts should be directed to discovering whether this perception is an accurate one and, if it is, to making an attempt to alter it, in the hope of reducing the repetition rate. It seems probable, however, that any negative attitude in the family is itself closely related to a history of earlier DSH episodes. It may therefore be mainly a reaction to a series of acts perhaps seen as manipulative, and the family's attitude might well not have played any causative role in initiating the patient's repetitious self-harming.

For this reason, it will be important to concentrate attention on 'first ever' episodes in elucidating the different forms that family reaction to DSH can take, and in planning experiments to reduce repetition by modifying family responses which are felt to be inappropriate.

From the behavioural standpoint, it would seem reasonable to expect that a patient's belief that those around him were unsympathetic towards him on account of his act would discourage the idea of repetition, but the findings of this study suggest otherwise. It might be fruitful to continue to study the effects of family and staff attitudes on repetition by carrying out experiments which allow some patients to be treated in a more overtly sympathetic way than others.

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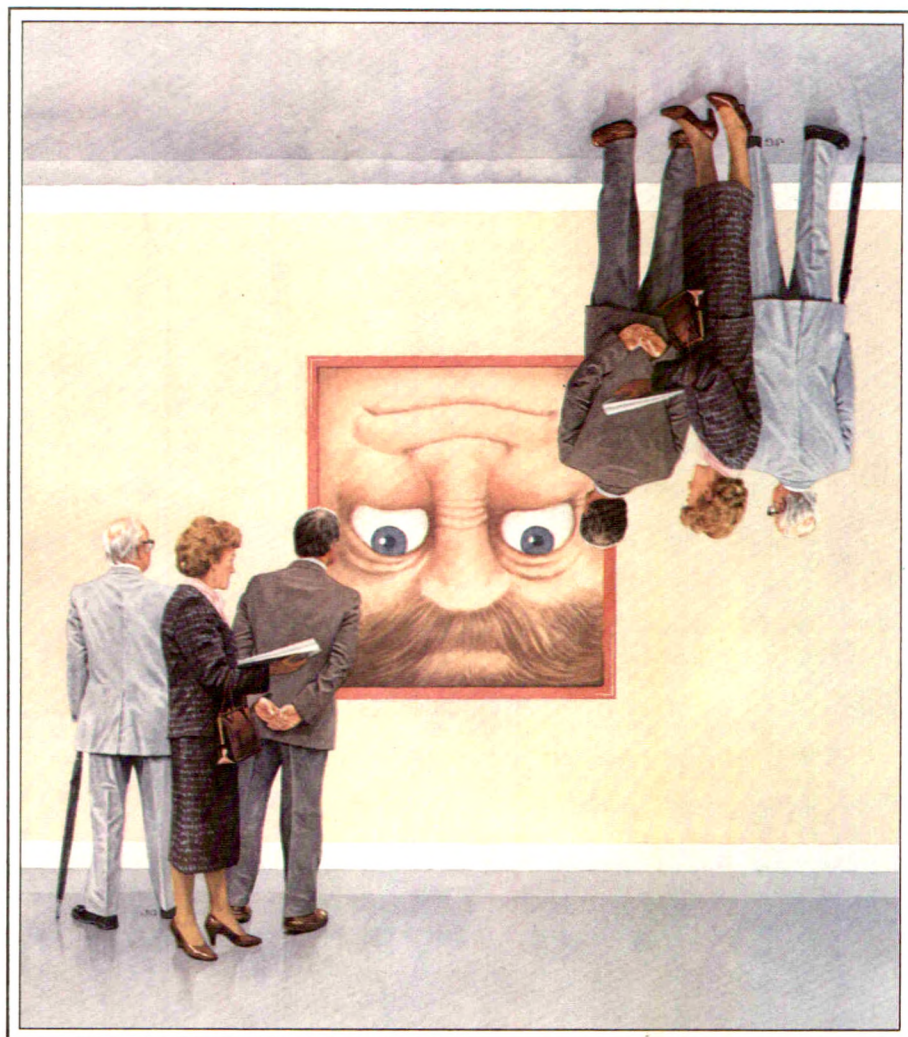
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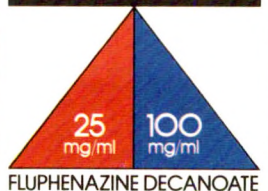


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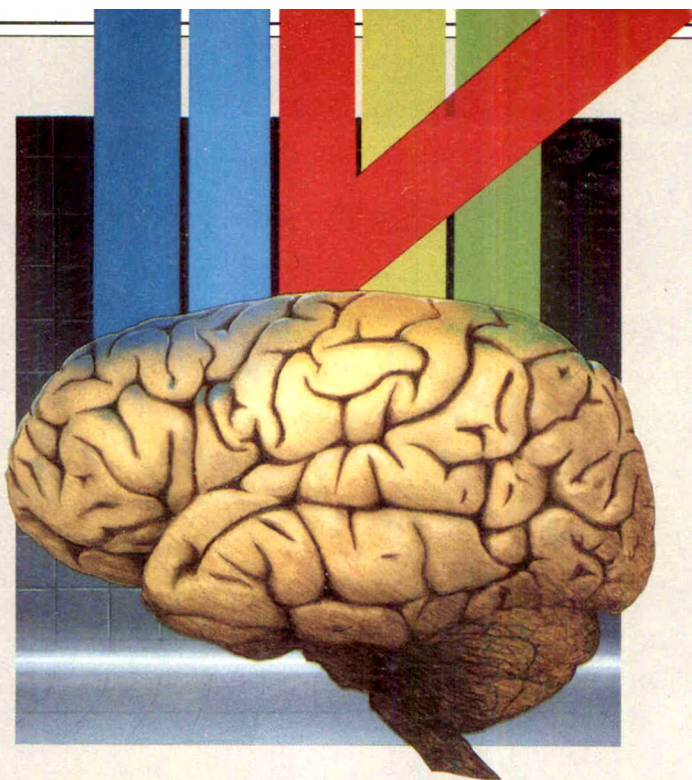
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An American Validation Study of the Newcastle Diagnostic Scale

I. Relationship with the Dexamethasone Suppression Test

M. ZIMMERMAN, B. PFOHL, D. STANGL and W. CORYELL

The Newcastle diagnostic index was completed on 159 depressed in-patients, who received the dexamethasone suppression test during their first week in hospital. Patients suffering from endogenous depression had a significantly higher rate of DST non-suppression, were older, were more frequently psychotic, and more frequently lost weight; even after controlling for these variables, DST non-suppression was significantly more frequent in the endogenous group. The relationship between Newcastle scores and the frequency of DST non-suppression was non-linear.

The Newcastle diagnostic scale (Carney *et al*, 1965) has been found to be related to ECT response (Vlissides & Jenner, 1982), response to tricyclic anti-depressant drugs (TCAs) (Rao & Coppen, 1979), and to the dexamethasone suppression test (DST) (Coppen *et al*, 1983; Holden, 1983), yet this scale has received almost no attention in North America. The present report is the first in a series examining its validity, and reports on the relationship between the scale and escape from dexamethasone suppression – a phenomenon which some (but not all) investigators have found to be specific to endogenous depression (Carroll, 1982). Moreover, since two of the ten items on the Newcastle scale (weight loss and nihilistic delusions) may themselves be related to the DST (Asnis *et al*, 1982; Coryell *et al*, 1982; Mendlewicz *et al*, 1982; Targum, 1983; Zimmerman *et al*, 1984), we have examined whether the association between the DST and the scale is due to their mutual association with psychosis or weight loss. Older age may also be associated with both escape from dexamethasone suppression (Nelson *et al*, 1984; Oxenkrug, 1983) and the Newcastle scale (Abou-Saleh & Coppen, 1984), and we therefore, tested whether age is responsible for the significant relationship between the Newcastle scale and the DST.

Method

Throughout a period of one year, the investigators sought the participation of consecutively admitted depressed patients who were at least 18-years old and did not have any of the medical and pharmacological exclusion criteria specified by Carroll *et al*, (1981). In addition, insulin-dependent diabetics (Cameron *et al*, 1984) and patients taking carbamazepine (Privitera *et al*, 1982) were excluded. Only 10% of

the potential participants refused to participate or were mute and unresponsive, and therefore could not give consent.

Faculty members, residents, and medical students obtained psychiatric histories. Two of the authors read these histories, and independently determined whether the patients met the DSM-III (American Psychiatric Association, 1980) criteria for major depressive disorder: those who did not were excluded from the study. This left a sample group of 161 patients. DSM-III criteria were also used to diagnose the patients as psychotic or non-psychotic and bipolar or unipolar; disagreements between rates were resolved by discussion. All diagnoses were made blind to DST results.

Within one week of admission (mean 2.5 days, s.d. 1.6 days), each patient was given 1 mg of dexamethasone at 2300 h; blood was sampled the next day at 0800 and 1600 h. Serum cortisol was measured by radio-immunoassay, using a specific cortisol antiserum obtained from Damon Diagnostics. There was no cross-reactivity with dexamethasone, progesterone, testosterone, or 11-deoxycortisol. The intra-assay and inter-assay coefficients of variation around the cut-off used to classify patients as non-suppressors were 11.1% and 12.2% respectively. Using the same procedure as the depressed patients, 33 normal controls completed the DST. The test's specificity was 97% (one normal was a non-suppressor) with a cut-off of 5 µg/dl, and 91% (three non-suppressors) using a cut-off of 4 µg/dl. Non-suppression, therefore, was defined as a serum cortisol level greater than 5.0 µg/dl at either sampling.

The in-patient psychiatric unit at the University of Iowa is a tertiary care centre, and the majority of patients had received treatment before admission. Unlike many research units, patients are not maintained drug-free for one or two weeks before the DST, but are treated promptly. A previous study from this centre found that medication status was unrelated to DST results (Schlesser *et al*, 1980). In the present sample, only 35 (22.0%) patients received no psychotropic medication during the month prior to admission; 69 (43.4%) patients were taking TCAs; 12

(7.6%) patients were taking monoamine oxidase inhibitors; 23 (14.5%) patients received lithium; 19 (12.0%) patients received other antidepressants such as trazodone or maprotiline; 50 (31.5%) patients took low doses of minor tranquilisers or hypnotics; and 41 (25.8%) patients received neuroleptics. None of the six classes of medication had any effect on DST suppression. For example, 35% of the patients who took a TCA during the month prior to admission were non-suppressors, compared with 36% of those not taking TCAs. Thirteen (37%) of the unmedicated patients were non-suppressors.

Two of the researchers completed the 24-item Hamilton Rating Scale for Depression (HRSD) (Hamilton, 1967), the Beck Depression Inventory (BDI) (Beck *et al.*, 1979), and the Global Assessment Scale (GAS) (Endicott *et al.*, 1976), either on the day the dexamethasone was administered or the day the blood was drawn. These measures were supplemented with questions from the Schedule for Affective Disorders and Schizophrenia (SADS) (Spitzer & Endicott, 1978) on distinct quality of mood, reactivity, and anhedonia. Two patients refused to answer several questions used to score some of the Newcastle items and therefore were excluded from all analyses. The cut-off scores used on the relevant HRSD and SADS items to determine the presence or absence of the Newcastle symptoms are detailed elsewhere (Zimmerman *et al.*, 1985).

A semi-structured life event interview (Zimmerman, 1982), covering the year prior to admission, provided the information to rate the 'no adequate psychogenesis' Newcastle item in 132 (83%) patients. In 91 (57.2%) patients, 'adequacy of premorbid personality' was assessed with a semi-structured interview designed to assess the DSM-III Axis II personality disorders (Pfohl *et al.*, 1984; Stangl *et al.*, 1986). Review of notes and unstructured clinical interactions with the patient were used to rate these two items in the remaining patients.

Data analysis

We used the *t*-test for two group comparisons of continuous variables, a one-way analysis of variance for three group comparisons, and the chi-squared test for comparisons using categorical data. The Mantel-Haenszel procedure was used to examine whether there was a significant association between the DST and the diagnosis of endogenous depression, after controlling for their mutual association with history of weight loss (defined as an HRSD score of 2 on item 16), psychosis or age.

Results

The average age of the 159 patients was 39.6 years (s.d. 16.1 years); 45 patients were male and 114 female. Forty-four (27.7%) of the sample were psychotically depressed. There were 148 unipolar depressives and 11 bipolar depressives. At baseline, the mean 17-item HRSD score was 22.5 (s.d. 6.3), the mean 24-item score was 29.7 (s.d. 8.9), the mean BDI score was 27.6 (s.d. 11.1), and the mean GAS rating was 38.9 (s.d. 8.7).

Using the conventional cut-off for endogenous depression of a Newcastle score greater than 5, patients suffering from endogenous depression ($n=59$) had a significantly higher rate of non-suppression than patients suffering from non-endogenous depression ($n=100$) (47.5% versus 28.0%, $\chi^2=6.16$, $P<0.02$). Moreover, the endogenous depressives were significantly older (45.4 compared with 36.1 years, $t=13.6$, $P<0.001$) were more frequently psychotic (49.1% compared 15.0%, $\chi^2=21.6$, $P<0.001$), and more frequently reported weight loss (72.8% compared 39.0%, $\chi^2=17.1$, $P<0.001$).

We examined the relationship between the DST and Newcastle classification for psychotics and non-psychotics, for patients above and below the median age of 34, and for patients who did and did not report weight loss. For each of these three distinctions, the overall Mantel-Haenszel χ^2 for association was significant. This suggests that these variables, when examined individually, were not responsible for the significant relationship between the Newcastle diagnosis and the DST.

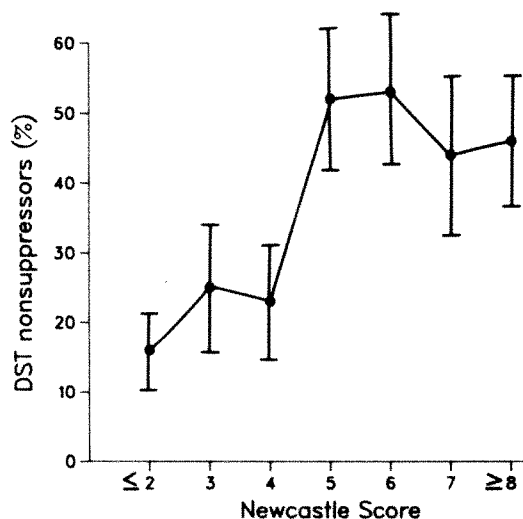


Fig. 1 Relationship between Newcastle scores and DST non-suppression.

Kendell (1982) suggested that two syndromes, such as endogenous and non-endogenous depression, can be distinguished by demonstrating a non-linear relationship between symptomatology and some independent variable such as treatment response, family history, or a laboratory measure. Fig. 1 shows that the association between Newcastle scores and the frequency of DST non-suppression is non-linear. The figure suggests that a cut-off of 4, rather than 5, should be used to distinguish between endogenous and non-endogenous depression. In fact, the 26 patients with a Newcastle score of 4 had a significantly lower non-suppression rate than the 23 patients with a Newcastle score of 5 ($\chi^2=4.45$, $P<0.05$). In comparison

with the findings based on a cut-off score of 5, when a cut-off of 4 is adopted, the non-suppression rate in the non-endogenous depressives is reduced from 28.0% to 20.8%, and the rate in endogenous depressives is slightly increased from 47.5% to 48.8%. When a cut-off of 4 is used, significantly more of the endogenous depressives so defined were non-suppressors (48.8% compared with 20.8%, $\chi^2 = 13.6$, $P < 0.001$). Based on the cut-off score of 4, the endogenous patients are still significantly older (43.8 compared with 35.0, $t = 3.58$, $P > 0.001$), more likely to be psychotic (40.2% compared with 14.2%, $\chi^2 = 13.4$, $P < 0.001$), and more frequently report weight loss (63.4% compared with 39.0%, $\chi^2 = 9.51$, $P < 0.01$). However, none of these variables accounted for the significant association between the DST and diagnosis (all three Mantel-Haenszel χ^2 tests of association were greater than 13.00, $P < 0.001$).

Discussion

Our results replicate the findings of Coppen *et al* (1983) and of Holden (1983), who also found that DST non-suppression was significantly more frequent in endogenous than in non-endogenous depressives, as rated by the Newcastle diagnostic scale. We have extended their results by demonstrating that psychosis, age, and reported weight loss (three variables which have themselves been found to be associated with DST results) are not individually responsible for the significant relationship between non-suppression and endogenous/non-endogenous diagnosis. A cut-off score of 4 maximised the difference between endogenous and non-endogenous groups with respect to DST results. Since the original publication by Carney *et al* (1965), little attention has been given to alternative cut-off scores and their effects on apparent validity.

Since the 1920s, there has been a continuing debate between the dualists, who maintain that endogenous and non-endogenous depression are distinct forms (Gillespie, 1929; Carney *et al*, 1965; Kiloh *et al*,

1972), and the monists, who argue that there is essentially a single disorder, varying only in severity (Mapother, 1926; Lewis, 1934; Kendell, 1969). Several investigators have contributed to this controversy by examining the distribution of symptom scores – a unimodal distribution being viewed as support for the monist position and a bimodal distribution as support for the dualists (Carney *et al*, 1965; Kendell & Gourlay, 1970). Eysenck (1970) cogently argued that the type of distribution is not relevant to the monist-dualist debate, but instead to the separate issue of whether a categorical or dimensional system of diagnosis is more appropriate. More recently, Kendell (1982) noted that a unimodal distribution of symptom scores does not mean that there are no neurophysiological or biological discontinuities. He suggested that a boundary between two syndromes can be established if the relationship between symptomatology and an independent variable is non-linear. We used the DST to search for biological discontinuity. Despite a unimodal distribution of Newcastle scores, their association with the DST was non-linear, and therefore inconsistent with the unitary hypothesis.

The ideal boundary between these groups is another issue. In the present study, a Newcastle score of 4 was the optimal point of discrimination. This finding requires replication both with other independent variables such as family history and follow-up course, and by other investigators using similar independent variables. A two-group classification may not be the most appropriate: cluster analytical studies of depression almost always yield more than two groups (Andreasen *et al*, 1980; Blashfield & Morey, 1979; Paykel, 1971). Examination of other independent variables may reveal that diagnostic groups which are relatively homogenous with respect to DST results are heterogeneous with respect to other aetiological or phenomenological factors.

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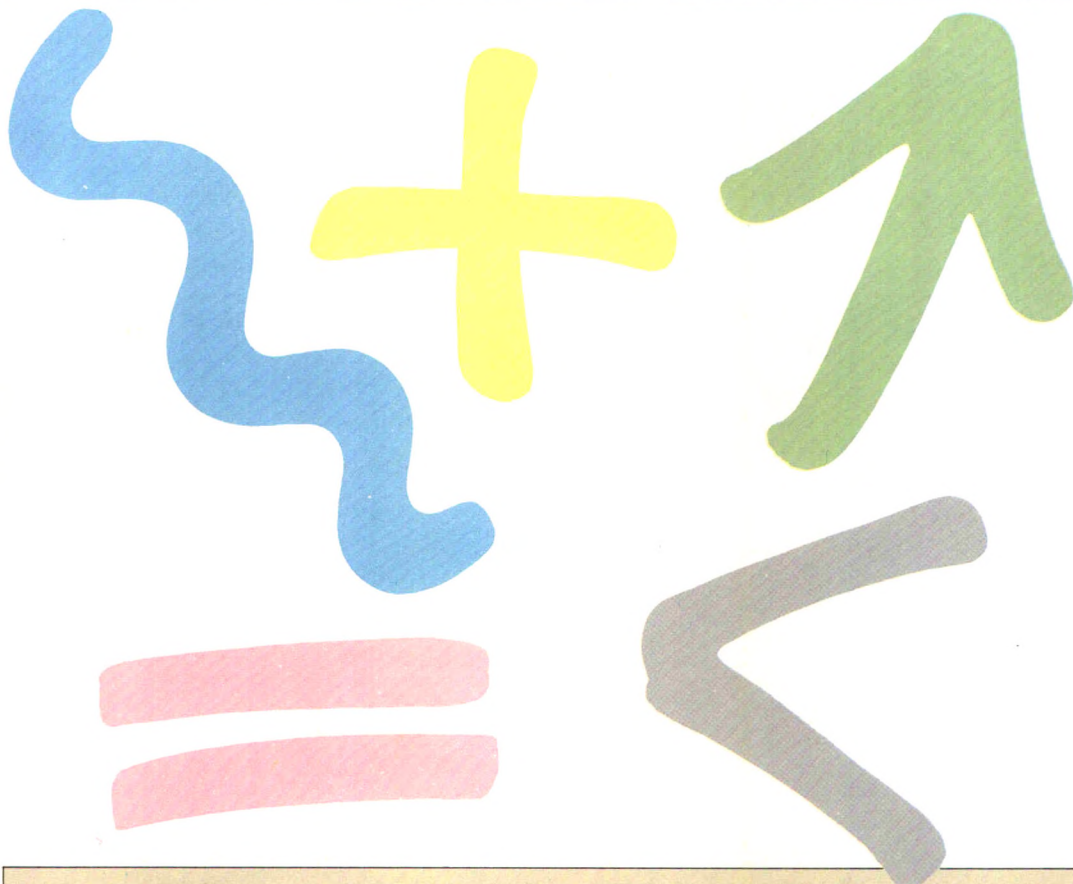
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8. Based on Basic NHS prices at September 1986.

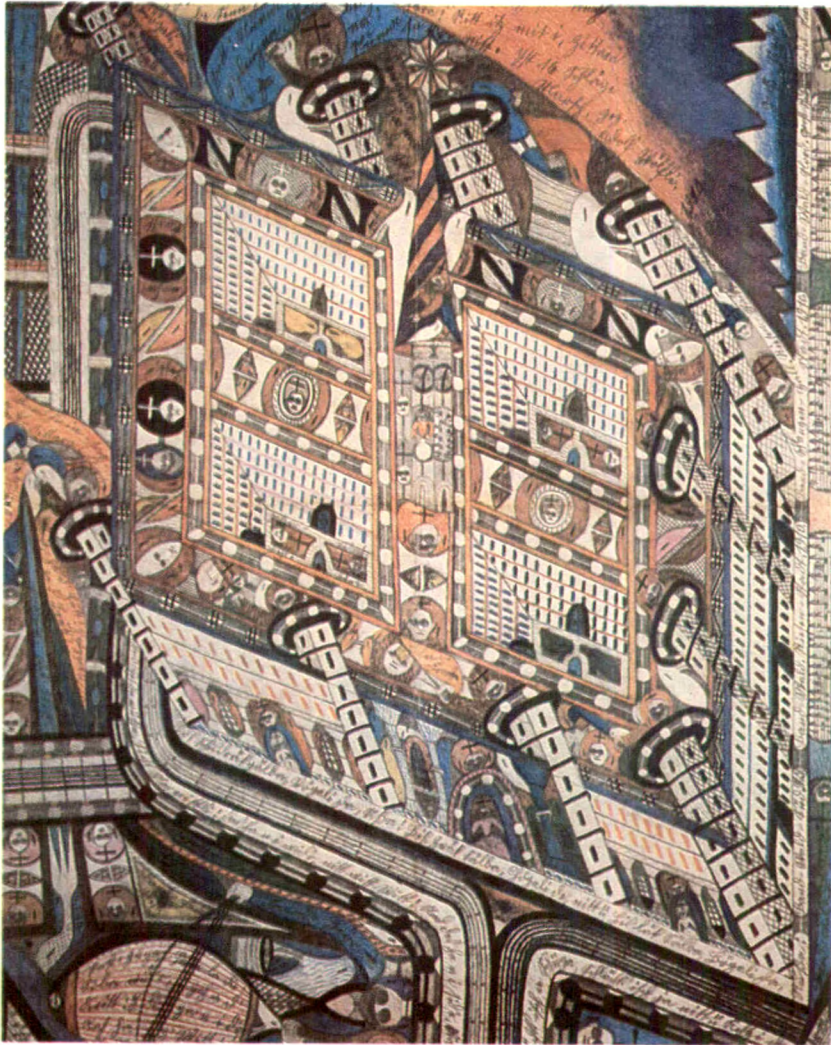


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Reduction in Hypochondriasis with Treatment of Panic Disorder

R. NOYES, J. REICH, J. CLANCY and T. W. O'GORMAN

Hypochondriasis was assessed in 60 patients with panic disorder and agoraphobia using the Illness Behavior Questionnaire. Before treatment, IBQ hypochondriasis scores were similar to those of a group of hypochondriacal psychiatric patients. In patients who improved with treatment, significant reductions in somatic preoccupation, disease phobia, and disease conviction occurred. Hypochondriasis appears to be a prominent feature of panic disorder and agoraphobia, and responds to treatment of the primary conditions. Our findings underscore the importance of providing adequate treatment and thereby avoiding wasteful use of medical resources and alienation of patients from doctors.

Hypochondriacal features have frequently been observed in patients with panic disorder and agoraphobia. Buglass *et al* (1977), for example, reported that irrational fear of bodily disease was present in 53% of the agoraphobic women they interviewed, and Harper & Roth (1962) found hypochondriacal worries in 60% of patients with phobic anxiety-depersonalisation syndrome. Similarly, Sheehan *et al* (1980) found that 68% of a series of patients with endogenous anxiety believed they might be physically ill despite medical reassurance, and Noyes *et al* (1980) found evidence of hypochondriacal tendencies in a series of patients with anxiety attacks. Even though the outcome of these patients was quite favourable, 50% acknowledged worry over health, compared with 24% of non-anxious controls. Apart from these observations, there has been little systematic study of the hypochondriasis that occurs in patients with panic disorder and agoraphobia.

An important question is whether the hypochondriacal features in these patients are symptoms of the disorder or traits of personality found among those who develop anxiety disorders. By itself, hypochondriasis is variously regarded. Some consider it a psychiatric syndrome, while others see it as a form of abnormal illness behaviour; still others regard it a cognitive or perceptual style (Bursky & Klerman, 1983). These conceptualisations fall on both sides of the distinction between state and trait: psychiatric disorders are usually regarded as state phenomena, whereas behavioural disturbances and cognitive style are more commonly viewed as enduring traits. The distinction may be important when it comes to treatment; if hypochondriasis is a state phenomenon, then it is more likely to improve with treatment.

Because hypochondriasis may have detrimental effects on the patient-doctor interaction, it is important to know to what extent it can be modified

by treatment (Pilowsky, 1980). In response to their hypochondriacal concerns, patients with panic disorder tend to offer physical symptoms as chief complaints, and this often causes a psychiatric diagnosis to be missed (Katon, 1984). After an initial examination, continuing fears of physical illness often contribute to poor acceptance of the diagnosis and requests for unnecessary investigations (Clancy & Noyes, 1976). The present study was designed to examine the effect of treatment of hypochondriasis as measured by the Illness Behavior Questionnaire (Pilowsky, 1967).

Method

The subjects in this study participated in a controlled trial of alprazolam versus placebo for the treatment of panic disorder at the University of Iowa. They were volunteers recruited by newspaper advertisement, and were screened using the Structured Clinical Interview for DSM-III developed by Spitzer & Williams (1982). All met revised DSM-III criteria for panic disorder (American Psychiatric Association, 1985), and were experiencing at least one panic attack per week. Of the 60 subjects who completed the study, 5 received a diagnosis of uncomplicated panic disorder, 14 had panic disorder with limited phobic avoidance, and 41 had panic disorder with extensive phobic avoidance (agoraphobia). In terms of ICD-9 categories (World Health Organization, 1980), 19 subjects had anxiety states and 41 had phobic states. The median duration of illness was 4.5 years, and the mean age was 36.4 years. Thirty subjects received alprazolam in a mean dose of 5.8 mg/d and 30 received placebo during the eight-week trial.

At baseline and at the end of six weeks on medication, subjects were asked to complete the Illness Behavior Questionnaire (IBQ) developed by Pilowsky & Spence (1975). This self-report questionnaire contains 52 items that deal with patients' attitudes and feelings about their illness, their view of their social situation, and reactions of significant others towards them and their illness. Fourteen of



TABLE I
Mean scores on the Whiteley Index of hypochondriasis from the Illness Behavior Questionnaire

Group	n	Males		Females	
		Mean	s.d.	Mean	s.d.
Panic disorder patients ¹	32	8.0	2.9	6.6	2.8
Hypochondriacal psychiatric patients ²	100	8.9	5.2	7.9	3.2
Non-hypochondriacal psychiatric patients ²	100	2.9	2.2	3.1	2.1
Malignant disease patients ²	47	3.2	2.7	4.5	3.2
Normal subjects ²	15	1.7 ³	2.4 ³		

1 This study

2. From Pilowsky (1967)

3 Males and females combined

the items, known as the Whiteley Index, have been shown to discriminate between hypochondriacal and non-hypochondriacal patients (Pilowsky, 1967). The Hamilton Anxiety Rating Scale (HARS) was also administered at baseline and weekly during the period of drug administration (Hamilton, 1959). Subjects who showed a decrease of 5 or more points on the HARS were considered to have improved. By this criterion, 32 subjects (14 men and 18 women) improved, and 16 subjects (8 men and 8 women) did not. Mean HARS scores for the improved subjects were 20.3 at baseline and 6.6 at six weeks. Mean scores for unimproved subjects were 18.4 and 19.4.

In order to examine the effect of improvement on the IBQ, we compared the number of improved subjects answering 'yes' to an item at baseline and 'no' to that item at six weeks with the number answering 'no' at baseline and 'yes' at six weeks. Subjects answering a question the same way on both occasions were excluded from the analysis. The sign test was used to examine the probability of obtaining the observed responses, assuming that subjects had an equal chance of responding in one of the two sequences described.

Results

Subjects with panic disorder obtained relatively high scores on the Whiteley Index. The mean number of hypochondriacal items affirmed by panic disorder subjects at baseline was 7.2 (s.d. 2.9), compared with 1.7 (s.d. 2.4) for a small series of normal subjects previously reported. Table I provides a comparison of scores on the Whiteley Index obtained from several groups, including hypochondriacal and non-hypochondriacal psychiatric patients, patients with malignant disease, and normal subjects, all reported by Pilowsky (1967). It shows mean baseline scores for male and female panic disorder patients that were nearly as high as those for hypochondriacal psychiatric in-patients. At baseline, a substantial proportion of panic disorder subjects affirmed items belonging to the dimensions of hypochondriasis measured by the 14-item scale, including bodily preoccupation, disease phobia and disease conviction (Table II).

A statistically significant reduction in scores on the Whiteley Index was observed after six weeks of treatment with alprazolam. Among improved subjects the mean fell from 7.2 (s.d. 2.9) at baseline to 4.4 (s.d. 2.9) after six weeks ($P < 0.0001$). In subjects who failed to improve, no statistically significant change occurred: the means were 6.6 (s.d. 4.0) at baseline and 5.6 (s.d. 4.6) after six weeks. Scores for three dimensions of hypochondriasis obtained by previous factor analysis were also significantly reduced in improved subjects after six weeks (Pilowsky, 1967). The mean score for bodily preoccupation (four items) dropped from 2.1 (s.d. 0.9) to 1.2 (s.d. 1.0) ($P < 0.01$), the score for disease phobia (three items) fell from 2.2 (s.d. 1.2) to 1.6 (s.d. 1.2) ($P < 0.02$), and that for disease conviction (three items) changed from 1.0 (s.d. 0.9) to 0.5 (s.d. 0.6) ($P < 0.01$).

Statistically significant changes were observed between baseline and six weeks on eight items dealing with bodily preoccupation, disease phobia and disease conviction (Table II). Many represented nearly 50% reductions in the proportion of subjects who reported 'worry about health', 'worry about serious illness', 'worry about disease brought to attention', 'something seriously wrong with body', 'bothered by many different symptoms' and 'bothered by many aches and pains'. Significant reductions at six weeks were also seen in 'silly thoughts about health', 'something seriously wrong with mind', 'more susceptible to illness than others' and 'upset with appearance'.

In contrast to this, relatively little change was seen in the proportion of subjects reporting interpersonal alienation as a consequence of illness. As is shown in Table II, treatment had relatively little effect on the response to three hypochondriacal items from the Whiteley Index having to do with interpersonal interactions surrounding illness: 'illness not taken seriously by others', 'annoyed by being told you look better' and 'hard to believe the doctor that there is nothing to worry about'. When we examined the response of improved subjects to all the items that might reflect interpersonal alienation, including several suggesting alienation from doctors, we found that although the response to 13 of the 16 items decreased with treatment, only one, 'illness affects getting along with others', showed a statistically significant reduction.

TABLE II
Response of improved subjects to the Whiteley Index: percentage answering "yes"

	Baseline	Six weeks
<i>Bodily preoccupation</i>		
Aware of bodily sensations	84.4	65.6*
Bothered by many different symptoms	75.0	37.5*
Bothered by many aches and pains	50.0	18.8*
<i>Disease phobia</i>		
Afraid of illness	71.9	59.4***
Easy to forget about self ¹	68.7	40.6**
Worry about health more than most	56.3	50.0***
Worry about disease brought to attention	24.0	6.3**
<i>Disease conviction</i>		
Something seriously wrong with body	43.8	9.4*
Illness not taken seriously by others	41.9	29.0***
Annoyed by being told you look better	12.5	9.4***
<i>Other</i>		
Worry about health	75.0	40.6*
Worry about serious illness	62.5	34.4*
Hard to believe doctor, that there is nothing to worry about	34.4	28.1***
Have symptoms of serious disease	21.9	15.6***

¹ Percentage answering "no"

* $P < 0.01$; ** $P < 0.05$; ***Not significant

Discussion

Our findings proved confirmation of a high level of hypochondriasis in subjects with panic disorder and agoraphobia. In fact, the scores we observed using the Whiteley Index of the IBQ were nearly as high as those previously obtained from a group of psychiatric patients that had been selected for hypochondriacal features. Also, the response to individual items from the IBQ indicates that a substantial proportion of persons with panic disorder and agoraphobia experience bodily preoccupation, disease phobia, and disease conviction, all of which are major characteristics of hypochondriasis (Pilowsky & Spence, 1983). In our population, these features were present despite repeated reassurance from doctors concerning the absence of physical disease.

We have demonstrated that the hypochondriasis associated with panic disorder and agoraphobia responds to treatment. A substantial reduction in IBQ scores was observed after six weeks of treatment, and this was accompanied by a clinically apparent decrease in hypochondriasis. Indeed, many subjects reported that treatment had relieved their preoccupation with bodily sensations, their fear of physical disease, and their belief that they might be

physically ill. An examination of individual items showed that these dimensions of hypochondriasis were all strongly influenced by the treatment. Because responses to the IBQ remained relatively stable among unimproved subjects, it is unlikely that non-specific factors such as repeated testing, medical contact, or emotional support could account for the changes found. In fact, the observed effect might have been greater had anxiety symptoms been eliminated by the treatment administered; some patients remained symptomatic despite substantial improvement.

There are several possible explanations for the association between anxiety and hypochondriasis observed in this study. The first is that anxiety may be associated with distorted cognitive assessment of health status. This possibility is supported by Beck *et al* (1974), who found anticipation of physical harm proportionate to the level of anxiety in patients with anxiety disorders. A second possibility is that anxiety may be accompanied by a state of heightened alertness, resulting in greater awareness of physiological processes; this possibility is supported by Tyrer *et al* (1980), who found anxious patients to be more aware of their heartbeats than normal controls. Anxiety may also be associated with physiological

arousal—a third possibility that is supported by physiological investigations (Lader, 1978). Autonomic nervous system activation can produce symptoms causing anxious patients to suspect malfunction of many organs, including the heart. In fact, all three of these possibilities have been described as mechanisms contributing to hypochondriasis (Bursky & Klerman, 1983).

Our findings suggest that the association between hypochondriasis and panic disorder may be an intimate one; hypochondriasis may in fact be an inherent aspect of the anxiety syndrome. Various authors have noted that hypochondriacal concerns are a consistent part of the clinical picture of anxiety states (Harper & Roth, 1962; Buglass *et al*, 1977; Noyes *et al*, 1980; Sheehan *et al*, 1980). Also, during panic attacks patients describe an overwhelming experience of hypochondriasis; at the height of an attack some patients report not only intense fear but a belief that their death, due to some catastrophic event, is imminent. This belief is based on the symptoms of a massive sympathetic discharge that suggests impending collapse. Many patients go to the nearest emergency room in search of life-saving interventions. All this occurs despite medical reassurance, and represents hypochondriasis in its most intense form.

It is clear from this study that the measurement of hypochondriasis is influenced by state anxiety. This means that valid assessment of hypochondriasis in panic disorder patients must take the level of anxiety, and perhaps depression, into account. Completely eliminating state anxiety in such patients may be difficult because of the chronicity of the disorder. With modern drug treatment, however, a proportion of patients become virtually free of symptoms, at least for the duration of the treatment. Consequently, re-

search to determine whether hypochondriasis is unique to anxiety disorders and the extent to which it is state-dependent in such patients appears to be possible. A comparison of anxious and depressed patients should yield information on the question of specificity.

Our findings underscore the importance of adequate treatment for patients with panic disorder and agoraphobia. They show that hypochondriasis is a treatment-responsive feature of this illness. Without treatment, hypochondriasis often leads to repeated consultations, examinations and laboratory evaluations in search of a physical explanation for symptoms (Clancy & Noyes, 1976). These activities not only waste medical resources but often leave patients even more hypochondriacal than before. Hypochondriasis is a stumbling block to the diagnosis and management of disorders that effective treatment might remove.

Our anxious patients, reported a degree of interpersonal alienation that diminished only slightly with a lessening of their anxiety. These patients typically reported that others do not regard them as ill or take their illness seriously; indeed, their own attitude towards their condition is often changeable and uncertain. At one moment they believe themselves to be seriously physically ill, while at the next they suspect that they are not ill at all, and should simply get control of themselves.

Successful treatment of anxiety disorders needs to take the hypochondriasis of the patients into account.

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Psychogenic Skin Disease: A Review of 35 Cases

N. P. SHEPPARD, S. O'LOUGHLIN and J. P. MALONE

In three years of clinical practice, the authors saw 35 cases of dermatological disorders of strictly psychological origin—8 patients with *dermatitis artefacta*, 8 with delusional parasitosis, and 19 who presented with skin complaints but showed no dermatological pathology ('dermatological non-disease'). All but two initially presented for dermatological opinion rather than psychiatric assessment, and 12 refused psychiatric referral. Demographic and clinical details of all 35 cases are given, including possible related factors, course, treatment, and outcome, and the cases are discussed in the context of the existing literature. Liaison between dermatologists and psychiatrists is strongly advocated.

The association between disorders of the psyche and those of the skin has been extensively studied. Evidence has been produced suggesting a higher prevalence of psychiatric disorder in dermatology out-patients and in-patients than in the general population and the general medical in-patient population respectively (Hughes *et al*, 1983).

A variety of mechanisms have been postulated as being of aetiological importance in psychogenic skin disease. These range from classical Freudian ideas of hysterical conversion mechanisms to ideas of the skin as an infantile preverbal means of communicating emotions which is brought back into operation at times of regression, or is repeatedly utilised by those who have failed physically or psychologically to develop the language to express feelings. There are also biological theories of specific neurophysiological disturbances.

In a review of psychosomatic aspects of dermatology, Koblenzer (1983) has offered a working classification for psychocutaneous disease which emphasises approach to treatment. She lists three main categories of conditions:

1. Conditions that are strictly psychological in origin
2. Conditions in which strong psychogenic factors are imputed, e.g. chronic urticaria and hyperhidrosis.
3. Conditions that are dependent on genetic and environmental factors, but in which the course of disease is substantially affected by stress, e.g. psoriasis, atopic dermatitis, and recurrent herpes simplex.

Eller (1974) describes a fourth category, i.e. skin disorders that are likely to affect the psyche, e.g. those associated with disfigurement, chronic pain, or itching.

In the first category, with which this paper is concerned, Koblenzer includes conditions such as *dermatitis artefacta*, delusions related to the skin, psychogenic purpura syndrome, and dermatological non-disease. Although these disorders occur relatively infrequently, early recognition is important for several reasons. Investigation of them is often extensive, repeated, and highly expensive, and may be a strong reinforcer of what can be regarded as abnormal illness behaviour. Ultimately, however, their prime importance lies in the fact that they may be a source of severe distress and extreme social disruption to those who suffer from them. It is our experience with this group of patients that we wish to describe.

Method

Patients with conditions falling into the first of Koblenzer's categories were collected by the authors in the course of their ordinary clinical practice over a three-year period. In all, 35 such patients were seen. The vast majority (all but two) had initially presented for a dermatological opinion rather than psychiatric assessment. Data on sex, occupation, age at presentation, nature and duration of symptoms, possible related factors, course, and outcome were obtained.

Patients were allotted to three main diagnostic groups:

- (i) *Dermatitis artefacta*, which may be defined as any form of self-inflicted skin injury in which the patient denies producing the lesions (Eller, 1974)
- (ii) *Delusional parasitosis*, in which patients have an erroneous but unshakeable belief that their skin is infested by parasites (Gould & Gragg, 1976)
- (iii) *Dermatological non-disease*, a common and serious disorder of cutaneous body image in which the patient presents with dermatological complaints but with no significant dermatological pathology on examination.

TABLE I
Summarized case histories of eight patients with dermatitis artefacta

Case number	Sex	Marital status	Occupation	Age (years)	Duration of symptoms	Type of lesion	Possible related factors	Outcome
1	F	Single	Bank official	35	3 months	Bruising and swelling of right knee	Previous episode of hysterical aphonia after father's death Frustrated career ambitions	Lesions cleared on occlusion, but further lesions developed which also cleared up. Refused psychiatric help
2	F	Married	Housewife	30	3 months	Ulcer on right ankle and below right knee	Previous treatment for depression after discovery of husband's infidelity with patient's sister	Lesions cleared in hospital; re-admitted one month later with further lesions. Cleared on occlusion Seen by psychiatrist, who treated her for depression
3	F	Single	Waitress	49	2 months	Erosive, weeping, non-pruritic malar rash	Work problems Content to remain in hospital	Initially treated as a secondary bacterial infection superimposed on a herpes simplex infection: no response. After six weeks, patient was observed by staff scratching lesions with a coat hanger. On confrontation, patient denied factual element and refused psychiatric referral. Lesions healed in one week
4	F	Married	Housewife	67	10 years, recurring	Sharply demarcated ulcer, dorsum of left hand	Long history of endogenous depression in a vulnerable personality, with a variety of environmental factors (including diabetes mellitus and family dysharmony) aggravating the situation	Lesion healed on occlusion
5	F	Single	Schoolgirl	10	14 months	Temporo-parietal hair-loss	History of school difficulty	Diagnosed as trichotillomania. Referred to child psychologist. Partial improvement after family therapy
6	F	Married	Housewife	?	2 months	Blisters on chest	Long history of marital dysharmony. Recently upset by a planned wedding in the family	Initially diagnosed as pemphigus. Attempted to enlist daughter to bring a bottle from home into hospital: husband intervened and discovered it was paranoiac. Discharged herself before a psychiatric evaluation could be carried out
7	F	Single	Unemployed	29	9 years	Purpura, painful, occurring spontaneously over a few hours, usually involving arms, legs, occasionally trunk, but sparing face	Dull, immature girl with restricted social life and dependent relationship with relatives, conflict re illegitimacy, family history of affective disorder	Initially queried as Gardner Diamond syndrome, but negative result to injection of autologous blood. Continued observation revealed factual aetiology. Referred for psychotherapy
8	F	Single	Schoolgirl	16	6 months	Bruising of right arm, shoulder to elbow	Bruising started following a fall on ice, when she injured an elbow. Subsequent crops of non-painful bruises, did not occur in any other site All investigations negative	Initially diagnosed as Gardner Diamond syndrome on clinical grounds. Injection of autologous blood not performed. Subsequently, diagnosis changed to factitial disease Refused psychiatric referral

TABLE II
Summarised case histories of eight patients with delusional parasitosis

Case number	Sex	Marital status	Occupation	Age (years)	Duration of symptoms	Nature of infestation	Possible related factors	Outcome
1	F	Married	?	48	?	"Things crawling all over me"	Previous history of recurrent visions of Christ and presence of stigmata on her hands 12-year history of unexplained pruritus vulvae	Seen initially by psychiatrist, who diagnosed "Hysterical, no treatment needed". Subsequently seen by dermatologist, who diagnosed delusional parasitosis
2	M	Single	Unemployed	54	2 years	Lice, initially in eye-brows but later spreading over entire body		Not seen by psychiatrist. Partial response to pumozide
3	M	Single	Accountant	56	15 years	Fleas, two varieties resembling wood-lice	History of early paternal loss, work difficulty, and residual disability after polio	Seen by psychiatrist, who diagnosed monosymptomatic encapsulated delusional disorder. Good response to pumozide and imipramine
4	F	Married	Retired teacher	79	6 years	Insects biting and itching	Estranged from family because of delusions. Moved from house for fear of contaminating family	Not seen by psychiatrist. Fully cured after direct firm confrontation by dermatologist
5	M	Single	Farmer	30	1 year	Insects infesting glans penis	Dull normal intelligence. Planning marriage. Guilt feelings re previous history of bestiality	Refused psychiatric help
6	M	Single	Labourer	62	2 years	Insects crawling under his skin	Previously diagnosed as hairy cell leukaemia. Referred by haematologist to dermatology clinic with a skin rash caused by applying TCP to his skin several times a day. Had been spraying his room with insecticides and insect repellants	Refused psychiatric intervention. Problem persists
7	M	Single	Unemployed	62	3 months	Lice crawling over him. O/E rash secondary to excoriation and application of disinfectants	Blind. Previous history of recurrent depression and alcohol abuse. Seen by dermatologist. Diagnosed as "neurodermatitis"	Resolved with treatment of depression
8	F	Widow	Housewife	52	3 months	"Live things" coming from her legs, which "could milk too much oil out of my skin in the bath". Produced a matchbox of "parasites"; in reality skin squames	Widowed and socially isolated. History of streptomycin-related hearing impairment	Accepted psychiatric referral. Depression diagnosed. Improved on antidepressant medication

TABLE III
Summarised case histories of 19 patients with dermatological non-disease, grouped according to symptomatology (facial/scalp or hair/genital/other)

Case number	Sex	Marital status	Occupation	Age (years)	Duration of symptoms	Nature of symptoms	Possible related factors	Outcome
Facial								
1	M	Single	Teacher	28	3 years	Facial redness O/E N.A.D	Long-standing ambivalent relationship with mother. Career problems. Marked obsessional traits	Minimal improvement with brief psychotherapy
2	M	Single	Factory worker	24	5 years	Facial redness. O/E N.A.D	Felt colleagues and girlfriends repeatedly remarked on his face. Anxious, sensitive, with marked feeling of inferiority	Refused psychiatric referral. No improvement after two years
3	M	Single	Farmer	25	3 years	Facial redness. O/E N.A.D	Aggravated by stress. Socially embarrassing. No response to topical steroids and tetracycline	Refused psychiatric help. Self-referral to a Harley Street plastic surgery clinic, where he was offered dermabrasion
4	F	Single	Secretary	46	"Several years"	Facial redness and tingling	Socially withdrawn. Previous history of depression treated with antidepressants	Some improvements with supportive measures
5	F	?	?	55	20 years	Facial burning		Refused psychiatric help
6	M	Married	Cook	35	15 years	Nose red and mis-shapen. O/E N.A.D.	Previous plastic surgery, 10 years before, felt it made him worse. Wanted further surgery	Depressed and anxious. Refused psychiatric help. No change after two years
Scalp/hair								
7	M	Single	Trainee accountant	25	"few years"	Scalp soreness. O/E N.A.D	Aggravated by stress. Associated loss of interest in work. Mother had history of a similar complaint some years previously during a stressful time	Accepted psychiatric referral, but symptoms had resolved when seen
8	N	Single	Salesman	20	8 months	Scalp tenderness and hair loss. O/E N.A.D.	Marked anxiety symptoms related to seeking work	Symptoms improved as anxiety abated
9	M	Single	Civil servant	21	1 year	Hair loss. Hot stinging of scalp on entering warm environment. O/E N.A.D.	History of neurotic traits in childhood. Blamed a "bad haircut". Career dissatisfaction	Seen by psychiatrists. Symptoms noted as "almost delusional in intensity". Improved without treatment, although remained convinced that a change had occurred
10	M	Single	Trainee pilot	22	2 years	Hair loss. O/E N.A.D.	Originally from Libya, working in Ireland for 2 years. Home-sick and lonely	Improved with reassurance and support

TABLE III
Continued

Case number	Sex	Marital status	Occupation	Age (years)	Duration of symptoms	Nature of symptoms	Possible related factors	Outcome
11	M	Single	Unemployed	43	16 years	Hair loss O/E male pattern baldness	Pre-occupied with hair loss. Had sought numerous dermatological opinions. Quit work and had been housebound for 15 years, enabled by over-protective, over-involved parents. Referred to psychiatrist for assessment for disability pension	Saw psychiatrist, but refused intervention
12	F	Single	Unemployed	30	Several years	Hair loss. O/E N.A.D.	Previously diagnosed and treated for schizophrenia	No improvement
13	F	Widow	Unemployed	53	1 year	Change in texture and colour of hair	Blamed change on a "bad perm"	Accepted psychiatric referral. Diagnosed as severe endogenous depression. Scalp symptoms fluctuate with mood, but still persist
Genital								
14	M	Married	Traveller	50	2 years	Scrotal itch and redness	Marked anxiety precipitated by guilt over extra-marital sexual contact	No marked improvement with reassurance
15	M	Married	Mechanic	55	1 year	Penile itch and redness	Guilt over extra-marital sexual contact. Concerned that he had contracted genital herpes	Improved with reassurance
16	M	Single	Farmer	23	?	Scrotal itch and burning	Anxiety <i>re</i> previous sexual contact. Feared he had a venereal disease	Improved with reassurance
17	M	Single	Fitter/welder	26	2 years	Scrotal itch	Anxiety <i>re</i> previous sexual contact. Feared he had a venereal disease. Marked obsessional traits	Accepted psychiatric referral. Currently attending psychiatrist
Miscellaneous								
18	M	Single	Medical student	22	2 years	Emitting foul odour. O/E N.A.D.	Felt friends had withdrawn because of odour	Refused to accept diagnosis. Diagnosed as personality disorder. Refused advice or psychiatric referral
19	F	Married	Housewife	34	1 year	Excessive axillary sweating. O/E N.A.D.	Associated severe anxiety symptoms. Wanted surgery for her problem	Refused advice or psychiatric referral

Results

We saw eight patients with artefactual skin disease (Table I). All eight were female. Age at presentation was ascertained for seven of the patients and ranged from 10 to 67 years, with a mean age of 29.5 years. The duration of symptoms at time of presentation ranged from two months to 10 years. The nature of the lesions varied from patient to patient, but included bruising, blistering, ulceration, skin rashes, and hair loss. All the lesions healed after occlusion, close observation of the patient, or confrontation.

Of eight patients presenting with delusions of infestation (Table II), five were male. Age at presentation ranged from 30 to 79 years, with a mean of 55.4 years. The duration of symptoms was determined in seven cases, and ranged from three months to 15 years, with an average of 4.4 years. Two patients had extensive previous psychiatric histories, and all had suffered marked social disruption because of their symptoms. Two patients were treated with pimozide, and showed some improvement.

We saw 19 patients with dermatological non-disease (Table III)—14 of whom were male. Age at presentation ranged from 20 to 55, with a mean of 30.7 years. Duration of symptoms was calculated accurately in only 14 cases: it ranged from eight months to 20 years, with a mean of 5.3 years. Symptomatology fell into three main groups: (1) facial symptoms; (2) scalp and hair symptoms; and (3) genital symptoms. Psychiatric diagnoses in these cases of dermatological non-disease included dysmorphophobic disorder, schizophrenia, major affective disorder, and anxiety states.

Discussion

Dermatitis artefacta

It is characteristic of *dermatitis artefacta* that the lesions, single or multiple, are bizarre in configuration and appearance, and are confined to areas accessible to the hands. A variety of means may be used to produce the lesions: noxious substances may be applied, or the angular border of a lesion may suggest the nature of the instrument used to produce it. Diagnosis is confirmed by showing that the lesions heal under efficient occlusion.

Rather than regarding *dermatitis artefacta* as an isolated syndrome, most authorities now see it as part of a wide spectrum of self-induced disease. Sneddon & Sneddon (1975), in their follow-up study of 43 patients with self-inflicted skin-lesions, showed that in the majority of cases, *dermatitis artefacta* was only one episode in a long history of psychogenic illness, and noted that recovery was linked to changes in patients' life circumstances rather than to formal treatment. In our series, four patients had previous histories of formal psychiatric problems (two with depression and one each with hysterical conversion and school refusal), while all eight were under signifi-

cant psychological stress at the time of production of their symptoms.

Lyell (1976) has reviewed possible reasons for the reluctance of doctors to make a diagnosis of *dermatitis artefacta*. They include an inability to believe that patients, especially the respectable and the intelligent ones, might wilfully deceive them; while fear of missing organic disease (coupled with the fact that the bizarre lesions suggest rare diseases or unfamiliar manifestations of more common ones) leads the doctor to continue the search for a physical cause. This is amply illustrated by Case 1, in which the patient had approximately £2000-worth of consultations and investigations before the correct diagnosis was arrived at.

Psychogenic purpura syndrome (Gardner-Diamond syndrome) was considered in the differential diagnosis of two cases (7 and 8) listed in Table I. This is an unusual and rare syndrome, distinguished by crops of painful ecchymoses and multiple systemic complaints. It was first described by Gardner & Diamond (1955). They were impressed that their patients (four women) had sustained physical injuries shortly before the onset of symptoms. They postulated that as a result, the patients had become sensitised to their own erythrocytes, and that subsequent extravasation of blood during the unrecognised trivial injuries of everyday life induced the purpuric lesions. Gardner & Diamond reproduced the typical bruises of what they called 'auto-erythrocyte sensitisation' by the intracutaneous injection of the patients' own blood, determining that the factor responsible for the reaction was present in the red cell stroma. Ratnoff & Agle (1968), re-evaluating the syndrome, commented on the severe emotional disturbances antedating the first purpuric lesions in all their patients. Furthermore, in a third of the patients they studied, the lesions could not be reproduced by injection of autologous blood. Ratnoff & Agle felt that the psychological factors might play a critical role in pathogenesis, and suggested renaming the syndrome 'psychogenic purpura' syndrome. As mentioned previously, this diagnosis was considered in two cases in our series, initial symptomatology in both cases corresponding closely to descriptions of the condition in the literature. However, the diagnosis was rejected on both occasions when continued observation led to direct evidence of a factitious aetiology.

Delusional parasitosis

The term 'delusional parasitosis' is preferable to older terms such as 'parasitophobia' and 'acaraphobia', which have been criticised for implying a

phobic rather than a delusional mechanism underlying the condition, and because a variety of infective agents other than acari are implicated by the patients.

The typical patient with delusional parasitosis is aged 40 or over and is female, the sex ratio being generally reported as 3:1, although five of the seven patients in our series were males. Patients usually present with detailed descriptions of the insects and of their burrowing, biting, or otherwise harming the patient. There may be physical evidence of excoriation or the use of antiseptics to eradicate the infestation, and the patients often produce spurious 'samples' in support of their claims. Patients frequently seem antagonistic towards medical staff, and vehemently oppose suggestions that they might be mistaken or that they should seek psychiatric help.

It has been maintained that there is a difference between patients who initially present to a dermatologist and those who are referred directly to a psychiatrist, the latter being more likely to have a recognisable psychiatric disorder. However, allocation of these patients to distinct psychiatric categories has been generally noted to be difficult, with patients receiving diagnoses as varied as affective disorder, paranoid schizophrenia (mostly older patients), and monosymptomatic hypochondriacal psychosis.

In making a diagnosis of delusional infestation, the necessity of ruling out actual infestation or organic illness such as vitamin B₁₂ deficiency, pellagra, or disseminated sclerosis needs to be stressed. Having said this, patients with delusional parasitosis are notoriously difficult to treat. Gould & Gragg (1976), rejecting earlier more pessimistic attitudes to treatment, have suggested sensible guidelines for the psychological management of these patients, primarily by the dermatologist. This model is essentially one of simple supportive psychotherapy. More recently, reports of successful treatment of the condition with pimozide have appeared (Riding & Munro, 1975; Reilly *et al.*, 1978). Delusional parasitosis has been well reviewed by Munro (1980).

Dermatological non-disease

The importance of the condition called 'dermatological non-disease' lies in the extreme distress it causes the patient. It may be socially crippling, and on occasion results in suicide. Patients may spend small fortunes in pursuit of a 'cure'.

Cotterill (1981), in a review of 28 patients with dermatological non-disease, found that in general, symptomatology was confined to three main sites—

the face, the scalp, and the genitalia. Only two of his patients presented with symptoms outside these areas. The distribution of the symptomatology amongst our series of patients was similar: six patients with complaints of facial abnormality, seven with scalp complaints, and four with genital complaints. Of the two remaining patients, one complained of abnormal body odour and the other of excessive axillary sweating.

The commonest facial symptoms in Cotterill's group were complaints of itching and burning, and a preoccupation with imagined facial hair. He noted this sub-group to be predominantly female, and felt they had the most ominous prognosis, three of his patients ultimately committing suicide. Of our own group of six patients reporting facial symptoms, four were male. They complained mainly of facial redness, with tingling and burning in some cases.

Amongst our group of patients with scalp symptoms, the commonest complaints were of excessive hair loss, and tingling or itching of the scalp. This is in keeping with Cotterill's experience. One patient who was subsequently diagnosed as having severe endogenous depression complained of a change in the colour and the texture of her hair. It is interesting to note that this patient and one other dated their symptoms from visits to hairdressers whose work they were dissatisfied with.

All four of our patients with genital symptoms were male. The commonest complaint was of scrotal itching, and in each case the symptom appeared to have been precipitated by a sexual encounter which had left the patient feeling guilty. All four initially feared that they had contracted a venereal disease. Symptoms improved with reassurance in two cases, but persisted in the remaining two. Again, these findings are very much in line with those of Cotterill (1981).

Cotterill noted that the majority of his patients could initially be given a diagnosis of dysmorphophobia. This is a condition in which sufferers become concerned and preoccupied with some external physical defect which they think is noticeable to other people, whereas objectively speaking they have no cause for complaint, their appearance being well within normal limits. It is essentially a disorder of the 'psychological' body-image (Hay, 1970). The question of whether dysmorphophobia involves a delusional conviction or a neurotic, hypochondriacal preoccupation has been extensively debated. In his review on the subject, Hay concluded that dysmorphophobia as a symptom was non-specific, that it could occur in different psychiatric syndromes, and that it was prone to occur in subjects with sensitive or insecure personalities. Cotterill stressed that the

commonest final psychiatric diagnosis in those with scalp symptoms was depressive illness.

Conclusions

Far from regarding these conditions as exotic rarities, we feel that they form an important and common cause of psychiatric morbidity (particularly dermatological non-disease). As mentioned above, early recognition and intervention is important. Who

should be the primary agent in the treatment of these patients? The fact that most of the literature concerning these disorders is to be found in the dermatological rather than the psychiatric literature emphasises the reluctance of many of these patients to avail themselves of psychiatric help, and the efforts of dermatologists to offer psychological help. We feel that this is an important area where liaison between psychiatrist and dermatologist can only benefit the patient.

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A Clinical Study of Gilles de la Tourette's Syndrome in Korea

Clinical data on 30 Korean patients of the authors with Gilles de la Tourette's syndrome are described, as well as data on seven other Korean cases from the literature. The overall characteristics and EEG findings were similar to those of Western patients, but there were fewer obsessive-compulsive behaviour problems, and family histories were less often positive.

Gilles de la Tourette's syndrome is an unusual condition, characterised by the onset in childhood or adolescence of recurrent, involuntary, repetitive, and rapid movements which include multiple vocal tics. Coprolalia, copropraxia, echo-phenomena, and obsessive-compulsive traits may also be present, but are not diagnostic prerequisites. Aetiologically, it has been considered at least partly a psychogenic disorder, but neurophysiological studies suggest that its basis is neurological (Shapiro & Shapiro, 1982), while Lees *et al* (1984) have reported evidence for heterogeneity.

Although many cases have been reported in western countries over the past 100 years, there have been fewer than fifty from Asia in the English-language literature. Only about ten reports of Asian cases could be found in the Australian MEDLARS and in Index Medicus. Although one was reported in India in 1962, the first Asian case is generally thought to be that of a Chinese boy in Hong Kong (Singer, 1963). The scarcity of such reports does not necessarily reflect the rarity of the syndrome, but rather the lack of awareness of it, and failure to publish cases in English-language journals (Lieh-Mak *et al*, 1978).

In Korea, six cases have been published since Lee & Sul (1968) reported the first case (that of a seven year-old girl); all of these were in Korean medical journals only. Suh & Park (1969) reported a 14 year-old boy with the syndrome who had suffered from sibling rivalry and a domineering and punishing father. Kim (1971) described a ten year-old boy, in whom the aetiological factors were said to be hostile identification and ambivalence to a father figure and an unconscious desire for self-punishment. Koh *et al* (1971) reported a 20 year-old male, whose typical symptoms improved with phenothiazine. Rhee & Kim (1973) described a 27 year-old male, in whom the aetiological problems were postulated as hostility and poor impulse control towards authority figures; he had also a typical obsessive-compulsive disorder, and haloperidol was used for the first time in Korea in this case. Kim & Lee (1973) reported a 20 year-old female, and Kang & Yoon (1974) a 14 year-old boy who has had phobias and compulsive behaviours;

psychodynamic factors were described as a tyrannical father figure and feelings of rejection.

In this report, we summarise the clinical data on 30 Koreans with the syndrome whom the authors have seen personally since 1976, along with additional data from the seven cases described above.

Method

Thirty patients were diagnosed as having Gilles de la Tourette's syndrome during the period 1976-1984 at the Department of Psychiatry, Yonsei University Medical Center, Seoul. All were evaluated psychiatrically by one of the authors, history of symptoms and family and developmental history were also obtained from semi-structured interviews with the patients and their families.

For final inclusion, all clinical data were reviewed again by both authors for the following diagnostic criteria: onset of symptoms between the ages of 2 and 15; presence of recurrent, involuntary, repetitive, rapid, purposeless motor movements, affecting multiple muscle groups; and multiple vocal tics. Ability to suppress movements voluntarily for minutes or hours, variations in the intensity of symptoms over weeks and months, and duration of more than one year, as required by DSM-III (American Psychiatric Association, 1980), were not considered necessary for the diagnosis; neither was coprolalia.

Twenty-nine patients met these criteria. In a further female patient, the syndrome began at the age of 18. Her symptoms, such as barking and multiple tics, were typical of the syndrome, had been present for about four years, and were controlled by haloperidol. She was therefore diagnosed as having Gilles de la Tourette's syndrome, and included in the study.

Eight of the cases were referred by neurologists, neurosurgeons, paediatricians and physicians, and nine by other psychiatrists in the same department.

Results

All 30 patients were native Korean citizens. The syndrome appears to be rare in Korea, considering that the total number of out-patients of this Medical Centre was about four million during the period. Twenty-four patients were males and six females. The average age was 11.8 years, and ranged from 6 to 32; 24 patients were in the range 8-16 years. One of the four adults was married.

No significant factor was reported in the birth history of any patient, but 15 had an abnormal developmental history, including hyperactivity and distractibility in nine cases, learning difficulties in four, enuresis in two, encopresis in one, and stuttering in two. Twelve patients were reported by parents to have irritable and impulsive personalities, eight to be competitive and egocentric, five to be obsessive and meticulous, and five to be shy and polite.

As far as the medical history was concerned, two patients had suffered from nephritis, two from dog bites, two from pulmonary tuberculosis, one from a car accident, and one from febrile convulsions. One patient had had a tonsillectomy. These previous problems did not seem to play a role in the initiation of the symptoms.

Ten patients were the youngest of their families, six were in between more than three siblings, four were oldest sons, and eight were only sons or daughters. Simple tics were reported to have been present in the siblings of only two patients. The presence of functional psychosis in first-degree family members was denied in all cases except one, where the grandmother died of an unspecified psychosis.

The syndrome began before the age of 15 in 29 cases and at age 18 in one female, who can be considered as an example of an adult-onset type of the syndrome. The mean age of onset was 8.7 years for males and 7.8 for females, while the commonest age of onset was between two and four years (eight patients). The mean duration of the illness was 3.8 years (range 0.1–22 years).

Various precipitating and situational factors might reflect psychodynamic influence on the development of symptoms, e.g. tonsillectomy in two cases, and beating by the father, elder brother, or schoolteacher in four cases. Eleven other cases were thought to show psychogenic factors, including emotional frustration, conflicts in child-parent relationships, and loss of self-esteem; two patients scarcely showed the symptoms at school, but frequently did at home.

Most patients reported that the symptoms, especially vocalisations, were usually aggravated by anxiety, tension, and other emotional stress. Fifteen reported anxiety, emotional uneasiness, and chest tightness when they tried to voluntarily control or inhibit symptoms—especially with coprolalia in four cases. Common cold with fever seemed to be related to recurrence in six cases.

After the initial symptoms, other parts of body became involved, and new tics developed which replaced or added to pre-existing ones; the general progression was cephalocaudal. These symptoms are shown in Table I. Obsessive-compulsive personality traits were noted in five patients, but such behaviour, e.g. touching and changing clothes, was reported in only three of these.

Only nine cases showed the classical syndrome with multiple motor tics, barking, and coprolalia; another eight had combined multiple motor tics and barking. Nine cases had multiple motor tics and other vocalisations, while four had only severe multiple motor tics. According to the criteria of severity of Shapiro *et al* (1973), 14 patients (47%) were rated as having marked and severe illness, i.e. progressive and chronic, without remission, and with serious social, academic, and occupational impairments.

No patients were evaluated as having schizophrenia or other psychoses. Ten children were reported to have

TABLE I
Initial and presenting symptoms in 30 Korean cases with Gilles de la Tourette's syndrome

Symptoms	Number of patients	
	Initial	Presenting
Eye twitchings	15	16
Tongue movements		3
Grimaces	4	9
Barking		14
Other vocalisations	4	9
Head and neck jerks	6	20
Arm jerks		12
Leg jerks		10
Trunk jerks	1	5
Coprolalia		9
Spitting		5
Echolalia		4
Compulsive behaviours		3
Stuttering		2
Bizarre mannerisms		2

marked adjustment reactions to peer relationships at school since the onset of the syndrome, e.g. one eight year-old boy refused to attend school because of teasing by peers. Two patients had suffered from nightmares. Four patients, who were older, were diagnosed as having reactive depression; one of them, a 17 year-old male, showed serious depression with suicidal ideas. The parents of six children reported that the personality of their children had changed since the onset of the syndrome to become hyperactive, violent, and impulsive.

Thirteen of 25 patients who had EEG examinations showed non-specific abnormal findings: random slow waves in three cases, random slow and sharp wave mixtures in five, mild focal abnormality in one, and a burst of high amplitude sharp waves in both hemispheres in four. Computerised tomography was carried out in six cases, and the findings were normal in all these.

Among previous treatments were anticonvulsant medication in five cases diagnosed as having a myoclonic epilepsy before referral, benzodiazepines in three, herbal medicine in five, and acupuncture in two. One case tried treatment by traditional Korean exorcism. All these treatments eventually proved to be ineffective.

After the diagnosis of Gilles de la Tourette's syndrome, all patients were treated with haloperidol. The initial dose was usually 0.5–3.0 mg/d, according to age and body weight, gradually increased by 0.5 or 1.5 mg until an effective dose was determined. This was different in each patient, and ranged from 1.0 to 12.5 mg, with a median of 2.5–3.0 mg/d; it did not seem to be significantly correlated with the body weight, age, severity of the symptoms, duration of illness, or age of onset. The period of treatment ranged from three weeks to three years.

With haloperidol, symptoms remitted completely in 21 patients and were partially alleviated in five others, after about one month; this was evaluated by clinical judgement of overall percentage decrease of frequencies of symptoms,

compared with the pre-treatment levels. The average improvement rate was 87%. Combined treatment with diazepam (3–10 mg/d) was helpful in five cases in which emotional tension was an obstacle to treatment. The side-effects of haloperidol were troublesome to many patients: diurnal drowsiness was most common, while acute dyskinesia was the major cause of discontinuing haloperidol.

Twenty-five patients discontinued medication against medical advice, after 3–12 weeks of treatment. In most cases, either the patients or their parents believed that treatment had been sufficient, but 17 patients visited the hospital again after a mean period of 2.6 months because their symptoms recurred; in four of these, the dosage needed to be increased to obtain the same effect as given by previous treatment.

Discussion

In this study, the clinical features of Gilles de la Tourette's syndrome such as age of onset, duration of illness, male predominance, non-significance of birth rank, and relatively high incidence of abnormal developmental problems revealed no differences from those in previous studies on European, American, or Asian patients (Singer, 1963; Morphew & Sim, 1969; Shapiro *et al.*, 1973; Woodrow, 1974; Fernando, 1976; Lieh-Mak, 1979; Nomura & Segawa, 1982; Chen & Lu, 1983; Lees *et al.*, 1984). The frequency of first symptoms, cephalocaudal progression of symptoms, waxing and waning clinical course, frequency of involved muscle groups, and incidence of EEG abnormality were also similar. The seven cases reported by other Korean authors had clinical features almost the same as in this report: the typical syndrome with coprolalia was found in two cases, and multiple motor tics (including vocal) in four. These findings suggest that the clinical history and general symptom pattern of Gilles de la Tourette's syndrome, although rare, are almost uniform throughout the world.

However, there were minor differences: in this study, neurotic traits were not reported as often as in Western series. In particular, obsessive-compulsive personality traits or behaviour problems were less frequent than in the cases reported by Morphew & Sim (1969), Woodrow (1974), and Lees *et al.* (1984). Other Korean authors have reported one case with a history of compulsive behaviour and two cases with obsessive-compulsive personality traits (Rhee & Kim, 1973; Kim & Lee, 1973). However, Shapiro *et al.* (1973) concluded from 34 European and Jewish patients and from the literature that obsessive-compulsive symptoms and traits are infrequent. The lesser incidence of tics or other psychiatric problems in the family histories of this series and of other Korean cases contrasts with the high incidence in Western studies, which had provided a genetic model

for the syndrome (Kidd *et al.*, 1980). There are two possible reasons for this: intentional denial by Korean parents, because of the prejudice against psychiatric illness; and failure to use a standardised rating scale to detect them.

The cause of the syndrome is still obscure. Although there is a trend to believe that it is a neurological disorder of the central nervous system (Shapiro & Shapiro, 1982), supported by the presence of non-specific abnormal EEG findings in half our cases, Korean authors have attempted a psychodynamic formulation. Hostile father figures and inhibited aggression (Kim, 1971; Rhee & Kim, 1973; Kang & Yoon, 1974) have been postulated as core problems; similar findings occurred in some cases of this series. Tics, particularly coprolalia and spitting, might indicate a disturbance in normal balance between a need for tension relief and the capacity to control such actions, suggesting that psychogenic factors may be aetiologically relevant (Ascher, 1948; Dunlap, 1960).

Haloperidol was as effective a drug for Korean patients as it is for Caucasians and other Asians, but the dosage must be individualised; the average improvement of 87% was similar to the rates reported in western countries (Bruun *et al.*, 1976; Woodrow, 1974). Three of the other seven Korean cases were also treated with haloperidol, while another two were treated with perphenazine and chlorpromazine. All these drugs were effective, and their dosage was decreased gradually. However, our study suggests that Korean patients need less haloperidol than Caucasians, who had 6–180 mg/d, frequently in addition to anti-parkinsonian drugs (Shapiro *et al.*, 1973). However, the same authors (Shapiro & Shapiro, 1981) have reported responses to remarkably low doses, e.g. 5 mg daily. Asians may have a genetically different constitutional responsiveness to drugs: Nomura & Segawa (1982) reported that haloperidol (0.75–3 mg/d) was effective for about 70% of 100 Japanese, Korean, and Philippine children with the syndrome, while Chen & Lu (1983) reported that haloperidol (0.5–3 mg/d) showed excellent to fair improvement in eight out of ten patients.

Our findings confirm that the Gilles de la Tourette's syndrome is seen in Korea, and has many features in common with those reported from Western countries. The aetiology probably represents multiple factors, which need to be understood in terms of emotional, behavioural, developmental, hereditary, and organic concepts. Evaluation and treatment should encompass a holistic approach, and include chemotherapy, academic help, counselling, and other psychosocial considerations.

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The Life Satisfaction Index - Well-being: Its Internal Reliability and Factorial Composition

The internal consistency and factorial composition of the eight-item Life Satisfaction Index - Well-being, adapted for use with elderly British samples, were examined. Cross-validation was carried out with a randomly drawn community sample of 155 rural people aged 65-89 years. The scale was found to have acceptable internal reliability, but may require modification.

Despite a lack of conceptual clarity, the construct of 'life satisfaction' has attained some popularity among gerontologists, who regard it as a useful indicator of adjustments and successful ageing. It has been observed that life satisfaction is as related to mental health (Liang, 1982). Since depression in later life is relatively difficult to diagnose (Bergmann, 1981), life satisfaction measures may provide a useful indicator of affect and subjective well-being. Gilleard *et al* (1981) reported that the Life Satisfaction Index - Well-being (LSI-W) (Bigot, 1974) was able to distinguish at both admission and discharge between clinically depressed elderly patients and normal volunteer controls.

Of the many life satisfaction scales, only the LSI-W has been standardised for use with a British population. It consists of eight items, derived from the 20-item Life Satisfaction Index - Achievement (LSI-A) (Neugarten *et al*, 1961) by item and factor analysis. There are two four-item sub-scales (Acceptance-contentment and Achievement-fulfilment), which assess respectively current contentment and satisfaction with past achievement. The sub-scales sum to provide an overall measure of well-being.

This study reports data on the internal consistency and factorial composition of items composing the LSI-W. These data are needed for several reasons: Bigot's scale was developed on a male non-random sample, not all of whom were 'elderly' as usually defined (they ranged in age from 55-79); and no cross-validations appear to have been carried out. Since our experience is that the LSI-W is quick and easy to administer, and acceptable to elderly sub-

jects, it could be a useful empirical tool. Data on its internal consistency would support this claim.

Method

The LSI-W was administered to 155 elderly people living in a rural community, as part of a study of life stress and depression; the response rate was 85% of a randomly drawn sample (Wilkinson *et al*, 1985). The sample ranged in age from 65 to 89, with a median age of 73; 85 respondents (55%) of the sample were female. Thirty-nine percent were of social class I and II (Registrar-General's classification, Office of Population Censuses and Surveys, 1985); 55% were married, 36% widowed, and 8% were single. Just over one-third lived alone.

The LSI-W was scored on a three-point Likert scale, with subjects responding 'agree', 'disagree' or 'uncertain' to each of the eight items (Table I). For items 1, 3, 4, and 5, agreement was scored 2 and disagreement 0; for items 2, 6, 7, and 8, disagreement was scored 2 and agreement 1. A response of uncertain to any item was scored 1. The maximum attainable score is 16. Analysis was performed using two SPSS-X procedures: 'Reliability' and 'Factor'.

Results

The mean LSI-W score for the sample was 10.99 (s.d. 3.74). The inter-item correlations ranged from 0.03 to 0.43, with a mean of 0.19. Coefficient alpha (Cronbach, 1951) was 0.66, lower than the value of 0.80 reported by Bigot (which tended to suggest a level of item redundancy). The item-total correlations were all above 0.20, with the exception of that for item 7, which was 0.14; its exclusion from the scale increases coefficient alpha from 0.66 to 0.69. Item 6 also performed poorly, correlating significantly with items 1, 2, and 9 only.

TABLE I
Principal components matrix for LSI-W with varimax rotation

Item no.	Scale ¹	Item	Factor 1	Factor 2	Communality (η^2)
1.	AC	I am as happy as when I was younger	0.816	-0.011	0.666
2.	AC	My life could be happier than it is now	0.687	0.138	0.491
3.	AC	These are the best years of my life	0.604	-0.079	0.371
4.	AC	The things I do are as interesting to me as they ever were	0.578	0.238	0.391
5.	AF	I would not change my past life if I could	0.176	0.648	0.451
6.	AF	Compared with other people of my age, I've made a lot of foolish decisions in my life	0.332	0.186	0.145
7.	AF	When I think back on my life, I didn't get most of the important things I wanted	-0.097	0.739	0.557
8.	AF	Compared with other people, I get down in the dumps too often	0.523	0.526	0.549
Eigenvalue			2.524	1.096	3.62
Variance: %			31.55	13.70	45.25

1. AC = Acceptance-contentment, AF = Achievement-fulfilment

Bigot reported a coefficient alpha of 0.7 for the Acceptance-contentment sub-scale and 0.60 for the Achievement-fulfilment sub-scale. In this study, however, the respective values were 0.65 and 0.41. Given the small number of items, the acceptance-contentment sub-scale looks impressive, with the item-total correlations ranging from 0.35 to 0.57 and a mean inter-item correlation of 0.32. In contrast, the mean inter-item correlations for the Achievement-fulfilment sub-scale was 0.16, and two of the item-total correlations (items 6 and 7) were below 0.2. Only one of the six correlations between items 5 to 8 was above 0.2, although four of the six were significant at the 5% level.

A principal components analysis of the LSI-W with a varimax rotation (Kaiser, 1958) gives rise to two factors (using the Kaiser-Guttman criterion whereby only factors having eigenvalues greater than unity are retained). Child (1970) has noted that when the number of variables is below 20, there is a tendency for this method to extract a conservative number of factors. The factors, their loadings, and communalities are shown in Table I.

Factor 1 accounts for 31.6% of the variance, while factor 2 accounts for 13.7%. The low communality for item 6 suggests that the item is unreliable; otherwise, the rotated factor matrix confirms Bigot's sub-scales. Factor 1 loads substantially on the Acceptance-contentment sub-scale, while factor 2 loads specifically on Achievement-fulfilment.

Discussion

Psychometric theory affirms that reliability is a necessary but not sufficient condition for validity. In this study, we have examined the internal consistency

of the LSI-W because in both research and applied settings it appears to be a useful indicator of affect and subjective well-being among the elderly.

According to Nunally (1967), the adequacy of a reliability coefficient depends on the purpose for which the measure is needed. For most purposes, the LSI-W reliability coefficient of 0.66 is perfectly adequate, particularly if this measure is used as part of a battery of questionnaires and tests as suggested by Bigot. Of some concern, however, is the poor performance of item 6, which is not explained by either factor, and we recommend that this item be omitted from the LSI-W. In their analysis of the LSI-A, Wood *et al* (1966) and Adams (1969) recommended that the same item be excluded from that scale. Item 7 also performed poorly in the item reliability checks, and could also be considered for rejection. Chown (1983) stated that "a person's evaluation of his or her life is likely to be influenced by past and present objective situations and by his or her own assessment of them". The LSI-W would appear to be a more adequate measure of current satisfaction than past achievement, and should be scored and interpreted as a single scale, rather than as two sub-scales.

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Severe Lactic Acidosis Following Paraldehyde Administration

A case of severe lactic acidosis following paraldehyde administration is described, and the adverse effects of paraldehyde reviewed.

Paraldehyde was introduced into medicine over 100 years ago by Cervello (1884) and Strahan (1885). It has been used in a variety of clinical situations, including the emergency treatment of convulsions associated with tetanus, eclampsia, and status epilepticus, basal narcosis, and obstetric anaesthesia. Its current use is limited to psychiatric units, particularly those without resident medical cover. Despite its widespread use during the past century, little is known about its pharmacology, metabolic fate, or toxicity. Although it has generally been regarded as a particularly safe agent, it has a very low therapeutic index, and large doses produce respiratory depression and hypotension (Goodman & Gilman, 1985). Drug-induced metabolic acidosis has been reported following prolonged use and after overdose (Beier *et al*, 1963). The following case report shows that severe acidosis can also occur after relatively small doses.

Case Report

A 30 year-old male patient was transferred to the psychiatric unit's medical ward in order to review his anticonvulsant therapy. Control of his fits had been difficult in recent months, with one or two *grand mal* seizures occurring every week. Physical examination and initial biochemistry were normal (Table I).

During the following day, the patient had three *grand mal* seizures within 12 hours. Fresh paraldehyde (a total of 40 ml) was administered intramuscularly during this time, since there was neither resident medical cover nor intravenous access to the patient. Six hours after the last fit, the patient was found to be hypotensive (blood pressure

80/40 mmHg) and tachypnoeic (respiratory rate 30 breaths/min). He was drowsy, but rousable. Biochemistry at the time (Table I) showed a severe metabolic acidosis, with an anion gap of +38 (normal range 10–14) and serum lactate of 27 mmol/l. The patient was resuscitated with normal saline and sodium bicarbonate solution, administered intravenously. No more paraldehyde was given. Diazepam was given intravenously to control two further seizures the following day, at which time his biochemistry had returned to its previous values (Table I).

Discussion

There are two main routes for the excretion of paraldehyde: 11–28% is exhaled, and 70–80% metabolised

TABLE I
Serial electrolyte results

	Day 1: mmol/l	Day 2: mmol/l	Day 3: mmol/l
Na ⁺	143	147	140
K ⁺	3.8	3.5	3.8
Bicarbonate	29	9	28
Cl ⁻	106	100	104
Urea	5.8	6.4	6.8
Creatinine	77	110	75
Anion gap	8	38	8
Serum lactate	—	27	1.3

in the liver. It has been suggested that the initial stage is depolymerisation to acetaldehyde, followed by oxidation of acetic acid (Hitchcock & Nelson, 1943), and then via the Krebs cycle to carbon dioxide and water. The acidosis produced by paraldehyde is in excess of that consequent to the oxidation of paraldehyde-derived acetaldehyde.

This may be due to enzyme inhibition during intermediate metabolism (Beier *et al*, 1963). Alternatively, a block in the Krebs cycle could produce a shift in the lactate/pyruvate system in favour of lactate, which could account for the high levels recorded in this case. *Grand mal* seizures themselves may cause a rise in lactate levels, but expected levels would be equivalent to those found in strenuous exercise (5 mmol/l) (Linter & Ryan, 1985). Furthermore, the serum lactate level dropped after the discontinuation of paraldehyde, although there were further *grand mal* seizures.

Previous reports of paraldehyde-induced metabolic acidosis have not provided information about serum lactate levels. In addition to its undesirable metabolic effects, paraldehyde has other drawbacks: it is difficult to store safely and unpleasant to administer (both for nurse and patient).

On exposure to light and air, paraldehyde decomposes to acetaldehyde, which is then oxidised to acetic acid. Several severe poisonings and fatalities

have been reported following the administration of partially decomposed paraldehyde (Agranat & Trubshaw, 1955). Because of this, paraldehyde must be stored in small amber airtight containers, and partially used bottles must be discarded. One study showed that a significant proportion of ward stocks may have decomposed during storage (Goodman & Gilman, 1985).

The administration of paraldehyde is unpleasant; it has a disagreeable smell, and when it is used parenterally a glass syringe is essential, since paraldehyde reacts with some plastics. Deep intramuscular injection to both buttocks is the usual route of administration, and severe, permanent sciatic nerve damage has occasionally followed; more frequently, sterile abscesses and skin sloughing occur at the injection sites. Intravenous administration is particularly hazardous, and may result in hypotension, pulmonary oedema, and venous thrombosis (Goodman & Gilman, 1985).

Thus, paraldehyde in therapeutic doses can cause a marked metabolic acidosis, sterile abscesses, nerve damage, and skin sloughing. The administration of partially decomposed paraldehyde has led to fatalities. We suggest that paraldehyde is an outmoded drug that has no place in today's medicine. Modern benzodiazepines offer a safer, more effective alternative.

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Munchausen's Syndrome and Drug Dependence

A man with Munchausen's syndrome was treated for drug dependence, which allowed him to establish and sustain a bond with a hospital, but eventually brought about an ethical dilemma.

Munchausen's syndrome is a rare condition, characterised by patients who repeatedly seek admission to hospital in a state of mental or physical distress, give plausible histories to support their complaints, and submit themselves to painful or even dangerous investigations and therapies, but then abruptly discharge themselves when discovered to have fabricated their stories and physical signs (O'Shea *et al.*, 1984). An association between this syndrome and drug abuse has often been noted (Blackwell, 1968), and Asher (1951) listed the desire to obtain drugs as one of the possible motives underlying the syndrome, although published case reports of patients where a diagnosis of both Munchausen's and drug dependence syndromes have been made are comparatively rare (Hammond, 1980; Mendel, 1974; Bursten, 1965). In reviewing 19 reported cases, Mendel (1974) found that six had a history of drug addiction, but the inclusion of those who had received or demanded large quantities of addictive drugs during hospital stay brought the number to 14. He concluded that these patients should be considered to be suffering primarily from drug abuse, if not addiction, and that treatment should emphasise the drug-dependent nature of the syndrome. Proper labelling would improve the therapeutic relationship.

The generally accepted view is that the craving for drugs reflects a craving for attention and hospital contact (O'Shea *et al.*, 1984; Scott, 1982; Enoch & Trethowan, 1979). Patients may use the diagnosis to gain entry into hospital (Cheng & Hummel, 1978; Hammond, 1980) or as a second line of defence when their fabrications are revealed. Barker (1962) pointed out that these patients do not usually experience withdrawal symptoms when admitted to hospital, although this in itself does not exclude the diagnosis of drug dependence; they may also abscond, despite the offer of maintenance opiate therapy.

We report a case of both Munchausen's and drug dependence syndromes. Although the patient is an unreliable informant, details of his early life, given to different doctors over the years, have been broadly consistent.

Case report

JS is a 42 year-old Scotsman who was born into a large working-class family, the ninth among 11 siblings. The youngest child died of a brain tumour, his father underwent operation for a chronic peptic ulcer on more than one occasion, and his mother had a history of diabetes and cataracts, and died following a cerebrovascular accident.

As a child, JS suffered from chronic otitis media, and required operation for appendicitis at the age of seven. During his late adolescence, he was admitted to hospital for gastroenteritis. He entered the Royal Army Medical Corps at the age of 19, after an unfinished apprenticeship; this introduced him to a medical milieu, where he served as an operating theatre technician, assisting the anaesthetist with intravenous procedures, and handling pain-relieving drugs. At the age of 21, he underwent a medial and lateral meniscectomy on his left knee, following a football injury, and was invalided out of the Army, he has since been largely unemployed.

Shortly after, he began to display the pattern of behaviour typical of Munchausen's syndrome. He would arrive in a casualty department in the early hours of the morning as an acute emergency, and be admitted to a medical or surgical ward. The presenting picture varied over the years: it included acute intestinal obstruction, mastoiditis, renal colic, and less frequently meningitis, cerebral abscess, prolapsed intervertebral disc, and mechanical disorders of the knee. To support the diagnosis, he would sometimes rely on past injuries (e.g. abdominal scars, chronic ear discharge), but would often falsify investigations (e.g. by contaminating urine samples with blood from a pinprick in the thigh). As a result, he underwent at least three exploratory laparotomies, a right and then a left radical mastoidectomy, and also left temporal burr holes following a suspicious brain scan. It is not clear whether he was actively seeking opiates in the early stages, but certainly from the age of 24 onwards, hospital case notes record such suspicions with increasing frequency. He would never remain in any one locality for more than a few months; by then the hospital would have liaised with his previous contacts, and be resistant to further involvement. Indicative of the extent of his travels is a 12-month period between 1968 and 1969, when admissions were recorded in Aberdeen, Blackpool, Manchester, London, and Jersey. He even crossed international boundaries: we have traced records of his contacts with hospitals in Switzerland (in 1977) and France.

His first contact with the psychiatric service was at the age of 16, following an overdose. He was at times referred for psychiatric assessment, but was uncooperative, and defaulted from any treatment offered. At the age of 28 (the year of his mother's death), he presented with quadriplegia; this was soon found to have no organic basis, and so he was referred for psychiatric treatment. Attempts were made to help him to express his difficulties and conflicts in psychological terms, rather than as physical symptoms, but he responded with aggressive behaviour and by absconding; in fact, he gained admission to another hospital with abdominal pain. On his return he took an overdose of aspirin, and later swallowed glass fragments of a light bulb, but without ill-effects. In the same year, the Home Office was first notified of his suspected drugs dependence.

At the age of 31, he accepted treatment at a drug dependence unit for the first time. The nature of his dependence was atypical, in that the drug he most desired was the relatively milder opiate pethidine, and the preferred route of administration intramuscular. He had no contact with illicit sources of supply, and generally received his opiates from

hospitals. It is also noteworthy that there is no record of physical withdrawal symptoms while in hospital under observation, as well as a suspicion that he may have sold some of his prescribed drugs. The only substance he abused was alcohol, and he has convictions for drunk and disorderly behaviour and for assault.

Over recent years, he had become more overtly drug-seeking, demanding greater doses of opiates; any attempt to limit the quantity or manner of prescription resulted in increasingly self-destructive acts. These usually involved cutting and piercing instruments, although he also overdosed, and once set fire to his clothing. He has cut his wrists to the extent of exposing fascia and nerves, and then refused treatment until he had received intramuscular pethidine. Since the age of 32, he had swallowed such objects as razor blades, stitch cutters, and needles on at least ten occasions; this resulted in many operations. Following the first laparotomy and gastrotomy, it was decided to withhold pethidine, but within days, he again swallowed razor blades. In 1985, when a casualty officer refused to supply him with opiates, he stabbed himself in the abdomen, penetrating the small bowel.

These repeated insults resulted in a number of complications. Five years ago, he developed a faecal fistula between the transverse colon and the anterior abdominal wall, requiring a colostomy and closure, which later led to a chronic ulcer and incisional hernia. He had also been treated for partial bowel obstruction due to multiple adhesions, although his recovery was at times suspiciously rapid. His drug dependence led to serious complications: repeated intramuscular injections produced infection, inflammation, and ultimately fibrosis of the muscles in the thigh and buttocks. The resultant limitation of movement means that his knees are fixed and immobile, and when he is seated, his legs stretch out parallel to the floor. In order to walk, the relative immobility of his hips force him to propel himself by swivelling his pelvis in a strutting, waddling gait. He has had numerous hospital admissions for abscesses in the groin and buttocks, and the frequency with which they recur indicate that he may deliberately provoke the sepsis, or at least prevent it healing. Over the past five years he has had 57 separate admissions, of which 12 were to psychiatric wards.

The two drug dependence clinics that have been involved in his care have tried various methods of containing his behaviour, including in-patient detoxification, compulsory detention orders, and daily attendance to collect his prescribed opiates. These failed, as did attempts to convert him from pethidine to the less easily obtainable methadone hydrochloride, and from intramuscular to oral forms of medication. His response to pressure has been to utilise his many physical disabilities to gain admission to other hospitals, and thereby the desired drugs. Failing this, he has restored to acts of self-mutilation.

Throughout, he has remained a loner, and the only relationships he has been able to sustain have been with hospital staff. When frustrated, he quickly becomes querulous, vocal, and threatening, and he can be manipulative with scant regard for the truth. He has served two brief prison sentences for assault and theft, and was recently fined for two shoplifting offences.

Discussion

Many explanations and theories have been advanced regarding Munchausen's syndrome. Enoch & Trethowan (1979) remarked that "in nearly all cases the early environment of these patients is unfavourable, leading in some or other way to their become conditioned, in a sense, to use symptoms as a means for seeking attention". A large family such as this patient possessed may have lacked the resources to satisfy the needs of all its members, and illness behaviour (Mechanic & Volkart, 1961) may then have become a powerful mechanism to evoke and receive the necessary care and attention. Role models were provided by family members, his own childhood illnesses, and his subsequent exposure as a paramedic. Reed (1978) noted the frequency with which symptoms develop soon after an illness which necessitated admission to hospital: in a patient with a personality disorder, admission at a time of social stress may lead to the discovery that hospital care brings short-term relief, whereas discharge leads to re-exposure to the stress of normal social interaction. JS had centred his life on institutions, passing from his family to the Army and then to hospitals, without developing any significant social contacts outside these structures. His encounters with the hospital service had usually been brief, initially evoking interest and concern, and leading to a multitude of investigations and treatments. This initial zeal on the part of hospital staff was replaced by a sense of defeat, with resultant feelings of frustration, anger, and eventual rejection, forcing him to search out new doctors in fresh localities.

By adopting the role of drug dependence, this patient succeeded in establishing a bond with a hospital clinic and sustaining it over a period of years, despite frequent interruptions. Recasting the problem in the mould of a drug dependence syndrome satisfied both this patient's need and the requirement of the health service for an acceptable mode of interaction. But this has, with the passage of time, brought about an ethical dilemma: the continued prescribing of intramuscular pethidine will lead inexorably to greater disability, yet the patient steadfastly refuses to allow any interference or alteration of his drugs. Furthermore, his self-destructiveness has shifted increasingly from a passive to an active form. Rather than a means to communication, drugs have now become an end in themselves, a tangible symbol of the attention he so craves. For this particular patient, the outcome has not met the optimistic expectations of those who have advocated an emphasis on drug dependence in the management of Munchausen's syndrome (Mendel, 1974).

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Treatment of Depression in Huntington's Disease with Monoamine Oxidase Inhibitors

Depression is common in Huntington's disease. It is customary to treat such patients with tricyclic antidepressants, although there is disagreement about their effectiveness. The use of monomine oxidase inhibitors for depression in HD has not previously been reported; three cases are described here. MAOIs appear to be of value in the management of depression in HD.

Psychiatric syndromes are among the defining features of Huntington's disease (HD). Huntington (1872) included in his description a "tendency to that insanity that leads to suicide." However, the nature of the psychiatric syndromes is disputed. Koehler & Sass (1984), reviewing mainly German publications, pointed to the preference for diagnoses of schizophrenia-like states or personality alterations. On the other hand, Bolt (1970) and others have pointed to the prominence of affective symptoms such as reactive mood disorders and apathy, while Folstein *et al* (1979) claim that severe depressive disorder is seen in up to 40% of cases.

Treatment methods include the pharmacological, psychological, and social. It has been suggested that HD patients with depression are helped by tricyclic

antidepressants (TCA) (Whittier *et al*, 1962; McHugh & Folstein, 1975; Folstein *et al*, 1979). and Folstein & Folstein (1981) reported that patients with very severe depression, who did not respond to amitriptyline, had been successfully treated with electroconvulsive therapy. Reports on the use of antidepressants, however, are not uniformly enthusiastic. Caine & Shoulson (1983) evaluated 30 patients with HD, of whom 24 had a definable psychiatric disorder; they noted that pharmacotherapy was modestly beneficial in some cases. Six patients received unspecified antidepressants; five of these experienced an improvement in the cardinal somatic features of their disorder, but the drugs had no effect on those ideational components typically associated with a depressed state. Shoulson (1981) evaluated 22

patients with HD over 13–14 months, and reported that depression lessened slightly in seven of ten patients treated with TCA, but that combined depression scores declined insignificantly; pharmacotherapy often caused adverse effects, and functional capacity declined in all patients even where depression seemed improved.

Monoamine oxidase inhibitors (MAOIs) were not discussed in these accounts, but many controlled trials have shown that they do have a place in the treatment of a variety of ill-defined depressive and anxiety states. I report here the use of MAOIs in the treatment of depressive disorder in three patients with HD; all had generalised chorea and a parent with a diagnosis of HD.

Case Reports

A, aged 23, was admitted because of tearfulness, refusal to eat, anxiety, early morning waking, diurnal variation of mood, and suicidal ideation. She had distressing delusions that everything and everyone resembled one of the nursing staff from a previous admission. She had previously failed to respond to clomipramine (150 mg daily), and was therefore started on phenelzine (15 mg t.d.s.). Her symptoms suddenly abated nine days later; she became cheerful and without thoughts of self-harm, while both her family and the staff considered her choreic movements to have improved. After a year's remission, she was readmitted with recurrent symptoms, shortly after reducing her phenelzine from 15 mg t.d.s. to 15 mg b.d. The larger dose was restored, and she again became much happier. She remained so, despite her chorea and moderate cognitive impairment, for a further year, after which therapeutic measures became unsuccessful.

B, aged 47, required five admissions for psychotic depression over four years. At the time of her fifth admission she was mute, sleeping poorly, agitated, and tearful. She had been taking clomipramine (150 mg nocte), which was discontinued and replaced two weeks later by phenelzine (15 mg t.d.s.). Her home facilities were reviewed, and better accommodation sought. For the following two years she remained well and cheerful on phenelzine, and did not require admission.

C, aged 69, was reported by a nearby hospital. She had been depressed for several years. After admission, isocarboxazid (10 mg b.d.) was administered, but produced debatable improvement in mood, and was therefore discontinued. The patient became clearly more depressed, with headaches and pains in hands and legs; the pains in her hands were such that she was unable to wash her clothes. Treatment with isocarboxazid (10 mg b.d.) was reinstituted. After one week she became happier, the pains faded, she was able to do her washing, and her choreic movements lessened.

Discussion

Patients A and B suffered from recurrent depression, but had not responded to a TCA. In this situation, "the psychiatrist is faced with the choice of electroconvulsive therapy or trying one of the newer drugs or an MAOI" (Shaw, 1977). Phenelzine was chosen with some hesitation, as non-endogenicity is the salient feature in predicting response to MAOIs (Brandon, 1982), and our patients would not clearly be so classified.

The sudden clinical response of A, nine days after the administration of phenelzine, and C, seven days after the reintroduction of isocarboxazid, supports the contention that this was due to the MAOI: Tyrer (1982) reported that a sudden and dramatic change in mood and symptoms takes place. Later relapse took place in A when she reduced the dosage of phenelzine, and her mood improved when it was reinstated in full dosage. B's response was probably due to a combination of favourable pharmacological and social measures. Additionally, the three patients' response to drugs was consistent with the observation of Caine *et al* (1978) that HD patients were exquisitely sensitive to rapid changes in administered psychoactive medications.

There is no reason to postulate that MAOIs may reduce chorea via a specific effect on the known biochemical defects in the basal ganglia of HD patients: i.e. a profound reduction in glutamic acid decarboxylase, the enzyme synthesising γ -aminobutyric acid (GABA); GABA depletion; and depletion of choline acetyltransferase, the enzyme synthesising acetylcholine. On the contrary, MAOIs increase cerebral dopamine, and as such could be expected to exacerbate the chorea of HD, which appears to result from relative overactivity of dopamine mechanisms in the brain (Marsden, 1983). Furthermore, the most effective drugs in reducing choreiform movements are catecholamine depleting agents such as reserpine and tetrabenazine, and dopamine receptor antagonists such as the phenothiazines and butyrophenones (Bird & Iversen, 1974). However, in two of our cases, it was interesting that improvement in chorea followed the use of an MAOI, and was presumably a consequence of mental well-being. The confidence-enhancing and energising effect of monoamine oxidase inhibitors may be pertinent, particularly in HD, where depression and demoralisation are frequently noted.

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Autocastration and Biblical Delusions in Schizophrenia

A young male castrated himself in response to the biblical passage, Matthew 19:12. Although extremely rare, autocastration usually occurs in men with chronic paranoid schizophrenia, with a long history of delusions of sexual guilt which can only be relieved by castration. Management of these patients is long-term, involving both pharmacotherapy and individual and family therapy.

The act of deliberate self-mutilation of the genitals in men is a rare phenomenon; Greilshiemer & Groves (1979) reported only the fifty-third such case in English. In their review of literature, Greilshiemer & Groves were able to identify three general groupings of genital self-mutilators:

- (a) psychotic men, including younger acutely psychotic men with sexual fears, older men with psychotic depression and serious somatic illnesses, and men prone to violence during pathological intoxication with alcohol
- (b) non-psychotic men; either those with character disorders who act out of rageful feelings against themselves or close objects, or transsexuals who premeditate their own transsexualising surgery
- (c) those influenced by sociocultural factors, including complex religious beliefs regarding their sexual group, and difficulty in the male sex role.

A case of genital mutilation that falls into Greilshiemer's first category of younger acutely

psychotic men with sexual fears is reported here. It is distinguished by the fact that the patient is one of a small group of remarkably similar schizophrenics who have committed autocastration due to religious delusions, based on a literal interpretation of the Bible's comment on celibacy (Matthew 19:12): "There are eunuchs born that way from their mother's womb, there are eunuchs made so by men and there are eunuchs who have made themselves that way for the sake of the Kingdom of Heaven."

Case Report

The patient was born in a Queensland provincial town, of elderly parents. He was one of non-identical twins, and the birth was difficult; the patient's mother described him as a "blue baby". He reached his developmental milestones at the usual ages. His father was a retired grazier, who taught his sons to castrate bulls – the same technique the patient used on himself. His twin brother was described as "totally different", and did not have a psychiatric history.

As a child, he was very quiet and close to his family; he had few friends. His mother described him as her "pet". His early school performance was fair, but he struggled in his senior years, failing Year 12. He worked for a year in the Public Service, but left this job, claiming all the men were homosexuals and smoked too much. He denied any homosexual or heterosexual experience: he had never had a girlfriend, and his only sexual experience was masturbation – the source of his guilt and anxiety. There had been no religious upbringing by the patient's parents, but at the age of 17, he had become greatly influenced by an eccentric elderly aunt, who was described by the patient's parents as a religious fanatic. She introduced him to the Bible, and he was baptised. His interest in the Bible increased, until every spare moment was spent sitting in his room reading the Bible from cover to cover. At the time of admission, the patient held a low-key job as a storeman; he lived at home with his parents. He did not drink, smoke, or take illicit drugs, and his only interests were the Bible and attending church.

In 1985, at the age of 31, he incised his scrotum with a razor blade, severed his testicles, and flushed them down the toilet. He was brought to hospital in hypovolaemic shock, with a haemoglobin level of 7.9 g/dl (normal range 13.0–17.0 g/dl); he was resuscitated and the bleeding points ligated. When surgically stable, he was transferred to the psychiatric unit.

The patient stated that he had castrated himself in response to Matthew 19:12; he felt evil, as he had been masturbating, and castration was the only way to gain forgiveness. He had considered cutting off his right hand, in response to Matthew 18:8, but considered that 19:12 was a specific reference to himself. He admitted to occasionally hearing non-hostile voices describing his actions, but these were not prominent in his illness.

The patient had considered castration for at least five years prior to actually committing the act; at times he had consulted surgeons, requesting this, and had been referred for a psychiatrist's opinion. In 1980, a self-inflicted

laceration to his penis required suturing, and he was subsequently admitted to another psychiatric hospital. A diagnosis of schizophrenia was made, and he was given fluphenazine decanoate injections (12.5 mg monthly). He received out-patient treatment over the next five years. Eight weeks prior to the present admission, he had appeared agitated, and refused his medication.

The patient was in good physical condition, and had no significant medical or surgical problems. There was no family history of psychiatric illness.

When he had been fully resuscitated, mental state examination revealed a vague, perplexed, man; speech was soft and quiet, and he presented a detached, flattened affect. His thought content conveyed his great sense of relief at having finally castrated himself, and at no stage did he request analgesia. His thought form was disordered, with loosening of associations and a marked inter-penetration of bizarre religious themes; there was no evidence of any underlying organic disorder, and all investigations, including EEG and computerised tomography, were normal.

The patient was started on major tranquillisers, with a corresponding rapid settling of his acute symptoms. He became more sociable and outgoing, but continued to be preoccupied with the Bible, and showed no regret for castrating himself. He refused to take replacement testosterone, for fear of getting renewed sexual desires. Because of these persistent religious delusions, simple religious arguments were incorporated into a supportive psychotherapy programme (Kushner, 1967), when his feelings of self-harm and sexual guilt were discussed.

The patient's parents were also interviewed, and found to be bewildered at their son's plight. After six weeks, the patient was discharged home on fluphenazine decanoate (50 mg i.m. fortnightly), and has remained well at subsequent follow-up.

Discussion

A review of the English-language literature on male genital self-mutilation confirms Greilshheimer & Groves' (1979) finding that such cases are extremely uncommon, and reports of cases of autocastration similar to that reported here are especially rare: only two were found.

Blacker & Wong (1963) reported four cases of genital self-mutilation, and from these identified five common elements: a lack of variety of human experience during the patient's formative years – particularly the absence of a competent male to identify with; overcontrolling mothers, who insisted on submissive masochistic responses from their sons; strong pathological feminine identifications, usually denied but apparent during intoxication, dreaming, and psychoses; physical repudiation, first symbolic and then actual, of their genitalia; and guilt, shame, and anxiety, often relieved by the self-mutilating act.

Unfortunately, Blacker & Wong do not offer a diagnosis in any of these cases, and only one case was

of an actual autocastration. Also, the histories of alcoholism, antisocial behaviour, and the absence of religious delusions are significantly different from the case reported here.

More marked similarities are found in the two cases reported by Kushner (1967). A 37 year-old divorced man with a five-year history of schizophrenia and extreme religiosity castrated himself as a means of seeking "purification of the spirit" for sexual guilt. Five years later he amputated his penis, this time citing as his reason Matthew 19:12. The second case was of a 42 year-old man with schizophrenia and bizarre religious delusions, who castrated himself in search of purification, again citing Matthew 19:12. Kushner comments on the extreme preoccupation with the Bible and the development of religious delusions in these two schizophrenic patients; as with the present case, he reported that they were ridden with guilt, especially regarding their sexuality.

Sweeny & Zamecnik (1981) noted that previous self-mutilation was a significant predictor of more such action in schizophrenia: such is the case in these autocastrators. They also report that a change in body appearance often precedes a self-mutilating act in schizophrenic patients: this was seen in one case described by Kushner, where the patient shaved his head prior to autocastration. Unfortunately, such predictors are only too easy to identify with hindsight.

In the management of self-induced genital injuries, Mendez *et al* (1972), as well as others writing in urological journals, stress the importance of preservation or replacement of testicular androgen activity. However, this was not possible in the case reported here, as the patient became quite anxious at the prospect of replacement testosterone rekindling his sexual desires and more guilt feelings. This is not surprising, considering the patient's delusions

and the way in which he disposed of his testicles originally. Greilsheimer & Groves (1979) noted that "those individuals adamantly opposed to surgical repair of the diserved genitals will almost always dispose of the organs at the time of the act; individuals ambivalent about the act may bring their organs with them to hospital." This latter group should be considered for surgical repair.

Management of this patient incorporated a three-phase approach. Firstly, treatment with major tranquillisers produced a lifting of the more acute psychotic features. Secondly, low-key psychotherapy used the patient's religiosity to discuss and, it was hoped, prevent further self-mutilating attempts. Finally, the family were involved to overcome their horror and disgust, and to encourage them to support their son. Further out-patient follow-up will be needed at frequent intervals for many years, due to the high risk of more self-mutilation.

Thus, a small group of schizophrenics who castrate themselves in response to a Biblical passage can be identified. Typically, these men are chronic paranoid schizophrenics whose psychosis attracted medical attention many years prior to autocastration. Following years of obsessive studying of the Bible, they develop a complex delusional system in which they feel the need to castrate themselves in accordance with Matthew 19:12, to atone for perceived sexual guilt. The actual autocastration is not impulsive, but follows careful deliberation by the patient, who has usually presented surgically with a request for castration before taking matters into his own hands. The need to recognise the serious implications of such requests is clear.

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A Case of Anorexia Nervosa with Klinefelter's Syndrome

This is the first reported case of anorexia nervosa in association with Klinefelter's syndrome. Although the patient was initially diagnosed as suffering from 'atypical' anorexia nervosa, Klinefelter's syndrome was cytogenetically proven and other organic pathology ruled out.

Case study

The patient, a 20 year-old single male, was referred to the Eating Disorders Unit in March 1985, by the Adult Endocrine Unit of the Johannesburg Hospital. His main complaints were loss of weight over two years, from 50 kg to 39.9 kg, and vomiting five times per week for the previous two months. He denied any fear of becoming obese, excessive exercising, or laxative abuse, but admitted to loss of appetite, drinking excessive amounts of liquids, and persistently feeling cold. He was found to be fatuous, a slow and fussy eater, and untruthful about his food intake.

He had previously been admitted to the Unit in 1978, when he was diagnosed as suffering from typical anorexia nervosa (AN); he was then placed on the Unit's treatment programme, which included individual psychotherapy and a nursing care refeeding plan. He responded slowly, but did not reach the ideal weight for his height and age; after five months he was discharged, mainly because he was uncooperative, and was referred because of delayed puberty to the Paediatric Endocrine Unit. Over the next six years, his weight increased from 31.5 kg to 50 kg, and his height from 154.1 cm to 163.4 cm, so he was still underweight.

At admission, physical examination revealed a cachectic patient with a eunuchoidal body, long arms, and excessively long legs. Axillary hair was absent, and he displayed a female distribution of bodily hair, with sparse pubic hair. His penis size was normal, but his testes very small—5 ml on the left side and 3 ml on the right (normal values 12-15 ml).

He was conceived when his mother was 37 years-old, and was the last-born of three children; the pregnancy was uneventful, but the labour was prolonged, and delivery was assisted with forceps. No psychiatric problems were reported in the family. The patient's father is a successful salesman, happily married to an occupational therapist. His brother is a business executive, and his sister is married and works as a magazine layout designer. All the family members are concerned about the patient—especially his mother, who has gone to great lengths to induce him to eat normally.

It was decided to regard the patient as an 'atypical' anorexic, and to investigate him thoroughly; this was precipitated by his continual loss of weight and vomiting, despite being on the behavioural programme. All routine blood, urine, and stool analyses were normal. Hormone levels, including investigation for Addison's disease, were within acceptable limits. A full gastrointestinal workup revealed no abnormality. Chest X-ray, EEG, X-ray skull and pituitary fossa, as well as three computerised tomography scans and visual field tests, were within normal limits. The only abnormality found was a bone age consistently five years behind his chronological age; as a

result, he was placed on testosterone and chorionic gonadotrophin, both of which were stopped on his present admission. Chromosomal analysis had not previously been carried out, but cytogenetic studies revealed a 47XXY karyotype in all 14 cells examined. The diagnosis of Klinefelter's syndrome was consistent with the physical signs, as well as with the mental status finding of fatuousness.

As a result of these investigations, the patient was regarded as a case of AN whose behaviour was affected by the associated Klinefelter's syndrome. He returned to the behaviour programme, and chlorpromazine was introduced. The vomiting stopped, and he began to gain weight. Unfortunately, the patient then again became more and more difficult and uncooperative. Three months later, he discharged himself from the Unit.

Discussion

Klinefelter's syndrome is a form of male hypogonadism, resulting from non-dysjunction of one of the parental gametes at the time of meiosis; advanced maternal age is known to increase risk of non-dysjunction. The only constant physical feature is small testicular size, but numerous other signs may be present, including congenital defects, neoplasias, endocrine abnormalities, and psychiatric disorders. The patient is usually infertile, fails to develop normal male secondary sex characteristics, and has retarded pubertal development. As the role of puberty may be an important aetiological factor in the development of AN, this feature of Klinefelter's syndrome is quite significant. It has also been highlighted by the tenuous relationship between Turner's syndrome and AN, of which there are 12 reported cases. It is suggested that these individuals have problems with puberty, autonomy, body changes, or body image (Garfinkel & Garner, 1982).

Our patient was psychosexually immature, but had heterosexual fantasies, and did not appear resistant to the development of male secondary sexual characteristics (as seen by his submission to hormone therapy). It has been suggested that significant homosexual conflicts precede AN in up to 50% of male patients, but no evidence of this was found in our patient. His parents are older and from a higher social class than average, but there is no noteworthy family psychopathology (Garfinkel & Garner, 1982).

The patient's anxiety and preoccupation about meals, together with his dishonesty, excessive time spent over every meal, and vomiting indicate a definite weight phobia.

It has been suggested that more serious pathologies exist among male anorexics, since the sociocultural

influence, regarded as an important factor among female patients, is of less significance in male anorexics.

As Klinefelter's syndrome is almost certainly under-diagnosed, it is suggested that atypical cases of male AN be subjected to chromosomal analysis.

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Books Reconsidered

*Dementia praecox oder die Gruppe der Schizophrenien: 'Eugen Bleuler**

MANFRED BLEULER AND RUDOLF BLEULER

Eugen Bleuler's motivation for his work with schizophrenic patients and for describing their psychoses in a manual (By Manfred Bleuler)

Bleuler's aim in becoming a doctor and his interest in schizophrenia are deeply rooted in his origins. He was born on 30 April 1857 (his father lived from 1823-1889, his mother from 1829-1898). All his ancestors were from Zollikon, which was, up to the end of the last century, a rural village an hour's walk from the city of Zürich. The population of Zollikon lost some of its ancient rights during the 18th century and had been increasingly subjected to the government of the city of Zürich. Up to 1830, an academic career was unusual and difficult for the rural population; it remained difficult during Bleuler's youth. It is astonishing, however, that in spite of the difficult access to academic education, the intellectual interest and the enthusiasm for the ideals of enlightenment grew even greater among the rural population.

It became increasingly important for the rural people to give an academic education to their young men. The hope grew: if our sons are academically trained they will be able to perform their duties as clergymen, judges, and particularly as doctors as well, or even better, than the old aristocrats of the city. Under these circumstances, Eugen Bleuler decided to become a doctor, and he understood medicine as part of natural sciences.

The first directors of the psychiatric University Clinic Burghölzli in Zürich (opened 1870) were highly qualified and internationally-known professors who became famous on account of their research in neuropathology and in neurophysiology. As Zürich at this time had no academic tradition, they had been called to their chairs from Germany.

The rural population felt that these great men had no personal contact with their psychotic patients on account of their academic interests and their language (the population spoke a German dialect). Under these conditions, Bleuler conceived the overwhelming wish to become a modern doctor who lived and spoke with them in their mother-tongue. He had the wish to become a psychiatrist such as the population around him had dreamed of - a psychiatrist who

could help his patients both by modern medicines and by understanding them personally and being close to them.

Bleuler completed his medical studies in Zürich in 1881. In the following years, he was an assistant in the psychiatric clinics of Waldau-Bern and Burghölzli-Zürich and also studied in Paris (under Charcot), in London, and in Munich (under von Gudden). One of his first publications as a young student concerned synaesthesia (1880). There followed papers on cerebral anatomy, neurology, and neurophysiology.

While an assistant of Auguste Forel in the Burghölzli Clinic, Bleuler was chosen as director at the psychiatric Clinic Rheinau, at the age of 29. He stayed there from 1886 to 1898. Rheinau was at that time a small village of farms. In the 8th century, a monastery had been founded there on an island in the Rhine. In 1867, it had been turned into a psychiatric hospital. Bleuler was not yet married, and lived there alone, in contact with his patients. He worked with them (mostly in agriculture), organised their free time (for instance, hiked with them, played in the theatre with them, and danced with them). He was also the general physician of the patients of the Clinic and the inhabitants of the village. During his life with the patients, Bleuler had always a memo-pad at hand, where he noted what touched and interested him in his patients' behaviour. He frequently noted in shorthand what the patients actually said.

In 1898, the government of the Canton of Zürich appointed Bleuler as Auguste Forel's successor as Director of the Psychiatric Clinic Burghölzli and as Professor of Psychiatry at the University. He did not wish to leave his patients in Rheinau, but in his new position in Burghölzli, he was much closer to his sick parents.

In his new position, Bleuler had less time for his patients, as he had many other duties. He worked in close touch with his staff, which included Carl Gustav Jung from 1900-1910; they discussed (in touch with Sigmund Freud) the psychodynamic life of schizophrenics.

In the first years of our century, a group of German psychiatrists, headed by Gustav Aschaffenburg, Professor in Cologne, planned to write a great *Manual of Psychiatry* (13 large volumes). Its main aim was the exact description of the manifold psychopathological phenomena, and an effort to outline disease entities

*Published by Franz Deuticke Editor (1911) Translation in English (1950) *Dementia Praecox or the Group of Schizophrenias* New York. International Universities Press

with specific cerebral causes. This was intended to be a continuation of what Kraepelin had inaugurated with his textbook.

Aschaffenburg knew Bleuler both on account of his contribution to the introduction of Kraepelin's conceptions and for his great but critical appreciation of Freud's concepts. Bleuler's motivation for writing the *Manual's* volume on *Dementia Praecox* clearly reflected his principal aims in life: he wanted to be a physician in the sense of his time and to contribute to the progress of medicine, and he wanted at the same time to be close to his patients and to know and study them personally. He was eager to describe psychopathology in an objective, scientific way and to suggest at the same time the importance of understanding the personal psychodynamic life of the individual patient.

The main text of the book

It commences with a short presentation of the history of the concept 'dementia praecox'. Bleuler introduced the term 'schizophrenic psychoses' both in order to eliminate the erroneous idea that the patients become 'demented' and to take into consideration that not all patients become psychotic at an early age. His designation also expressed his doubts in regard to the assumption that 'dementia praecox' corresponds to a disease entity. The following main text, under the title 'Symptomatology', describes in an exact and objective way his observations while living with schizophrenic patients in Rheinau. The chapter is subdivided into 'elementary functions' (the dissociated associations and affectivity, the ambivalence) and 'secondary symptoms' (as for instance hallucinations, delusions, morbid attitudes). All these descriptions are mainly based on Bleuler's daily notes while with schizophrenic patients. These notes filled several thousand small pages written in shorthand. This is the first and most important characteristic of the book: *it is mainly based on personal and direct observations.*

The second characteristic of the book consists of *an endeavour to discover the psychological individual background of the psychotic symptoms.* This is not done by theoretical considerations but by noting observations which concern the psychological background. From Bleuler's description of 'symptoms' emanates his empathy for the patient to the reader. To what is such an emanation due? Certainly not by direct calls for empathy! It may be due first to the fact that Bleuler gives well-observed examples of symptoms noted in much more detail than an impersonal description. Bleuler also repeats again and again that the symptoms were observed in a patient whose emotional and intellectual inner life is essentially

intact, but frequently hidden. Thus, we gain the impression that we are in duty bound to accept the schizophrenic as one of us, and as one who merits our help. The same effect is gained from the demonstration that many 'symptoms' are symbols for emotionally laden ideas of the patients, just as the contents of our dreams are often symbols. Bleuler also describes how patients suffer from ambivalence, from contradictory emotions and impulses (due to double-bind in modern jargon). As it is true of everyone's life to overcome ambivalence, the description of the schizophrenic's difficulties suggests the idea: "He is one of us and we can feel with him".

An example: A schizophrenic woman from the Burghölzli Clinic had asked to see Bleuler. When he saw her, he found her mute and in catatonic stiffness. He suddenly noticed, however, a little movement of the patient: for a short while, she touched Bleuler's wedding ring. This movement suggests the interpretation that the patient wanted to express: "I look like a stone, but I am a woman with healthy feelings appreciating marriage". Bleuler never maintained that his impression of his patient's motivation for touching the wedding ring really corresponded to what was going on in her thoughts. The observation, however, qualified his impression as possibly realistic. And it is certain that Bleuler's impression due to objective observation aroused his empathy for a patient 'looking like a stone'—and it evokes the empathy of the book's readers for catatonic patients.

Chapters superseded by modern research

While Bleuler's exact, minute descriptions of his schizophrenic patients (suggesting empathy through objectivity) are still of value and interest in modern times, the following chapters on course and outcome of psychoses and on the families of schizophrenics have lost their importance in the light of new research since 1911.

Bleuler, however, had already shown that the course of schizophrenic psychoses is extremely varied—with one exception: the schizophrenic never becomes 'demented' and never loses his intellectual and emotional life, even if it is often hidden behind his dissociation of thoughts and emotions. Bleuler mentioned that many schizophrenics recover so far that they can live a healthy life outside hospital. He hesitated, however, to speak of absolute, complete recovery. One of the reasons lies in the critical evaluation of the patient's prepsychotic personality. If prepsychotic personality difficulties continued to exist after social recovery, Bleuler asked himself critically: was perhaps the prepsychotic personality disorder already a sign of the oncoming psychosis?

He feared that the designation of complete recovery was uncertain in such a case, and was not permitted by objective, scientific thinking. Furthermore, for many years Bleuler's main duties did not allow him to follow-up the course of many patients after their discharge from hospital, particularly not during his isolation in Rheinau. Compared with his great experience with hospitalised patients, his experience was restricted with patients discharged from hospital.

Theoretical considerations

One of Bleuler's main aims in choosing and following his career was to arrive at an understanding of the schizophrenic symptoms as expressions of an inner psychodynamic life. And one of the main aims of his book was to describe the results of these endeavours, and to introduce them into modern psychiatry. He studied the schizophrenic's inner life essentially in the same way as we study the inner life of neurotics, of healthy men, and of ourselves. He included in this study ideas of Freud, and discussed them with him.

Bleuler was successful in his endeavours to recognise schizophrenic 'symptoms' as reflecting the patient's life experience, his cognition, his emotions, his fears and hopes. His way of analysing the schizophrenic's inner life enabled him – and enables us – to feel with the schizophrenic, to acknowledge him as 'one of us', a man as we are, worthy of the same social and medical care when in stress and difficulty. The great importance of this becomes evident when we realise how strong tendencies in the opposite direction have frequently existed in all cultures and at all times. All too frequently, the schizophrenic was considered to be no longer one of us, to be wracked and ruined for good, to be a demented and degenerated man, or a man possessed by evil spirits or demons. Even in the psychiatry of our century, there has been a strong trend in this direction, declaring that the inner life of the schizophrenic was incomprehensible to the healthy.

While Bleuler understood the schizophrenic symptoms as a direct or symbolic expression of the patient's inner life, he never claimed that his psychological understanding explained the patient's psychosis. In other words: he did not understand why psychologically intelligible parts of the patient's inner life lose control by experience and logic, to a degree which characterises him as a psychotic, as a man who loses his usual social position. Bleuler understood, for instance, that a schizophrenic's ambivalence is essentially of the same nature as ambivalence of the healthy, that it is due to contradictory emotionally laden life experiences, to the double-bind. However, Bleuler did not understand why this ambivalence becomes overwhelming, why

the patient thinks, feels, and acts in many respects as if there were different souls in him, as if he consisted of different personalities, that he becomes 'split' to a psychotic degree.

It was logical in this situation to assume that hidden behind the schizophrenic symptoms, which are psychodynamically intelligible, is concealed a 'primary disorder'. Like most psychiatrists of his time, such as Kraepelin and Sigmund Freud (and many other psychiatrists of our times), Bleuler supposed that the 'primary disorder' was a metabolic or anatomical cerebral disturbance. Bleuler mentions, however, the possibility that anomalies in personal development could be a primary cause of schizophrenic psychoses. Bleuler looked for symptoms of the 'primary disorder' which were independent of the psychodynamic life. He suspected some physical findings to be 'primary' symptoms, but we know today that some of them are of another nature. He believed that the splitting (the dissociation of thoughts, of emotions, of attitudes and of acting) were close to 'primary symptoms', while he understood most of the other symptoms of the psychosis as due to the intrapsychic, psychodynamic life, as being 'secondary'.

The terms 'primary and secondary' symptoms of schizophrenic psychoses are no longer in use, and sound old-fashioned. The essential content of Bleuler's theoretical conception, however, is astonishingly similar to the conception of most modern psychiatrists: they agree with Bleuler that the symptoms of the schizophrenic psychoses can be understood psychodynamically, and they agree with Bleuler that this understanding does not, as yet, answer the great question: why has the schizophrenic lost all consideration for reality and logic in many ways during his fight to harmonise the inner disharmonies (the double bind) of his life experience? Why has he become psychotic in spite of an inner life which we feel to be near our inner life? In this regard too, most modern psychiatrists hypothesise like Bleuler that behind the psychodynamically intelligible morbid symptoms exists a hidden morbid background of the psychosis. They no longer call it, as Bleuler did, 'primary disease', but their description of 'vulnerability' with regard to schizophrenic psychoses or hidden disturbances only visible by 'markers' corresponds closely to Bleuler's conception. As Bleuler did, most modern psychiatrists suppose that this 'primary disease', this 'vulnerability', is due to an abnormal cerebral function – today, in particular, of a neurohumoral origin. As in 1911, however, up to the present time, no specific derangement such as the deepest and specific derangement of schizophrenic psychoses has been discovered.

The chapters on 'Therapy' (By *Rudolf Bleuler*)

Bleuler excluded the superfluous and the questionable, and based therapy on an established effective foundation. Considering the treatment methods at the present time, we may understand his position. But this attitude explains also why Bleuler expressed himself carefully about the dialogue with his patients.

What principles are specified in his book? The symptomatology of the schizophrenic patient is essentially comprehensible. The underlying factors are the therapist's profound empathy, his sufficient knowledge of the patient's personal history and of psychodynamics, as well as his awareness of the fact that healthy, although split off or distorted parts of the personality, persist even in the most disturbed patient. Uncovering psychotherapy, as with the neuroses, helps only in the less severe cases. The therapist practises with his patient mainly reality-testing and impulse-control. Daily routine and living together in the clinic are aspects which should be embedded in a structured environment. Acting-out destructiveness must be prevented, but at the same time *freiraum* and tolerance should be taken into account, more so than would be possible outside the clinic. The vehicles of therapy are dialogue and an active community life.

And what if we had to summarise the bases of treatment in the present day? We would confirm the fact that therapy should be less uncovering and more structured. In our opinion, the following is true: first, the therapist is not just a good, knowing, and commenting partner in a dialogue, but is the person who fulfills the patient's desire for a parent figure and who, in certain areas of the relationship, is omnipotent and able to fuse with him. This means that the therapist expresses clearly what he thinks to be correct or incorrect. He is capable of verbalising the unspoken feelings and thoughts of the patient, and presenting them in a framework which provides security. He makes no secret about his own feelings and associations which arise in the therapy, or about his personal gain. He confirms the experiences communicated to the patient in the dialogue in a well defined framework on the level of action, which in the end result, is more important for the patient. And, step by step, he draws himself out of his parent role, but is available to the patient if necessary for years. Second, understanding is less causal and analytical than purposive and synthetic; abnormal experience is declared as an expression of a healthy, constructive striving, and must be led out of its distorted and threatening state. Third, because anxiety-raising feelings and impulses are not repressed into the uncon-

scious, but instead are split off into other conscious areas of the personality, it is not the task of therapy to let them become conscious, but to (re)integrate them into the whole of the personality. Fourth, disturbed functions should not just be pointed out, but also corrected and actively trained, for example, cognitive and affective control of reality, phantasising and symbolising, organising and weighing of matters, coherent communication, metacommunication of contradictions, etc. Fifth, the healthy resources of the patient and his family are not just an implicit presupposition for therapy; they should be explicitly applied and strengthened.

If we compare this description with Bleuler's basic statements of 1911, we notice that the latter have maintained their validity up to the present time, but have been developed further and made more precise. Concerning the realisation of these principles in the clinic – Bleuler's main field of work – a certain loss is apparent. There was a state of distress concerning personal and financial matters; there were the difficulties of the pre-neuroleptic era, and the extensive relinquishment of private life. However, one cannot avoid seeing that the clinical setting at the time was more a community of life and destiny between patients and nurses than it is today, and in this sense, it came closer to the ideal of the active therapeutic community.

Conclusion

The main text of Bleuler's book consists of a careful and objective description of his observations during long-term living with schizophrenics – a description which demonstrates the direct and symbolic correlation of the schizophrenic's symptoms with his inner, psychodynamic life.

It is furthermore important that he shows again and again that the schizophrenic is never 'demented' in the sense of Bleuler's time; that he preserves in his background the intellectual abilities and emotional life of the healthy. From Bleuler's minute descriptions of what is healthy in the schizophrenic, and of the correlation of his symptoms with an inner life close to our inner life stresses, comes the conception that the schizophrenic patient is not foreign to us, that he is not unintelligible, and that we can develop empathy for him. The schizophrenic patient needs our help, as does any patient.

For the care of the schizophrenic patient, an active therapeutic community is important. It is essential in therapy that nurses, doctors, relatives, and others are in a natural way near the patient, sharing parts of life with him.

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Psychiatrists on Popper

Sir: When a philosopher's work becomes as well known as that of Sir Karl Popper, it must be expected that it would be mis-quoted and mis-interpreted on occasions. Popper appears, however, to suffer more than most, often at the hands of psychiatrists. There have been two recent examples of this.

Taylor (*Journal*, July 1986, 149, 37-41) in his fascinating article on 'Hysteria, Play-acting and Courage', uses Popper's concepts of worlds I, II and III in support of his argument that hysteria is not a mental state but a product of human culture more akin to play-acting. In Popper's terms (Popper & Eccles, 1981) this is therefore a world III phenomenon: world III being the universe of human artifacts such as novels, plays, and gardens. World III develops when consciousness or the human mind (world II) acts upon the material world (world I) to alter it in a meaningful way. Diagnosis, according to Taylor, is a world I phenomenon. This, I think, can be contested. Diagnosis is a concept and hence belongs either to world II or III. For example, in a disease (diagnosis) carcinoma of the lung, the world I phenomenon which relates to this diagnosis is the cancerous tissue itself, which, in itself, is not the diagnosis. That requires a mind (world I) before it can come into existence as a world III phenomenon. Hence, since all diagnoses/diseases are world III phenomena, the use of Popper's concepts in no way advances Taylor's argument that hysteria is different from other diagnoses, an argument which may nevertheless be correct.

The second example by Mathers (*Bulletin*, May 1986, 103-104) criticizes Popper's theory of science as it applies to psychiatry and, in particular, psycho-analysis. Popper's epistemology is a demonstration of the logical asymmetry between verification and falsification in the proof of theories. Mathers disagrees, preferring to believe in the possibility that falsifiability is not a prerequisite for testability and that theories can be *proven* to be true as well as to be false. In that case any theory which has been as decisively proven to be true as many have been proven false must be produced in evidence for this startling assertion. Of course many theories are held to be true by the scientific community because their falsifiability has been used to test them so frequently that a sort of Popperian truce has been called for the

time being – but always with the possibility that an experiment at sometime in the future might disprove or modify the theory.

Mathers makes a further assumption which is attributed by implication to Popper, that non-science (psycho-analysis) is the equivalent of non-sense. Popper has pointed out that this is not the case. Indeed it would be difficult to devise an experiment to falsify Popper's own epistemological theory, but I doubt if he would accept that it is nonsense. Hence, Popper would not necessarily be led into asserting that analysis is non-sense only that it is non-science. Dr Mathers has therefore inadvertently performed for analysis more of a disservice than did Popper.

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Tardive Dyskinesia and Parkinsonism

Sir: We read with interest the letter by Drs Oyeboade & McClelland (*Journal*, July 1986, 149, 122-123). Their results coincide with our clinical observations that coexistence of drug-induced parkinsonism (PS) and tardive dyskinesia (TD) in the same subjects is not exceptional, and that the intensity of the two disorders is not correlated. Nevertheless, we do not think that 'an independence of their relative severities calls into question the currently held view that PS results from a blockade and TD a hypersensitivity of dopamine receptors'. Nor do we consider that 'the two conditions are either mediated through different dopamine systems or through independent but related neurotransmitter systems'.

Easier explanations are available. Some nigro-striatal dopaminergic regions could be hypofunctioning as a result of the neuroleptic blockade of these neurons, while other regions could have become 'hypersensitized' after a prolonged blockade at the same time. Differing levels of receptor sensitivity in the corpus striatum have been proposed by Carlsson (1970). Moreover, observations of parkinsonian tremor shifting into a dyskinetic movement would support the hypothesis that dopaminergic hyperfunction, clinically TD, would develop after

hypofunction produced by dopaminergic blockers, clinically PS (Goetz *et al*, 1982). A decrease of striatal dopaminergic neurotransmission by neuroleptics increases sensitivity for the posterior appearance of TD determining the later localization since it affects the same substrate and implies a topographical correlation of both disorders (Garcia Ribera *et al*, 1985). Crane (1972) showed that 'tardive dyskinesia is more likely to develop in patients with pseudo-Parkinsonian symptoms than in patients not exhibiting such manifestations'.

In similar cases, such as L-dopa induced dyskinesias in patients suffering from idiopathic Parkinson's disease, the severity of both disorders is frequently not related without calling into question the coexistence of dopaminergic hyperfunction and hypofunction. In spite of the apparently opposite theoretical support, coexistence of both disorders could be the expression of two different moments in the same physiopathological process. Prospective studies are needed to clarify how and where both drug-induced disorders appear, disappear or shift.

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Psychotherapy and Placebo

Sir: Professor Eysenck (*Journal*, May 1986, **148**, 610) says that I failed completely to understand his point. I feel that, on the contrary, it is he who has not understood mine.

I share Professor Eysenck's scepticism as to the 'specificity' of the various types of analytic psychotherapy. I have long thought that any positive or negative effects of the various types of psychotherapy have more to do with the 'non-specific' personal relationship between therapist and patient than with 'specific' factors inherent in the particular therapeutic technique employed — Professor Frank's

'shared therapeutic functions' in fact. That such factors are, to some extent, also operative in behavioural as well as analytic psychotherapy was suggested by the study of Sloane *et al* to which I referred. However, if it is the case, as Professor Eysenck states, that comparative studies show a clear superiority for behaviour therapy over placebos in general and the other psychotherapies in particular, then this is, of course, an important finding, which I never questioned.

The actual words of Professor Eysenck to which I referred and to which I objected were — 'Do we have the right to... get the State to pay us for treatment that is no better than a placebo?' My objections to this were threefold. Firstly, I wished to draw attention to the confusion that can occur in psychotherapy research because both the 'specific' treatment in question (psychotherapy) and the 'non-specific' placebo with which it is compared are both presumed to be psychological in their mode of action. Attention to the conceptual confusion surrounding the terms 'specific' and 'non-specific' in placebo theory has recently been drawn by Professor Grunbaum. His article supports my contention that the allotment of the various therapeutic factors to the categories 'specific' and 'non-specific' (or in his preferred terminology 'characteristic' and 'incidental') is purely arbitrary and relative to the theoretical standpoint of the investigator concerned. Unless we are clear about this Eysenck's statement that 'psychotherapy is no better than a placebo' could be reduced to the vacuous proposition 'psychotherapy is no better than psychotherapy.' I objected secondly because a great deal of psychotherapy is in fact done with little extra cost to the State, and thirdly because the phrase 'no better than a placebo' appears derogatory — especially insofar as people tend to associate placebos with inert pills. In his letter he says that 'placebo treatment is as successful as psychoanalysis and psychotherapy in general...' This at least implies acceptance of the fact that general psychotherapy does have some degree of success even if this is not quantitatively greater than that of placebos in general. Furthermore, the fact that the effects of psychotherapy may not be *quantitatively* greater than those of placebos in general does not, of course, imply that they are *qualitatively* identical. Few, for example, would exchange a therapeutic relationship (whether in a personal or a professional setting) for an inert pill even though both could be regarded as placebos insofar as they are 'non-specific' and psychological in their effects.

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The Prevalence of Mental Illness Among People with Mental Handicaps

Sir: A recent publication of the Royal College of Psychiatrists (Wilkinson & Freeman, 1986) contains the following statements:

"Nearly one half of mentally handicapped children and adults suffer from associated psychiatric disorder. Among mental handicap hospital populations, the prevalence is between 30% and 60%" (p. 117).

"Several surveys indicate the prevalence of severe mental illness (psychosis) in mentally handicapped people is between 11% and 13% of hospital residents" (p. 122).

The discussion which followed suggested a weight of opinion in favour of the first quotation.

Two years ago I summarised all the evidence I could find (over a dozen studies) on the subject (Ineichen, 1984). Most of those which gave a figure based on diagnosed mental illness, rather than vaguer measures such as 'disordered behaviour' clustered around the 10 to 14% mark. The highest figure of all was 58.8%, including only 15.8% severe cases which warranted 'continuous and perhaps intensive in-patient care from psychiatrists' while the rest require 'at the most occasional psychiatric specialist attention' (Williams, 1971), the figure of 'up to 60%' has been in circulation for a decade or more. I am still waiting to find a single study which justifies its use.

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Violent Behaviour in Psychiatric Hospitals

Sir: I read with interest the paper by Pearson *et al* (*Journal*, August 1986, 149, 232-235) and felt that the

report of the high incidence of violent acts at mealtimes was worth further discussion. Phillips & Nasr (1983) noted a peak of violent incidents resulting in seclusion or restraint between noon and 2.00 p.m. and in a study of my own, incidents resulting in seclusion were about twice as common at mealtimes than at any other time. There was a tendency for non-psychotic rather than psychotic patients to be involved in such incidents, but the type of incidents were no different from those occurring at other times.

Kinzel (1970) suggests that schizophrenic patients are prone to disturbed behaviour at times when they perceive their "body-buffer zone" being encroached upon, particularly if from behind, and Bigelow (1972) states that "the crowding of strangers, especially near such valued resources as food" may result in aggression.

I believe that study of the facilities used in psychiatric units for the serving and eating of food by patients may allow modifications to be made, so reducing the incidence of violence.

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KINZEL, A. F. (1970) Body-buffer zone in violent prisoners. *American Journal of Psychiatry*, 127, 59-64.
PHILLIPS, P. & NASR, S. J. (1983) Seclusion and restraint and prediction of violence. *American Journal of Psychiatry*, 140, 229-232.

Schizophrenia and Ethnicity

Sir: One of the most intriguing facts to emerge from the World Health Organization's International Pilot Study on Schizophrenia (World Health Organization, 1974) is that two-year outcome was the higher percentage of good outcome schizophrenics in Third World countries as opposed to First World countries.

The CATEGO Class S Schizophrenia Study (*Journal*, December 1985, 147, 683-687) is presently being analysed regarding outcome at two years. Preliminary findings indicate that the Xhosa schizophrenics in the sample have a better outcome at two years than the White schizophrenics, but the reason for this is not known at this stage. However, there are distinct differences in terms of cultural factors, attitudes to mental illness, compliance with maintenance medication, expressed emotion and living in extended families, which are of great importance in

transcultural psychiatry. These are facts which have a bearing on the nature and management of the disease and it is to be deplored that Drs Sashidharan and Lipsedge have politicised this information in their recent letter (*Journal*, April 1986, 148, 484).

Scientific truth is a bastion of strength in a world which is confused by misinterpretative and exploitative statements. It is particularly distressing in this regard that Drs Sashidharan and Lipsedge have cast aspersions on the scientific integrity of the *Journal's* Editor and assessors by stating that an article "with politically loaded definitions of little scientific import" was published.

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Reference

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Depression in Schizophrenia: Prevalence and Treatment

Sir: With regard to the paper by Elk *et al* (*Journal*, August 1986, 149, 228-229) although the point has been made previously elsewhere it seemingly needs to be made again.

The "Population Registration Act, 1950 (as

amended)" does not define homogeneous "ethnic groups" but groups together "whites, blacks and coloureds" whose members may have very different origins, languages, cultures and social circumstance from co-members of the same so-called groups. By accepting this paper in its published form I feel that the *Journal* has been done a disservice and that another layer of veneer has been placed on the pseudo-scientific basis of apartheid.

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Control of Auditory Hallucinations with Earplugs

Sir: I read with interest Dr Birchwoods article (*Journal*, July 1986, 149, 104-107). I can recall very clearly two schizophrenic patients who wore earplugs to shut out the 'voices'. The first one was a male in his forties that I saw at Castle Peak Hospital in Hong Kong in 1970 and the second was a male patient in his sixties at Springfield Hospital in London in 1977. As I recall, the first patient wore earplugs in both ears while the second only in his left. Both of them told me that the earplug helped to block out the voices. At that time I said to myself "this is just part of their thought disorder" and paid no further attention to it. Another lesson that we should listen more to what our patients say.

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A HUNDRED YEARS AGO

A Lunatic Imprisoned Again

Why do the City Fathers persist in imprisoning Herbert Percy Freund, seeing that he is certainly "a person of unsound mind"? Is it that they deem it less costly to keep him one-third of his time in gaol than to maintain him altogether in an asylum? The question of humanity ought to have some little weight as against the question of economy. Besides which, it may one day happen that this lunatic will no longer

be contented with declaiming against St Paul's Cathedral as "that house of idols over there". He may turn iconoclast on his own account; or, instead of predicting the downfall of the City, he may do something towards the fulfilment of his prophecies. The policy of the City magistrates is neither sound nor safe, and it is certainly anything but benevolent.

Reference

Lancet (February 20, 1886) Pp 368

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.

Book Reviews

Mental Health Law (2nd edition). By BRENDA HOGGETT. London: Sweet & Maxwell. 1984. Pp 370. £12.95.

Mental Health Act Manual. Edited by RICHARD M. JONES. London: Sweet & Maxwell. 1985. Pp 318. £16.75.

Mental Health Services—Law and Practice. By LARRY GOSTIN. London: Shaw & Sons. 1986. Catalogued pages. £85.00.

The Mental Health Act 1983 (England and Wales) is a complex document, like all Statutes, and it is often difficult to follow or is capable of a variety of different interpretations. In comparison with its predecessor there is now an increased emphasis on the rights of mentally disordered patients, greater responsibilities for the social worker, entirely new sections about consent to treatment and the establishment of a Mental Health Act Commission and a Code of Practice which is still very much in the early phase of creation. Inevitably the Act has stimulated the production of a number of new books providing guidance and interpretation and these are three excellent examples.

Each provides a different perspective, and complements the other two. The first is a helpful text; the others works of reference.

Professor Hoggett's book is unquestionably the most readable of the three. The first edition, entitled simply *Mental Health*, was I believe, unknown to most psychiatrists as her (legal) publishers neglected to publicise it to doctors and that was a great pity. The second edition confirms Professor Hoggett as a leading authority in the field of mental health law and it is highly recommended. It deals broadly with all legislation which affects the mentally ill, discusses policies underlying it, the way it is used, interpretation and weaknesses. The material is very well presented, interesting, well-referenced and indexed and the book is reasonably priced.

All three authors are lawyers. Professor Brenda Hoggett is a Law Commissioner. Richard Jones (described as editor but apparently author) is a social worker, solicitor and senior lecturer in social administration at Cardiff. He is also a Mental Health Act Commissioner. His book consists of detailed 'annotations' of each section of the Act (each of which is reproduced) so that the result is rather like

reading a book consisting entirely of footnotes. Most of the comments are supported by reference to authorities such as decided cases, other legislation, memoranda or other authority. Some are the author's own opinions. It is primarily a lawyers manual but would also find its place as an important reference book for specialists in mental health law from any profession, but it is not a book for the student or for those who only require general guidance. But it usefully also collates details of all the forms used in applying the Act, the Regulations, Court of Protection Rules and relevant DHSS circulars.

Larry Gostin is also a lawyer, well known in this country as Legal Director of MIND during the passage of the Act and now Senior Fellow in Health Law at Harvard. He has produced a veritable *magnum opus* which replaces Shaw's *Mental Health Services*, a long out-of-date and much smaller publication. This text is published for the first time in loose-leaf format with the intention that it can be regularly up-dated with supplements provided by the publishers as the law changes. It consists of eight major parts dealing with the structure of the Mental Health Services, personnel (managers, doctors, nurses, social workers, relatives—their roles and functions), admissions to hospital (civil and criminal), the interface between the criminal law and the mental health services, discharge and guardianship, the therapeutic relationship, patients' rights and criminal offences. All of these topics are discussed fully and indeed objectively, with detailed references to case law and literature. Virtually all the relevant statutes, (e.g. the whole of the Mental Health Act 1983), regulations, schedules and circulars are reproduced in full. It is a remarkable achievement and a valuable work of reference but it is not bedside reading. The pages are not numbered and it is difficult to find one's way around; the value of the book would be enhanced by an index. It resembles Gray's *Anatomy* or *Archbold* in size and the publishers might usefully consider a more manageable format if this book is to reach mental health professionals (or even families and patients themselves as the preface hopes) as well as lawyers. It deserves to do so although it is most likely to find its place in the library or on the desk of the enthusiastic specialist.

ROBERT BLUGLASS, *Professor of Forensic Psychiatry, University of Birmingham*

Contemporary Issues in Schizophrenia. Edited by ALAN KERR and PHILIP SNAITH. London: Gaskell (Royal College of Psychiatrists) 1986. Pp 483. £17.50.

This handsomely produced volume is intended as an update of the first special publication of the *British Journal of Psychiatry* in 1967. That was a much more modest affair with the text of ten lectures on schizophrenia sponsored by the RMPA. This time the editors have assembled 29 papers and three 'discussions' on schizophrenia which appeared in this *Journal* between 1975 and 1984. They tell us that "many interesting scientific papers have not been included, preference being given to papers presenting broad reviews". The papers have been edited by the omission of tabulated data and, surely a mistake, of summaries.

The book contains seven sections. The first, on general and historical concepts, includes Kendell's influential 1975 paper on the concept of disease. Six other papers are reprinted, among them one by Szasz with a convincing riposte by Roth. In the second section, on classification and phenomenology, there are two papers on first rank symptoms (by Koehler and Mellor) as well as papers on the US:UK project, DSM-III and other nosological topics. Organic aspects, in the third section, are covered by five papers. Topics include the role of dopamine as well as the viral hypothesis, but there is no paper on CT-scanning. Genetic aspects are covered in four papers in the next section. The editors have been able to find only two suitable papers from the *Journal* for the fifth section on social aspects. One is a speculative piece but the other is a follow-up study by the Northwick Park group of schizophrenics discharged from Shenley. Section six deals with treatment and the three reprinted papers deal only with drug treatment. Finally there is a section on outcome.

The editors' problem is now clear. It is not possible adequately to illustrate contemporary issues in schizophrenia by reprinting 'broad reviews' from any single English language journal. For example, the 1982 volume of *Psychological Medicine* contained reviews by Marsden on motor disorders in schizophrenia and by Stevens on the neuropathology of schizophrenia. Neither topic is represented in the present volume. American and Scandinavian journals during 1975-1984 also often published important papers on schizophrenia, particularly on pharmacology and on community care.

The editors' answer to this problem was to commission 15 new papers. They asked eight "researchers, prominent in a particular field, to provide a new review of their field of interest" and seven "dis-

tinguished psychiatrists to present a series of overviews of the present status of knowledge".

Contributions from the researchers are presumably intended to be surveys of the recent literature and there are eight of them. Topics include cognitive impairment, genetic counselling, expressed emotion, chronic schizophrenia in the community, token economies (in 1986!), rehabilitation, and depressive symptoms in schizophrenia. The seven overviews deal with historical aspects, nosology, organic, genetic and social aspects, pharmacological treatment and outcome. Most of these 15 contributions are up to date, readable, brief yet comprehensive. They occupy 144 pages of text and might have made a useful successor to that earlier publication. Alternatively the editors might have made a collection of the most important papers on schizophrenia in English in the last ten years. The present book is an unsatisfactory compromise.

J. L. GIBBONS, *Professor of Psychiatry, University of Southampton*

Psychotherapy of the Combat Veteran. Edited by HARVEY J. SCHWARTZ. Lancaster: MTP Press. 1984. Pp 315. £22.50.

The main subject of this book is the Vietnam Veteran (VV) and the main therapies are psychodynamic. Combat neuroses in World Wars 1 and 2, the survivor syndrome and the post-traumatic stress syndrome are briefly discussed, principally to illustrate the differences between the long-term emotional problems of the VVs and of combatants in other wars. After a summary, by the editor, of the psychoanalytic approach, with an illustrative case history, there are chapters on the history of the concept of war neurosis; psychoanalytic psychotherapy and the VV; unconscious guilt; transference, counter-transference and the VV; nightmares; brief psychotherapy; and group psychotherapy. Schwartz then writes about the 'Fear of the Dead'; and there two 'cuckoo-in-the-nest' chapters, one on treatment with a drug (a monoamine oxidase inhibitor) and another on the Israeli experience in six wars since 1948.

The clinical features of combat trauma, described by most contributors, include the overwhelming nature of the trauma, producing 'severe damage to the ego' and regression to a primitive level of functioning. Schwartz especially, emphasises the importance of unconscious guilt and the difficulties during treatment (for therapist and patient) because of regression to these infantile levels. Typical symptoms are recurrent, repetitive nightmares; memory flashbacks to the traumatic events; anxiety, irritation,

depression and panic attacks; aggressive outbursts; and somatic symptoms. The VVs have problems with alcohol and other drugs and in their marriages and relationships at work. They often commit suicide, sometimes murder and frequently end up in prison.

These are the general features of the combat neuroses. An additional crucial problem for the VVs is that they are not accepted by their family and the local community when they come home. Neither their actual mothers nor Mother-America welcome them home as heroes. And their images of masculinity, often personified in John Wayne or John F. Kennedy, cannot be fulfilled.

I can only draw attention to a few details. Schwartz highlights the contrast between the apparent lack of conscious guilt, whereby there are no regrets for cruel actions performed and humanitarian acts left undone, and persisting unconscious guilt. In the chapter on brief psychotherapy McKinnon writes about 'dulled forgetting' and 'intrusive remembering' and the importance of changing this 'dysbalance' between remembering and forgetting. Parson describes a multi-group approach, using three different types of groups for three stages of treatment. The first group is supportive and fairly superficial; next comes an insight group and, finally, a recovery group in which wives, sisters, widows and other VVs participate, in preparation for life in the world outside. All the authors describe differences in treatment techniques for the VVs compared with those used for other combatants and with dynamic psychotherapy in general. The focus is on the actual trauma and its relationship to present difficulties rather than on the patient's early life. The authors advise against early interpretations because of the danger of too rapid regression; they emphasise the need for and the difficulties in holding these men. Another problem is the concreteness of the VVs memories: these men cannot develop metaphors and symbols to deal with their terrifying experiences; events which took place ten years before seem to be actually occurring today.

Although these features are present with other very disturbed patients what does seem to be different is the non-acceptance of the VVs by their families and communities. Schwartz, in his chapter on the 'Fear of the Dead' proposes a ritual home-coming welcome—a cleansing and guilt-reducing ceremony—to exorcise the horrors.

I found this book both boring and interesting. Boring because of repetition and overlap amongst the various authors; and because psychoanalytic terms are used extensively, which will make it difficult to read for many and a complete turn-off for some. But the book is interesting because it faces the

contrast between conscious and unconscious mechanisms, because it gives practical descriptions of therapy, and because it not only includes failures as well as improvements but also highlights difficulties for the therapist. It has the usual characteristics of a multi-author volume, the standard of content, style and rigour of approach vary but on the whole I found it well-written. Schwartz in particular writes vividly (in spite of split infinitives) and some of his phrases—'fantasy of attack by the vengeful dead', for example—linger. The many clinical illustrations are useful; the authors are self-critical although, I suppose inevitably, there are no controls nor independent assessments of results. References are apposite and the index is accurate and sensible. The book should be read, although not necessarily bought, by anyone interested in the combat neuroses; it will also interest therapists who look after very disturbed patients.

At least until now, little has been written about therapy of any sort for the VVs. But two books on the subject were reviewed recently in this *Journal* (1986, 148, 354–355). The VVs were also the subject of two American television programmes—*Lou Grant* and *Hill Street Blues*—in the spring of 1986. It looks as if the problems of the Vietnam Veterans will no longer be ignored.

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Behavioral Epidemiology and Disease Prevention.

Edited by ROBERT M. KAPLAN and MICHAEL H. CRIQUI. New York: Plenum. 1985. Pp 450. US\$69.50.

Epidemiologists have long studied the functioning of health care systems and their impact on the health of populations, but more recently attention has been increasingly focused on the relationship between individual behaviour and disease. As interest in behavioural epidemiology has developed the diversity of background of the various investigators has become increasingly exposed. This is particularly apparent cross-nationally and may account for the relative paucity of data.

In this useful book, Kaplan and Criqui bring together contributions from many of the relevant fields in various NATO countries to provide an international and inter-disciplinary perspective. The book is divided into four sections: Methods in Epidemiology; Epidemiology; Stress; Behavioural Interventions.

Selected methodologic issues in the interface between epidemiology and behavioural science are

reviewed in the first section, the first paper providing an overview of current epidemiological research methods. Response bias in surveys is reviewed while Kaplan describes general health outcome measures and the inter-relationship between epidemiology and policy science.

A wide ranging series of papers providing epidemiological evidence that behaviour is related to the development of chronic diseases is presented in the second section. Some of the evidence, such as Burkitt's work on the importance of high fibre in diet, is familiar but other studies investigating dietary components in heart disease, the conflicting evidence on alcohol and mortality, the possible effect of diet on cancer risk, and the benefits of the control of diabetes are less so and provide thought-provoking additions to the literature.

The third section on stress moves into an area of considerable speculation. Evidence is accumulating from a number of countries that the development and duration of illness is related to mechanisms for coping with stress, and that good social networks can buffer its effect. The problem of measuring life stress and social support is difficult, particularly cross-nationally. This is an exciting field and the papers in this section provide insights into the inter-relationship between psycho-social processes and disease.

The final section of this book focuses on the adequacy of behavioural methods for the prevention and treatment of chronic disease, and behavioural interventions in the light of their expected effectiveness are reviewed.

Although it would have been valuable to have had further contributions on methodology, particularly other important sources of bias such as selection, this book provides a welcome addition to recent literature on epidemiology and behavioural interventions by attempting to integrate these areas from an international perspective.

ELIZABETH WATSON, *Research Fellow, Department of Clinical Epidemiology, The London Hospital*

Child Abuse and Neglect: What Happens Eventually?

By KIM OATES. New York: Brunner/Mazel. 1986. Pp 209. \$25.00.

Child psychiatrists are increasingly involved in the assessment and management of abused children and families and, alongside social workers, health visitors and paediatricians, contribute in an important way to child care planning in the courts. Assessing the long-term risks when an abused child remains with its natural family, the adequacy of past or future

interventions, and the likely effects of separations or rehabilitation is extremely difficult as each child, family and support network together form an individual irreversible experiment and the stakes are painfully high. We are dependent on sound research to guide us and unfortunately because of the overwhelming problems this research entails, the literature, while substantial, does not give us many of the answers we need.

Kim Oates, who is Professor of Paediatrics in Sydney, has provided a major contribution to the field, particularly as he covers in a comparative way, both physically abused children and those who have failed to thrive. In this book he describes two prospective studies comparing these groups of children with carefully matched controls. The first half of the book consists of a thorough review of the literature with suitable emphasis on the rather neglected problem of growth failure and emotional deprivation. Perhaps because growth failure is so easily reversed by hospital admission and many of the children catch up physically later on, they are mainly managed by paediatricians. However, the study confirms that these children remain emotionally and intellectually handicapped in adolescence compared with controls from similar social backgrounds. The study also demonstrates that the families of physically abused children show more adverse psychosocial characteristics than those of the nonorganic failure to thrive group which may lead to greater psychological or community interventions with this group. However, one of the main gaps in this otherwise thorough book is the paucity of detail about interventions with all the families. It seems that almost all the children were with their natural parents at the time of follow-up, but it is unclear how often their management involved court appearances, hospital admissions, foster care or other separations between presentation and follow up, which may have affected the outcome. Provision of community child care by the equivalent of social workers and health visitors does seem less developed in Australia than in Britain where such professionals come under close scrutiny whenever there is a disaster. It would be invaluable to know whether a similar prospective study here would produce similar results. Meanwhile, this book highlights the long term emotional and cognitive handicaps suffered by children who have been abused and neglected and emphasises the need for a continued child centred approach even when there is no longer concern for the child's physical health and safety.

A. HALL, *Consultant Psychiatrist, Emanuel Miller Centre for Families and Children, London*

Health, Race and Ethnicity. Edited by THOMAS RATHWELL and DAVID PHILLIPS. London: Croom Helm. 1986. Pp. 278. £17.95.

This collection of papers centres around the contributions of contemporary medical geography which has moved from biological and spatial concerns to a more sociological position. Useful introductions restate the value of *race* as a sociological term, and point out that population genetics has little role in determining variations in health status: a valid point, but it means that the politics of recent intervention in thalassaemia and Tay-Sachs go unexamined here.

Most of the papers concentrate on literature reviews, and statistics proliferate at the cost of concentration on specific issues such as the rickets debate. There are no contributions by psychologists, psychiatrists or anthropologists and, with one notable exception by Jenny Donovan, there is little on the client's expectations and perceptions. Despite the anti-racist perspective, the general approach is positivist with a rather free and unexamined use of such notions as 'stress', 'support' and 'oppression' without suggesting how these work out at the level of the individual patient or in the subdominant groups' self-identity, behaviour and resistance.

Only one paper is specifically concerned with mental health. John Giggs gives a helpful account of the demographic, statistical and diagnostic difficulties in accurately determining the rates of schizophrenia in minority groups. Using the Nottingham Psychiatric Case Register he replicates previous studies of high rates of schizophrenia in minorities but finds that these rates vary with the particular socio-economic area of the city more than is the case for the white British-born population.

The book is regrettably cheaply produced on the now nearly ubiquitous word processor and includes numerous typing errors. However, there is much important advice and warning here for administrators, community physicians and psychiatrists, particularly on the ethics of research with minority groups. As numerous contributors point out, most work conducted in this area highlights the exotic and cultural aspects, selects out minorities themselves as special 'problems', and totally ignores questions of the accessibility and acceptability of health services or the influence of economic factors. These statistical analyses of minority health have nearly always ignored essential, nosocomial factors.

ROLAND LITTLEWOOD, *Senior Lecturer in Psychiatry, Birmingham University*

Medical Applications of the Behavioural Sciences. Edited by JONATHAN J. BRAUNSTEIN and RICHARD P. TOISTER. London: Year Book Medical. 1983. Pp. 528. £24.75.

This large volume serves as an introduction to, what the editors call, "the art of medicine". Their argument is the well-established one that full consideration should be given to the biological, psychological, social and cultural factors affecting the patient's presentation when making a diagnosis or formulating treatment plans. Unfortunately, like

so many texts on "whole person" medicine, this book fails to demonstrate how these factors might be worked with in practice. Each of the twenty four chapters is a discrete entity in its own right and, in the absence of editorial commentaries, the onus is on the reader to achieve a synthesis of the diverse concepts presented and to consider their clinical implications.

The text is divided into five sections, the themes of which are biology and behaviour, psychological factors, social and cultural influences, the life cycle and doctor/patient issues. In their endeavour to provide a comprehensive review of the field, the editors have included everything from neural transmitters to ethnic influences on symptom formation. Laudable though such an ambitious project might be, a consequence is that relatively little space is devoted to such well-researched areas as chronic pain and biofeedback, while the developmental and social chapters contain much that is speculative. It is a matter of opinion as to whether the achievement of balance is more important than an accurate reflection of the state of scientific knowledge in each of the areas covered.

Like so many introductory text books, the majority of authors fail to acknowledge controversies and discrepancies in the fields they are reviewing. With few exceptions, classic studies from the sixties and seventies are reported faithfully, with no mention being made of more recent work which casts doubt on the validity or generality of these findings. Again it is debatable as to whether an introductory text should sacrifice accuracy in the interest of clarity. Perhaps the most misleading chapter is that on psychological theories which gives the impression that such concepts as the Oedipus complex and operant conditioning are the cornerstones of contemporary thinking about human behaviour.

Despite these criticisms, the book has much to recommend it if used judiciously. Medical students and Membership candidates will find it a highly readable introductory text and, in view of the excellent index, it is ideal for quick reference and revision purposes. However, in view of its piecemeal style, the text adds little to our understanding of the complex issues surrounding symptom formation, illness behaviour and the design of individually tailored treatment programmes. Yet despite failing in their primary objectives, the editors have nevertheless produced a collection of useful introductory chapters which will be read avidly, and hopefully cautiously, by both undergraduate and postgraduate medical students.

DOUGAL MACKAY, *District Psychologist, Barrow Hospital, Bristol*

Pharmacotherapy of Affective Disorders: Theory and Practice. Edited by W. G. DEWHURST and G. B. BAKER. Beckenham: Croom Helm. 1985. Pp. 628. £40.00.

Books devoted to the topic of affective disorders have burgeoned over the past decade, reflecting the increased biochemical and pharmacological interest in this area. *Pharmacotherapy of Affective Disorders* is a multi-authored work, written in a large part by contributors from Canada and the United States, although there are three

European writers. The problem of many books with diverse authorship is the difficulty of maintaining links between the separate chapters. This is not wholly avoided in this volume, but there is a coherence about the contributions which makes it easier to assimilate than many books of this type. It is divided into sections dealing with the theories of the origin, epidemiology and diagnosis of affective disorders, the biochemistry of anti-depressants and actions and pharmacology of these drugs. The contributions are nicely shared between clinical and basic science workers from known centres of excellence.

The book provides an excellent reference to the biochemistry of affective disorders with very extensive discussion of classificatory schemes. A number of chapters would have been rendered more comprehensible by a summary of the main issues raised. In many ways the authors are not opinionated enough—it would be of value to know their own views about the merits of the various mechanisms and theories that they are discussing. This partly mirrors the conservatism and the caution of the Canadian editors, but it would not be out of place to guide the reader by indicating the credence of the evidence presented. Although often more readable than many North American contributions to the literature, some of the chapters suffer from lack of tabulation and figuration and the word processed print increases the impression of congestion making for irritating reading in one sitting. This seems to be the penalty for rapid publication.

The calibre of the book is reflected in its authorship, which includes such names as Flor-Henry, D. L. Murphy, Feighner, Lapierre and Bland, with contributions from our own Stuart Montgomery and Brice Pitt. All researchers in this area should have access to this book; psychiatrists and neurochemists will each find information of value in its sixteen chapters, and it should be widely quoted in *Citations Index*.

STEPHEN P. TYRER, *Consultant Psychiatrist,
Royal Victoria Infirmary*

Recent Advances in Postpartum Psychiatric Disorders.

Edited by DAVID G. INWOOD. Washington: American Psychiatric Press. 1986. Pp. 127. \$12.00.

It was the publication of James A. Hamilton's monograph *Postpartum Psychiatric Disorders* which whetted my appetite for the topic more than twenty years ago. Since then, however, American involvement in research in non-psychotic postpartum disorders has been very small compared with that in the United Kingdom and Scandinavia.

This little book is therefore at least to be welcomed as an indication that the Americans are getting in on the act, though I found the contents rather disappointing. In the first chapter the editor's classification which includes 'adjustment disorder with depressed mood (also known as maternity blues)' with an onset from the first day to six weeks postpartum, unfortunately blurs the distinction between 'the blues' and post-natal depression and suggests that 'peaking' of blues symptoms takes place at about the tenth day, when research indicates that it occurs on days

4-5. Two chapters give accounts of research which really belong in journals where they would have "the benefit of review by the scientific community". This phrase is taken from the chapter on 'Biological Aspects' by Jan Campbell and George Winokur which I found the most useful in the book.

It is nice to see a contribution by James Hamilton on 'Therapeutic Management' though I think he is too protective of patients against psychiatric labels (to the point of wanting to spare them a proper psychiatric interview) and I don't know why he has reservations about the use of tricyclic anti-depressants in serious postpartum depression. A robust account of a Mother and Baby Unit in the Royal Hallamshire Hospital, Sheffield by Joan Sneddon and R. J. Kerry, is surprising in the context suggesting that there is no comparable American experience to draw upon. Finally, there is a claim by another Dr Inwood for a bigger role for the paediatrician in the management of the peripartum than most of us would expect.

Members of the Marcé Society may be interested in a copy of this appropriate, modestly priced book, otherwise it will not attract many readers on this side of the Atlantic.

BRICE PITT, *Professor, Community Mental Health Unit for the Elderly, St Charles Hospital, London*

The Elderly in Modern Society. 2nd edition. By ANTHEA TINKER. Harlow: Longman Group. 1984. Pp. 369. £6.95

A second edition of this valuable handbook three years after the first reflects the importance of the 15% of our population over the age of 65 and the range and speed of the changes which affect them.

The book is intended not only for students of social administration but for all who are concerned with the elderly. Its 350 odd pages are packed with information needed by the very wide variety of disciplines so concerned and off-hand I cannot think of a more comprehensive dossier of sociological data, between the covers of a modestly priced paperback.

There is something here for everyone; for the psycho-geriatrician, the demographic background and chapters on health, personal social services, community care, evaluation and assessment are particularly useful.

Developments since the first edition was written in 1981 are conveniently considered in a 70 page final chapter and this is followed by a fascinating scrap book of 53 'documents' including apt quotations from Mark Abrams and Alex Comfort; observations on the elderly who are old, cold or found dead; suggestions by the elderly themselves about how they could be helped; snippets from the Beveridge & Seebohm reports and a variety of interesting tables.

A third edition must be due very soon and I look forward to it. My only suggestion other than 'more of the same' is that a better perspective could be given on how old people fare in our society by providing some information about what happens in other countries.

BRICE PITT, *Professor in the Psychiatry of Old Age, St Charles' Hospital, London*

Presentations of Gender. By ROBERT J. STOLLER, London. Yale University Press. 1986. Pp. 219. £23.00.

The title of this book could be misleading. It does present a sensible explanation for the development of gender identity, but the whole theory could be summarised in one sentence. Extremely feminine boys are, according to Stoller, the logical outcome of excessive and prolonged closeness with the mother and abandonment by a distant passive father. The main contributions of this book are, however, not this but it's critical appraisal of the analytic methods of research, it's questioning of the validity of psychoanalysis as a science and it's challenging of some of the most basic concepts in analytic theory. In this book, Stoller refutes one of Freud's most central views, namely that developmental arrest is caused by conflicts at a critical stage of psychosexual development. Stoller argues convincingly that non-conflictual gratification, e.g. a blissful and enjoyable relationship between a son and his mother, can lead to the developmental arrest of this son's masculinity. He also challenges the view that perversion is not possible until one has passed through oedipal conflict, by presenting a case of erotic fetishism in a two and half year old boy, the first case to be reported in analytic literature. Even more upsetting to the analysts is his challenge of Freud's belief that the male sex is the more superior, and that masculinity is the natural original mode of gender identity. In Freud's view the child grows up to be a boy unless his masculinity is interrupted. Stoller argues that it is femininity which is primary and that to become a boy the child has to sacrifice his femininity and give up his gratifying bond with his mother. He supports this view by clinical material, cross-cultural observations and by the biological fact that the foetus does not develop maleness unless androgen is added.

Apart from its theoretical and intellectual value, the book has very little practical value unless the reader is a practising analyst. The author does argue, however, against the liberal use of sex change and suggests treating the causes of the disorder rather than complying with them by allowing the patient 'to remain maximally and permanently ill'. Many clinicians will agree with him, but there is only one catch: he does not tell us how to do that!

Apart from minor irritations like the numerous footnotes, qualifications and explanatory statements which distract more than clarify and the fact that the book is written in the first person as if it is an autobiography, this book makes fascinating reading.

WAGUIH R. GUIRGUIS, *Consultant Psychiatrist,
Psychosexual Clinic, St Clement's Hospital, Ipswich,*

The Lead Debate: The Environment, Toxicology, and Child Health. Edited by RICHARD LANSDOWN and WILLIAM YULE. Beckenham: Croom Helm. 1986. Pp. 286. £25.00 (hb), £14.95 (pb).

This book tells us the many uses of lead today round the world, the precautions in collecting storing and analysing samples for lead and the provisos in interpreting the answers, the lead in waters, foods, air, paint, dust and other corners of the human environment, and the human physiology of lead. This forms an extensive background for reviewing in the last 80 pages the various attempts to define what lead may do in modifying the intelligence, attainment and behaviour of children. It is a dispassionate account, intended for a wide non-specialist readership (who may find it heavy going at times), which lacks however one extra chapter to spell out the economic and political implications of imposing controls on lead use: the balancing out of the risks and advantages of one act or another.

Although the book is called *The Lead Debate* it is much more useful than simply as a late starter to cool some of the passion roused by lead in petrol. Nearly everything it says applies in principle to the uses of nuclear power and radioactivity, or to other metals such as zinc, manganese, tin, cadmium aluminium, or lithium, which may be biologically inert at one level, possibly beneficial or even essential at another level, and toxic at yet a third. It is therefore helpful reading for psychiatrists as trace metals come to the fore in neuropathology and even in treatment.

Scientifically two complex issues stand out, the physiological and the psychological. The natural environment contains lead in various forms, and it is natural for the human body to contain some in its tissues and to metabolise it. The excretion of lead, and even more its gastrointestinal absorption depend on the diet: eat more iron (or more calcium) and you take in less lead. Hence, straightaway the difficulty of defining at what point exposure to lead begins to matter. Matter for what? Psychological tests are notoriously crude and imprecise, even of intelligence let alone of attention, memory, or personality, and on the other hand brain metabolism can stand a good deal of distortion from drugs and vitamins before (rather suddenly) behavioural abnormality appears. At what point does lead intake affect sleep or appetite or sex-life or work output? We are back in one of the central problems of psychiatry, how to quantify mental states.

JOHN CRAMMER, *Reader Emeritus in Biological
Psychiatry, Institute of Psychiatry, London*

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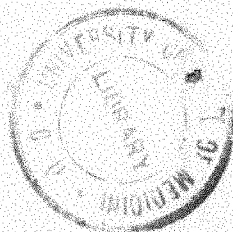


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The psychiatric hospital itself caters for a limited number of acute inpatient admissions, medium to long term rehabilitation of psychiatric patients including those with alcohol related problems and psychogeriatric patients, as well as a unit for the young brain damaged persons with behaviour problems. A Unit for intellectually handicapped persons is also sited on the hospital campus.

Toowoomba is a very attractive city with a population of 74,500, within two (2) hours driving time of Brisbane. It is renowned for its civic pride, schools, restaurants, and the annual Carnival of Flowers. It is the gateway to the rich farming area of the Darling Downs.

Classification is I-27 under the Public Service Award and salary at present is \$59,984 per annum. Usual Queensland Public Service conditions apply (e.g. compulsory superannuation, 5 weeks annual leave etc). A hospital residence together with milk, vegetables, fuel and light is available free of charge.

Financial assistance towards removal expenses may be provided to appointees from other States or overseas. Applicants must be registered or eligible for registration as a medical practitioner in the State of Queensland and must hold appropriate postgraduate qualifications in psychiatry. Administrative experience would be an advantage.

Further particulars may be obtained from Dr. P. J. Tucker, Assistant Director-General (Mental Health Services) and Director of Psychiatric Services, State Health Building, Brisbane on (07) 227-7537.

Applications should be made containing full particulars of name, address, telephone number, date of birth, marital status, qualifications, experience and present employment and furnishing copies of testimonials and the names of two referees.

Envelopes should be marked 'CONFIDENTIAL' and be forwarded to: Advertised Vacancy Officer, Department of Health, 14th Floor, State Health Building, 147-163 Charlotte Street, BRISBANE. Q. 4000. Closing Date: 5.00 pm Monday 22 December 1986.



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Adolf Meyer

Some Recollections and Impressions

J. WORTIS

Adolf Meyer was famous when I first met him in 1933. I was at that time a psychiatric resident at Bellevue Hospital, and Meyer was Psychiatrist-in-Chief at Johns Hopkins Hospital. He had been asked by the sponsors of a fellowship I was given to act as guide and supervisor, and had agreed. I met and corresponded with him throughout the tenure of that fellowship, to 1940, spending a year in his department in Baltimore in 1938-1939, and saw him irregularly thereafter.

His department at Johns Hopkins was the most prestigious in the country: everybody aspired to train there. He sat on nearly every committee of importance, on editorial boards, on important commissions, and his advice was sought for many key professorial appointments at leading universities. Though Meyer was well aware of his authority, his bearing was modest and simple. In his office he would not address you from a big chair behind his desk, but placed you in an easy chair, while he moved to an ordinary chair himself. He was short of stature, with thin hair and high forehead, wore a Van Dyke, spoke with a German-Swiss accent, and was very much the European professor.

He was not the kind of person who called you by your first name on brief acquaintanceship: we knew each other for 15 years, but he never addressed me by my first name. He was abstemious in his habits, started his day early, worked through the luncheon period and left a little early when his wife, who drove the family car, generally called for him. He regretted the separation of home and place of work. "When you live away from your work place," he remarked, "you lead a divided life." He did not smoke, and I do not think I ever saw him drink a cocktail, or indeed any wine. At large social gatherings it was generally Mrs Meyer who would maintain the conversation: Meyer was more passive, and would remain observantly quiet or responsive to attentions. He seldom engaged in casual conversation with members of the house staff, but would invite all of us in small groups in succession to have dinner at his home once a year.

His manner was always mild. He was a good listener, and when he spoke was never peremptory,

but cautious and moderate in his pronouncements, modifying or qualifying his phrases to produce a very characteristic circumlocution. His thinking also seemed to be many-faceted and complex rather than incisive or decisive, a quality that hampered his writing ability. He used to complain that he was educated in too many languages and could never attain literary proficiency in one, but I think the real difficulty was his tendency to excessively complex formulation.

Adolf Meyer had entered the American scene at the turn of the century at a crucial point in American psychiatry. There was on the one hand the vast custodial system for the psychotic in the state hospitals, and on the other hand the private practising neurologists (like Weir Mitchell) who were being called upon increasingly to deal with 'neuroses'. Aside from the state hospital system, psychiatry was privately practised by the neurologists. Scientific activity in psychiatry was mostly limited to descriptive or nosologic interests (Kraepelin) or to neuropathology. The new psychodynamic trend (Janet and Freud) was just beginning to emerge. At this juncture Meyer, based in the state hospital system, schooled and distinguished in neuroanatomy and neuropathology, and committed to the biographic individualized approach to case study, embodied the special combination of background and interests that the times required. The creation of a psychiatric chair for him at Johns Hopkins was innovative and set the pattern for similar developments in medical schools throughout the country.

He gathered a remarkably good staff around him. It was not only that Meyer picked good people: the most promising or best connected people also sought him out. Dr Esther Richards was perhaps the first woman to hold an important post in the Johns Hopkins faculty (the Phipps Clinic always had a high representation of women), Richter, Gantt, Diethelm, Muncie, Rennie, Kanner, Katzenelbogen and others all made their mark, and innumerable distinguished careers—both American and foreign—began there. For a long time there were always a few young residents from Britain in training there, and other foreigners came and went.

Though Meyer was by no means a system builder, the Phipps Clinic was a model of quietly efficient operation, achieved in an atmosphere of friendly relaxation. Everything had its place and everybody his or her role. Commands were not issued, but things were expected and had to be done, otherwise you would be embarrassingly conspicuous. The morning conference in the little library for example would start at nine: if you came late, everybody would see, and you would miss part of the presentation. Before nine Dr Meyer would regularly confer with his senior staff in his office, where news would be exchanged and decisions made: nothing of importance escaped attention. The non-professional staff was stable and loyal and stayed on for years. Senior staff similarly had little turnover. If there were rivalries and factions they were kept within discreet and polite limits, though Meyer once told me with a mischievous smile that whenever anybody left the staff he had a feeling of relief.

Meyer opposed the formal diagnostic preoccupations of the Kraepelinians, came to oppose the extreme and intense psychodynamic concerns of the psychoanalysts, and moved psychiatry a considerable distance from medicine. Though he maintained a broad all-inclusive frame of reference for psychobiology, in practice he discouraged and discounted the medical dimension. He refused to admit cases of general paresis to the Phipps Clinic because he considered them essentially medical problems. He maintained his love for neuroanatomy (every resident was expected to make a plasticine brain model under his tutelage) but considerations of neuroanatomy or neuropathology practically never arose in clinical conference, and I recall one case of brain tumour in an inpatient at the Phipps Clinic that was long undetected. The psychophysiological laboratories of Richter, Katzenelbogen, and Gantt intruded hardly at all into the clinical thinking at Phipps. As an example of Meyer's catholicity of interest they told a story of an announcement at the Phipps Clinic of a lecture at the hospital on 'The Chemistry of Doubt'. Nobody showed any surprise, until it was disclosed that it was all a typographical error: it should have read "The Chemistry of Gout".

With the widely publicized advent of insulin shock therapy, Meyer was quite seriously concerned, for he regarded this as a threat of resurgent medical emphasis in an area where he felt humane psychological interest should prevail. He accepted an invitation to speak at a meeting at the New York Academy of Medicine devoted to the sensational new treatment, but wrote me (1937):

There are two extremes in the attempts to play the savior role in psychiatry: work at the root—which is evidently *not* insulin work—and importations which have next to nothing specific to do with psychiatry but exploit the patient and resources through and for imported interests; and this is the case with insulin. I am always sorry to see the latter get on the top. Whenever it does, my interest wanes. I have allowed the paresis problem to pass into the domain of the lues department because paresis is a 'dirty' experiment of nature without localizing or any other control being possible. I am willing to leave it to the spirochaetist. And with insulin we deal with even more of an importation apt to divert the attention completely from the illness by absorbing the attention in the direction of something pharmaceutical. I am not belittling the interest and the experiment, but I hope you understand me fully.

It is too easy for non-psychiatrists to assume a dominant position and to become more or less antipsychiatric in the field. Therapy by the way of experimental pathology, along lines which have nothing to do with the disease process except as a starter and in the role of medical dominance acceptable to the patient and relatives and the profession will always have to prove itself. As soon as it becomes dominant by diverting the attention from what after all is the crux of it all, viz., the therapy at the root and in terms of life, it should only hold itself if success justifies the always disturbing disruption and usurpations by some narrow technician. I naturally shall not inject this note into the discussion of January 12. The meeting will anyhow be one *in partibus infidelibus*; i.e., governed by non-psychiatric interests and all I can expect to do is not to stir up the problem of psychiatry, but to balance what is sound in the work.

When insulin treatment was finally used at the Phipps Clinic, and the results were positive, he discouraged publication.

Meyer did bring into his conferences a broad medical culture and a strong moral commitment. His wide travels, his knowledge of languages and his studious habits gave him a breadth of information, much of it based on personal knowledge, that raised his discussions to a high level. At the same time it came as a pleasant shock to me, after my years at Bellevue Hospital, to hear Adolf Meyer insistently ask after every case presentation, "What are we going to do for this patient?" a query that was seldom heard amidst the prevailing psychodynamic interests in New York City.

Meyer's remarks at the daily morning conferences were often rambling and hard to understand. I sometimes felt like the little boy in "The Emperor's New Clothes" and was tempted to cry out that the emperor had nothing on, but deference to the aura of his presence was too pervasive to be dissipated. Actually the aura, in spite of its diffuseness, was beneficent; one could get the drift even when the details were obscure: the fog was luminous. The drift

generally involved an eminently humane effort to see a patient's symptoms and problems in the full context of his past life and present situation, to approach him sympathetically, to utilize his positive attitudes and assets and to teach, guide, assist in any way that would help the patient to return to his society and to his social goals.

Meyer's prominence and moral commitment often lead to cautious involvement in liberal causes. The involvement was always cautious because Meyer respected the established order and would do nothing that might appear to be disruptive. He was identified in those years, for example, with the effort to develop friendly relations with the struggling new socialist proletarian USSR, but at the same time was much disturbed by the attempts of some physicians at Bellevue to join a bargaining union. I am sure he did not think he was tainted by any racial bias, but he acquiesced to local custom: no Negro was ever admitted to the Phipps inpatient service, and Blacks held only menial jobs at the Clinic.

He had no interest in private practice, and was a strong believer in medical planning, in preventive medicine, in the public health model, and in government participation in all levels of health service. He wrote to me (1937):

The ideal of medicine is to make itself unnecessary. I wish it could be taken out of the economically competitive ranks. But in a power-crazy period the power-craze will be a deplorably strong item if not a necessity, whether under the sheepskin of "communism" or under the bald display of dictatorship, both living on might and hatred. It looks as if a new species of animal ought to sweep aside the present 'regimes'. The problem of self-maintenance of worker and work is difficult to solve since the sound features of 'bourgeoisie' were corrupted by greed and anti-greed and supergreed.

In an earlier letter to me (1934) he wrote:

Science has played too lone and high and mighty a game; so has industry and so have the humanities. They made puppets of man and are reaping their harvest. And who are 'they'? *We*, who are part of them and have to learn to sense the responsibility.

He preserved a polite but distant relationship with his fellow Swiss professor at Johns Hopkins, Henry Sigerist, whose book on Soviet Medicine was causing a stir. In spite of his general approval of government support of health services, he tried to dissuade me from joining an effort to set up a conference at Johns Hopkins in 1938 to promote national health insurance, because it was associated with Sigerist's name and was regarded by some as radical. He was

strongly opposed to the rising threat of Hitlerism, and devoted one morning's conference in 1938 to a discussion of Hitler. The occasion was a telegram he received from one of the Washington news agencies asking whether he thought Hitler was psychotic. The formulation of his response was typically Meyerian: The trouble was not that Hitler was diseased, but that he was healthy. And yet, Meyer went on to say, he had a disorder, and the cause of his disorder was his success: the treatment must therefore be his failure.

Another worthy cause for which Meyer's help was solicited (in 1938) involved the effort to rally support for a well known musician who had been sentenced to a very harsh long term of imprisonment because of a homosexual act. Meyer asked me to visit the prisoner and to prepare a report on the case, and was very helpful in securing his parole, and later his pardon. In asking me to report on the case, he wrote:

That some humans are homosexual may be completely beyond their control, either a matter of start (or heredity-determined spontaneity), or due to particular factors in habit-formation, special experiences such as seduction, or owing to narrowing factors in the sex opportunities and tendencies, or by venture and accident, and unwillingness to know and live up to the rules and laws of the body social. What then are the facts that bring the case to observation and judgment? The first concern to me in such a case is the nature of the charge and the facts at hand.

Without specification of the events I should not consider the mere accusation: solicitation and seduction and corruption? Public nuisance? Likely to have caused damage or to forebode damage to additional victims? In what way and to what extent is his case different from the partner? What is to be punished and what to be attained and how? For the protection of society? For treatment of his predicament? As a public deterrent (in general principle or as a clear demonstration of what the facts are and what the law aims to cover)? What does the letter of the law allow in the way of latitude? Were there ever any efforts to get treatment before or since the events and what would such 'treatment' aim at? What evidence is there on the question of dependability in the past and in case of release? It may be that your conversation and discussion of the case and the facts furnished give the picture needed. I lack the data. It may have been mutual masturbation with a minor? I should like to make clear that my letter will be based on an intimate knowledge of the facts of the case.

He had his own mild style of waging polemics. One adversary would mimic Meyer by saying, "Never mind the theory: let us have the facts. What you believe is theory; what I believe is fact." Meyer for his part said this person reminded him of Charlie Chaplin (to whom he did indeed have a certain resemblance). Of another unfriendly soul, he simply said, "He is not a man of strong personal

attachments." He wrote to me when I was seeing Freud in Vienna:

Freud interested me from his debut in the literature and while I felt from the beginning of my medical work keen on the inclusion of all of human nature in the scientific reality and objective world even just as I found it, the systematic organization he gave to it all was and is always stimulating and like a fabric of true art and life. I saw and heard him at Worcester in 1909 and called on him in 1923 and regret that the personal contacts could not have been more frequent and closer. I hope you will be a live link to whom I may turn when you get back to these shores. To cover such a life-work with the author while you allow yourself to vibrate true to yourself and to the atmosphere must be a great satisfaction.

When I complained of what I regarded as Freud's narrow dogmatism, he wrote:

Do not let yourself be misled by the natural concentration and limitation Professor Freud puts on himself. It is a mixture of temperament and wisdom and fate, which had best be accepted so that it gets its fullest expression. I sometimes envy that capacity; at any rate I see its great force, actually I am able to put myself in the position of the varieties of personalities and movements without any sense of resentment, although one regrets at times the lack of reciprocity. I should give a great deal to have or to have had your opportunity so as to be in a position to be thoroughly fair toward a force that crystallized some of the most fateful meanderings—perhaps too fatefully. I understand your dilemma, but trust that you can get a most interesting sense of the principles in pure culture without loss to your ultimate digestion of it all.

Because of his special prominence his chairmanship was renewed year by year after normal retirement age, but he finally began to fail, and his successor John Whitehead was appointed. Though Meyer was invited to retain his beloved neuroanatomy laboratory, he made little use of it, and was offended by what he regarded as Whitehead's excessive independence. When he was struck down by a cerebrovascular accident soon afterwards he made only partial recovery and never fully regained his faculties.

In June 1948 my wife and I had occasion to spend some time in Baltimore; I thought I would revisit Meyer. I knew that Meyer was an aged invalid; then in his 82nd year, and that this might be my last opportunity to see him. I sent a note ahead to which Mrs Meyer responded cordially asking us to join them at dinner. Since I recorded some notes in the evening after returning to our hotel, I can recall many details.

We found Mrs Meyer as vivacious and hospitable as ever; their daughter Julia and grandchild Kiki

were there as well as Dr Meyer's devoted former neurological laboratory assistant. We arrived a few minutes late and Mrs Meyer told us that Dr Meyer already had to take his seat at the table because it was so hard for him to get about. He sat with a napkin pinned to both of his lapels looking pathetically like an animated wax effigy bearing a certain resemblance to his former self.

Mrs Meyer took the initiative at the table conversation, with Dr Meyer from time to time making amiable and measured remarks, sometimes of questionable relevance and intelligibility. Very early in the evening he described his neurological disability as "a hemiplegia without hemiplegia." He went on to explain that he could grasp an object such as a glass quite well and could negotiate it, but could not control involuntary movements: his right hand jolted whenever he coughed. He said he found he had more dexterity if he twisted his head and body around to the left and he persisted in repeating these neurological observations about himself throughout the evening, later rising three times to demonstrate his prowess. He appeared to have no realization of any intellectual decline and even said explicitly that his conscious thinking processes were quite intact.

He displayed very little curiosity about anything I was doing, and spoke casually of his own isolation and of the disfavour in which the psychoanalysts seemed to hold him "because I was never analysed." "Grinker was here" he said, "sitting right here at the table. He is a somewhat colourful person who has given himself a name but I felt somehow that we were not in agreement." Indeed Meyer spoke in a friendly way of a whole series of associates and common acquaintances and did not say a harsh word about any of them. Of Whitehead he said with what sounded like a remote approximation to bitterness, "He has been here just twice." He remarked gratefully that "the neurological association seems to have remembered me," a reference to his recent election as an honorary member of the New York Neurological Society.

I recall his saying, "The analysts are always interested in putting things in their place. I have always wanted to put things together to see the relationship of things." He deplored the fact that no one seemed to be at hand to explore his neurological disability systematically. "Why don't you do it yourself?" I asked. "One cannot examine one's own unconscious," he replied, "You cannot study your own sleep, because you are asleep." With typical perseverance he came back to the phrase about examining one's own unconscious again and again.

He seemed to approve of my derogatory reference to private psychiatric practise as an unfortunate necessity, but when I spoke of the emerging interest in social psychiatry he said, "You have to be careful not to give it a political connotation." He stretched his hands forth as if arranging something on the table and added enigmatically, "There is communism, and yet I am not a communist."

Toward the end of the evening he said, "My library is in order, I have my study and my books where anyone who wishes to read may come. But," he went on, shrugging his shoulders, "who will want to come?" Later he said, "Death is an indeterminate sentence. It may come in 24 hours, though that is unlikely, but still," he repeated, "death is an indeterminate sentence."

This was an evening with the invalid Adolf Meyer some 20 months before he died. In retrospect little that was meaningful was said. There was not a single reference to any important current event nor was any important relationship with anybody described. There was in a sense something hollow about the occasion, but there was still something about Meyer which suggested an epoch, a breadth and a certain grandeur.

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His thoughts were still immersed in his past work and interests. Since Meyer had always been opposed to medical overemphasis in psychiatry it seemed ironic that his efforts to recall the past were so hampered and fragmented by his neurologic failings. He was aware only of the motor components, but the diffuse and elusive quality of his talk seemed like a caricature of his earlier tendency to complex and sometimes perplexing expression. "I have a hemiplegia" he had told me that evening, "but no alexia. I have no agraphia because I can write in a scribble though it is hard, but still I can write. I feel there is a difference between my sides, but what it is I do not know. Something is lacking, but I do not know what. A man cannot read his own unconscious."

He added, I thought perhaps significantly, that in 1933 following the Detroit psychoanalytic meeting he first noted a difference between his sides. I had the disturbing thought that a slight asphasic insult may have developed at that time which was glossed over respectfully through the years by the many devoted pupils and associates.

Subcortical Dementia

Neuropsychology, Neuropsychiatry, and Pathophysiology

J. L. CUMMINGS

Subcortical dementia refers to a clinical syndrome characterised by slowing of cognition, memory disturbances, difficulty with complex intellectual tasks such as strategy generation and problem solving, visuospatial abnormalities, and disturbances of mood and affect. The syndrome was first described by Kinnier Wilson, but further progress in development of the concept has occurred only within the past ten years. Subcortical dementia occurs in degenerative extrapyramidal disorders and has also been identified in inflammatory, infectious, and vascular conditions. Histologic, metabolic, and neurochemical investigations implicate dysfunction primarily of subcortical neurotransmitter systems and subcortical structures or subcortical-frontal connections in the genesis of the syndrome. Subcortical dementia contrasts neuropsychologically and anatomically with disorders such as dementia of the Alzheimer type that affect primarily the cerebral cortex. The clinical characteristics of subcortical dementia reflect the interruption of fundamental functions (motivation, mood, timing, arousal) mediated by phylogenetically and ontogenetically early maturing structures.

Dementia is an acquired persistent impairment in intellectual function involving several domains of mental abilities and resulting from brain dysfunction (Cummings & Benson, 1983). Dementia was once regarded as a unitary disorder of brain failure with homogeneous mental status alterations, but recent studies of intellectual changes in specific disease processes reveal that dementia has variable manifestations and that the nature of the cognitive and behavioural alterations can be correlated with the distribution and severity of brain pathology. Preliminary investigations have demonstrated two principal patterns of neuropsychological deterioration within the dementias: 1) a cortical pattern with intellectual decline including loss of such abilities as language, learning, perception, calculation, and praxis and manifested by aphasia, amnesia, agnosia, acalculia, and apraxia; and 2) a subcortical pattern resulting from disturbances of functions such as motivation, mood, attention, and arousal evidenced by psychomotor slowing, memory impairment, affective and emotional disorders, and difficulties with strategy formation and problem solving. Anatomically, the cortical pattern is produced by diseases involving primarily, but not exclusively, the association cortex of the cerebral hemispheres and the medial temporal lobes, whereas the subcortical pattern occurs in disorders with predominant

involvement of basal ganglia, thalamus, and brainstem structures (Albert *et al*, 1974; McHugh & Folstein, 1975; Albert, 1978; Cummings & Benson, 1983, 1984). Alzheimer's disease and Pick's disease are examples of cortical dementing processes; the dementia syndromes associated with the extrapyramidal disorders (Huntington's disease, Parkinson's disease, progressive supranuclear palsy, etc.) exemplify the subcortical pattern of intellectual impairment.

The relationship of dementia to cortical dysfunction is widely known and accepted, whereas the correlation between subcortical pathological involvement and dementia is more controversial. This review will present the available information regarding subcortical dementia and the contribution of subcortical structures to human intellectual and emotional function. A brief history of the idea of subcortical dementia will be presented first; followed by a discussion of the neuropsychological and neuropsychiatric characteristics of subcortical dementia; finally the pathology, pathophysiology, and possible mechanisms of subcortical dementia are described.

History of subcortical dementia

Subcortical dementia was first described by S. A. K. Wilson in 1912. In the course of describing the

disease that came to bear his name, Wilson commented on the "psychical symptoms" and noted that in contrast to "senile dementia" and dementia paralytica, his patients had no evidence of agnosia or apraxia, and memory and comprehension deficits were less severe. They manifested "narrowing of the mental horizon" manifested by a diminished capacity to store and retain mental impressions, and they evidenced "facility, docility, and childishness" with lack of insight and poor judgment. He observed that the symptoms closely resembled those occurring in Huntington's disease and Parkinson's disease. At autopsy, Wilson's patients had severe pathological changes in the lenticular nucleus and globus pallidus; the cerebral cortical structures were preserved. Thus, in addition to establishing the relationship between an extrapyramidal movement disorder and disease of the basal ganglia, Wilson must also be credited with the first description of a syndrome now recognised as subcortical dementia and with demonstration of its association with basal ganglionic dysfunction.

After Wilson's initial contribution linking intellectual impairment to basal ganglia disorders, observations regarding dementia in patients with thalamic and basal ganglionic diseases continued to accumulate, but no systematic formulation of the relationship was attempted for approximately 60 years. In nearly simultaneous publications, Albert and colleagues (1974) and McHugh & Folstein (1975) called attention to the subcortical origin of the dementia of progressive supranuclear palsy (PSP) and Huntington's disease, respectively. Albert *et al* (1974) noted that patients with PSP typically exhibited forgetfulness, slowness of thought processes, alterations of mood and personality, and impaired ability to manipulate acquired knowledge. They emphasised the absence of cerebral cortical involvement in PSP and observed that similar behavioural alterations had been described in patients with other subcortical degenerative processes such as thalamic degeneration, olivopontocerebellar atrophy, and Parkinson's disease and that all these disorders failed to produce the aphasic, agnostic, and apractic deficits characteristic of dementias associated with cortical dysfunction. Similarly, McHugh & Folstein (1975) described abnormalities of problem solving, insight, judgement, abstraction, attention, concentration, and motivation in patients with Huntington's disease. They contrasted these deficits with the aphasia, alexia, apraxia, and agnosia of Alzheimer's disease and with the amnesia of the Wernicke-Korsakoff syndrome. Noting the prominence of subcortical degeneration in Huntington's disease and the relatively limited and variable cortical involvement, they suggested that

the mental changes were a product of the subcortical pathological alterations.

In the past decade, the seminal observations of Albert *et al*, (1974) and McHugh & Folstein (1975) have been supported and extended. Among the extrapyramidal syndromes, subcortical dementia has been found in Parkinson's disease (Albert, 1978), idiopathic basal ganglia calcification (Cummings *et al*, 1983), and spinocerebellar degenerations (Hart *et al*, 1985) as well as PSP, Huntington's disease, and Wilson's disease. Thalamic degeneration, likewise, produces a similar pattern of intellectual deterioration (Stern, 1939; Schulman, 1957; Katz *et al*, 1984). Subcortical dementia has also been identified in non-degenerative disorders: infarction of the basal ganglia and thalamus produces the characteristic mental status syndrome (Cummings & Benson, 1983; Guberman & Stuss, 1983; Graff-Radford *et al*, 1984), and similar behavioural changes have been observed in patients with granulomatous and post-encephalitic involvement of the same region (Pilleri *et al*, 1981; Hier *et al*, 1983). Among metabolic conditions, the subcortical pattern of dementia has been identified in parathyroid dysfunction where basal ganglionic calcification is often prominent (Bachman & Albert, 1984). This aetiologically heterogeneous group of disorders shares a similar pattern of intellectual compromise and a common preferential involvement of subcortical grey matter structures.

Characteristics of subcortical dementia

Neuropsychological and neurobehavioural aspects

Dysfunction of subcortical structures results in abnormalities in speed of information processing, attention and concentration, memory, word list generation, abstraction and categorisation, judgement, problem resolution, strategy formulation, and visuospatial abilities. The dementia syndromes of Parkinson's disease and Huntington's disease have been studied most extensively and will be cited to provide examples of these deficits.

Speed of cognition

Slowness of central processing is a ubiquitous abnormality that contributes to many of the deficits of subcortical dementia. Originally considered a reflection of retardation of movement (bradykinesia), recent studies have shown a slowness of information processing (bradyphrenia) in excess of the motor system abnormalities. Evarts *et al* (1981) compared parkinsonian patients with aged controls

matched for severity of motoric slowing and found that the Parkinson's disease patients had significantly slower reaction times indicative of slowed central operations. Wilson *et al* (1980) demonstrated that the speed of memory scanning is increased in elderly Parkinson's disease patients compared to elderly control subjects, and Horne (1971) showed that post-thalamotomy Parkinson's disease patients had normal reaction times for tasks with no response delay but were significantly slower than control subjects when the tests were made more complex by requiring a delay in response. Rafal *et al* (1984) failed to demonstrate a retardation of central processing in their patients, but they found that dopamine treatment improved performance on two complex reaction time tasks and did not facilitate simple reaction times, suggesting that abnormalities of dopaminergic function were contributing to response latencies of the more complicated procedures. Stern and colleagues (1984) found a significant relationship between reaction times and abnormalities of norepinephrine metabolism in Parkinson's disease patients.

Slowing, unrelated to motor disturbances, has also been noted in patients with Huntington's disease and PSP (Rafal & Grimm, 1981; Brouwers *et al*, 1984).

Attention and concentration

Attention, concentration, and vigilance may also be disturbed by dysfunction of subcortical mechanisms. Parkinson's disease patients may manifest abnormalities on tests of concentration including digit span, digit symbol, trail making tests, and serial cancellation tasks (Reitan & Boll, 1971; Pirozzolo *et al*, 1982), and patients with left-unilateral Parkinsonism exhibit disturbances of visual attention and cancellation (Villardita *et al*, 1983). McHugh & Folstein (1975) emphasized that Huntington's disease patients had abnormalities of attention and concentration that contributed to their memory and cognitive deficits.

Memory

Memory disturbances are a cardinal feature of subcortical dementia but have been only partially characterised and contrasted with memory abnormalities in cortical dementing illnesses. While remaining oriented and performing adequately on simple memory tests, Parkinson's disease patients explored with more detailed testing manifest abnormalities in visual memory, learning of superspan word lists, logical memory, paired associate learning, tactile memory, and recall of remote personal and

sociopolitical information (Warburton, 1967; Reitan & Boll, 1971; Pirozzolo *et al*, 1982; Freedman *et al*, 1984; Globus *et al*, 1985). The deficits in recall of new information are partially aided by cues and by choice recognition manoeuvres suggesting a greater deficit in recollection of stored information than of encoding new material (Tweedy *et al*, 1982). Impaired recall of remote information is equally severe for all decades of the patient's life; the temporal gradient characteristic of Korsakoff's syndrome is lacking (Freedman *et al*, 1984). A relationship between altered catecholamine function and memory impairment in Parkinson's disease is suggested by the correlation between memory performance and cerebrospinal fluid catecholamine metabolite ratio (Mann *et al*, 1983) and the observation that verbal memory performance improves following levodopa administration (Marsh *et al*, 1971; Halgin *et al*, 1977).

Memory disturbances in Huntington's disease resemble those of Parkinson's disease. Impaired performance in tests of recent memory are among the earliest detectable abnormalities of Huntington's disease patients, and analysis of the recall deficits suggests disturbances in both encoding and retrieval (Caine *et al*, 1977; Butters *et al*, 1978). Compared with Korsakoff's syndrome, patients with Huntington's disease show no improvement with reduced proactive interference but are significantly aided by using recognition as opposed to recall paradigms (Butters *et al*, 1976; Martone *et al*, 1984). Like Parkinson's disease patients, those with Huntington's disease have remote memory deficits involving all decades (Albert *et al*, 1981). There is a significant correlation between the severity of memory abnormalities and the extent of caudate atrophy seen on computerised tomographic (CT) scans of the head of Huntington's disease patients (Sax *et al*, 1983).

Memory abnormalities have not been as well studied in other subcortical dementing disorders, but slowed memory scanning is evident in patients with Friedreich's ataxia, and memory is impaired in PSP (Albert *et al*, 1974; Davis *et al*, 1985; Hart *et al*, 1985).

Visuospatial abilities

Visuospatial deficits not attributable to motor system abnormalities including visual discrimination, visual organisation, spatial orientation, drawing, angle perception, and rotational task disturbances have been identified in patients with Parkinson's disease (Mortimer *et al*, 1982; Pirozzolo *et al*, 1982; Boller *et al*, 1984; Globus *et al*, 1985). Parkinsonian patients also have difficulty judging vertical and horizontal

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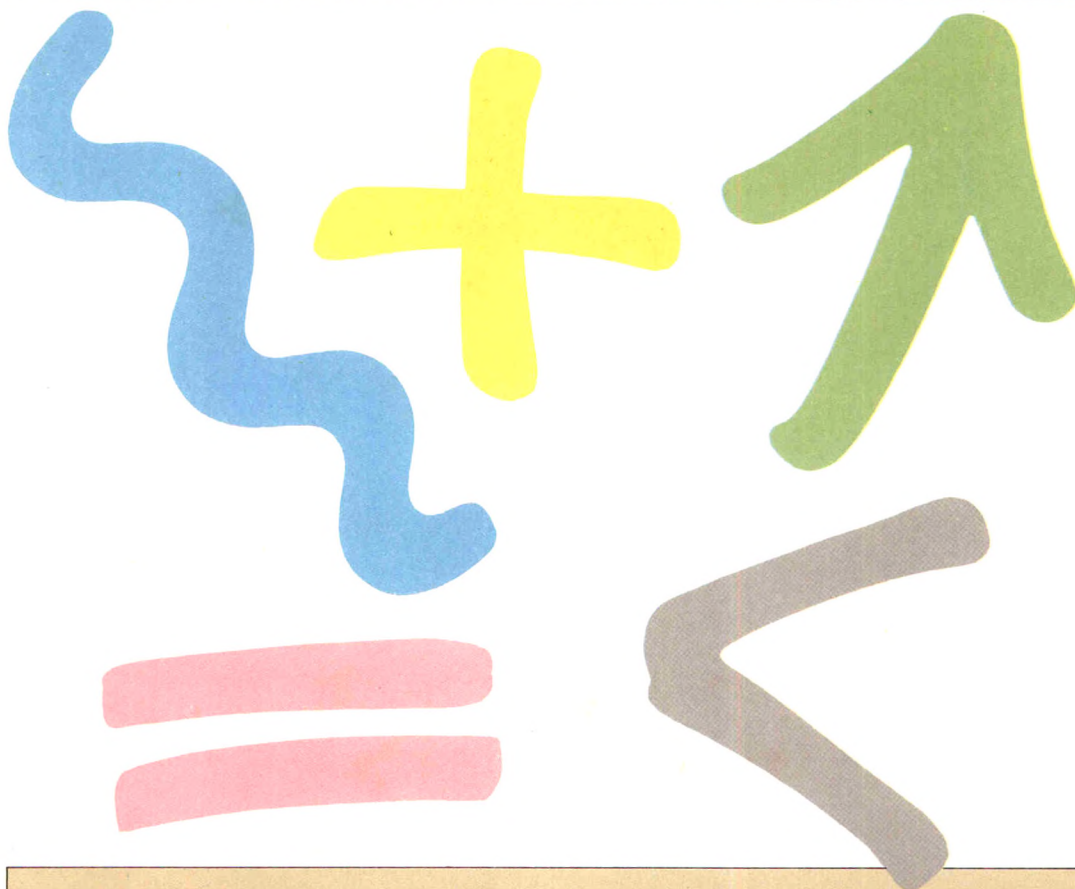
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Contra-indications Not recommended for overactive patients, or for those with Parkinsonism, severe arteriosclerosis, senile confusional, or advanced renal, hepatic, or cardiovascular disease, intolerance to other neuroleptics, pregnancy.

Further Information Overdosage should be treated;

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axes visually and with regard to their own posture when vision is obscured (Proctor *et al*, 1964; Danta & Hilton, 1975). Gainotti *et al* (1980) compared patients with Parkinson's disease, Huntington's disease, and Alzheimer's disease and found the latter to be significantly more impaired on tests requiring copying of designs. In another study comparing patients with cortical and subcortical dementias, Brouwers *et al* (1984) demonstrated that Alzheimer's disease patients had more difficulty with reproduction of constructional models, whereas Huntington's disease patients had greater impairment on a road map test. Kimura *et al* (1981) found that patients with PSP manifest abnormalities on tasks involving visual search or visual scanning abilities.

Cognition

Patients with Parkinson's disease also suffer from a variety of cognitive deficits. Category naming (verbal fluency, number of members of a specified category named in one minute) is diminished in many parkinsonian patients (Matison *et al*, 1982; Lees & Smith, 1983). They may also exhibit abnormalities on the similarities subtest of the Wechsler Adult Intelligence Scale (WAIS) and have difficulty shifting set on tasks requiring abstraction and categorisation (Talland & Schwab, 1964; Reitan & Ball, 1971; Lees & Smith, 1983; Globus *et al*, 1985). The latter deficit is partially reversible with levodopa therapy, supporting a relation with the underlying dopamine deficiency (Bowen *et al*, 1975).

Huntington's disease patients exhibit a similar pattern of cognitive deficits. Category naming is impaired early in the course of the disease, and subtests of the WAIS dependent on abstraction and concept manipulation are performed poorly (Boll *et al*, 1974; Butters *et al*, 1978; Josiassen *et al*, 1982).

The cognitive deficits of PSP similarly include poor abstraction, difficulty shifting sets, and poor manipulation of information (Rafal & Grimm, 1981; Maher *et al*, 1985). The cognitive deficits of the spinocerebellar degenerations have not been well studied but appear to resemble those of other subcortical dementias and lack the features indicative of cortical dysfunction (Carter & Sukavajana, 1956; Chandler & Debin, 1956; Cummings & Benson, 1983; Trauner, 1985). Similarly, the cognitive abnormalities of idiopathic basal ganglia calcification and Wilson's disease share the characteristics of subcortical dementia (Knehr & Bearn, 1956; Cummings *et al*, 1983).

Language

Compared with other areas of neuropsychological function, language is largely spared in the subcortical dementias. In Parkinson's disease, Huntington's disease, and PSP patients perform normally or manifest mild deficits in picture and object naming, but they lack the more severe disturbances of comprehension and the paraphasic abnormalities characteristic of the cortical dementias (Butters *et al*, 1978; Matison *et al*, 1982; Bayles & Tomoeda, 1983; Cummings *et al*, 1985; Globus *et al*, 1985; Maher *et al*, 1985). The few studies of Wilson's disease reveal relative preservation of vocabulary, whereas abstraction and conceptualisation abilities are compromised (Knehr & Bearn, 1956; Goldstein *et al*, 1968).

Summary and contrast with cortical dementias

Analysis of the neuropsychological profile of subcortical dementia allows the pattern of intellectual disturbances to be contrasted with the abnormalities manifested by patients with dementias involving predominantly the cerebral cortex (Table I). Subcortical dementias are characterized by psychomotor retardation, whereas the cortical dementias manifest normal psychomotor speed throughout most of the clinical course (Cummings & Benson, 1986). Memory disturbances are present in both cortical and subcortical dementias, but subcortical disorders have a prominent recall deficits and are benefited by clues and recognition tasks whereas the memory abnormalities in cortical dementias are a product of encoding failure and are aided little by recall facilitation strategies. Language abnormalities provide the most readily observable distinctions between cortical and subcortical dementias: subcortical conditions manifest no abnormalities or have mild impairments of confrontation naming and auditory comprehension, whereas cortical dementias evidence fluent paraphasic verbal output with severe anomia and moderate to severely impaired language comprehension (Cummings & Benson, 1983; Cummings *et al*, 1985). Visuospatial abilities are abnormal in both cortical and subcortical dementias. Alzheimer's disease patients have greater difficulty performing constructional tasks, and patients with subcortical dementias are more impaired on tests involving egocentric spatial operations such as map reading and judgement of vertical and horizontal axes (Proctor *et al*, 1964; Danta & Hilton, 1975; Boller *et al*, 1984; Brouwers *et al*, 1984). Abnormalities of abstraction and categorisation are present in both cortical and subcortical dementias

TABLE I
Contrasting features of cortical and subcortical dementias

<i>Characteristics</i>	<i>Subcortical dementia</i>	<i>Cortical dementia</i>
Neuropsychological features		
Severity	Mild to moderate throughout most of the course	More severe deficits earlier in the disease course
Speed of cognition	Slowed	Normal
Memory		
Short term	Recall partially facilitated by clues and recognition tasks	Encoding deficit; aided little by clues
Remote recall	No temporal gradient	In early stages, temporal gradient may be present
Language	Normal or mild anomia	Anomia, comprehension deficit, paraphasia
Visuospatial skills	Impaired; poor manipulation of egocentric space (map reading, judgement of horizontal and vertical)	Impaired; poor model copying
Cognition	Poor abstraction and categorization	Impaired early; late untestable
Neuropsychiatric features		
Personality	Apathy, irritability	Indifference, occasional disinhibition
Depression	Common	Uncommon
Mania	Infrequent	Absent
Psychosis	Common in some disorders, may have complex delusions	Common; simple delusions
Motor system		
Speech	Dysarthria	No dysarthria
Posture	Flexed or extended	Normal (flexed late)
Gait	Hypokinetic or hyperkinetic	Normal (until late)
Speed	Slow	Normal (until late)
Adventitious movements	Tremor, chorea, dystonia	None or myoclonus
Tone	Abnormal (hypotonic in choreic disorders; hypertonia in parkinsonian conditions)	Normal early; rigid or gegenhalten in later stages
Topography of pathologic changes	Most intense changes in striatum and thalamus	Frontal and temporo-parieto-occipital association cortex and hippocampus most involved
Neurochemical changes	<i>Huntington's disease:</i> decreased GABA, angiotensin converting enzyme, substance P, cholecystokinin-like immunoreactivity, and CAT <i>Parkinson's disease:</i> decreased dopamine, noradrenalin, serotonin, methionine-enkephalin, CAT, neurotensin, bombesin, cholecystokinin-immunoreactivity, GABA <i>PSP:</i> decreased dopamine, variable loss of CAT	<i>Alzheimer's disease:</i> decreased CAT, somatostatin, and norepinephrine
Aetiologic disorders	Degenerative extrapyramidal syndromes, subcortical infarctions; subcortical infectious, metabolic, and neoplastic conditions	Alzheimer's disease, Pick's disease

but are more severe in the former. Thus, psychomotor retardation is most characteristic of subcortical dementias and aphasia is most indicative of a cortical dementing disease; other neuropsychological abilities are affected by both cortical and subcortical conditions, but their effects within these domains are often qualitatively distinguishable.

Detection and characterisation of the subcortical dementias depends on utilising techniques sensitive to the intellectual consequences of subcortical dysfunction. Previous failures to establish differences between cortical and subcortical dementia utilised simple mental status questionnaires insensitive to the distinguishing features of the different patterns of intellectual deterioration (Mayeux *et al*, 1983). Future investigations of the intellectual deficits of subcortical dementias should focus on the differences between individual subcortical disorders (e.g. Parkinson's disease vs Huntington's disease), the relationships among the individual components of the dementia syndromes (e.g. association of the memory, visuospatial, and cognitive abnormalities), and comparative studies of cortical and subcortical dementias.

Neuropsychiatric aspects

In addition to intellectual impairments, abnormalities of personality, mood, and reality testing are common in the subcortical dementias. Like the intellectual disturbances, the neuropsychiatric alterations are products of the subcortical anatomic and biochemical changes, providing insight into the contributions of these regions to motivation, mood, and emotional function (Cummings, 1985a).

Personality alterations

Wilson (1912) called attention to the personality alterations associated with hepatolenticular degeneration when he observed that the patients appeared "docile" and "childish". Albert *et al* (1974) noted that patients with PSP were apathetic or irritable and had brief outbursts of rage. Similar changes have been observed in patients with idiopathic Parkinson's disease. Patients are less involved in family and social affairs, initiate fewer interpersonal interactions, and are more content with inactivity. Minnesota Multiphasic Personality Inventory (MMPI) profiles of Parkinson's disease patients have shown elevations on scales indicative of depression, hypochondriasis, and schizophrenia-like thought (Beardsley & Puletti, 1971; Hoehn *et al*, 1976).

The behaviour of Huntington's disease patients is

characterised by apathy, inertia, hostility, and irritability (Rosenbaum, 1941; Goodman *et al*, 1966; McHugh & Folstein, 1975; Mayeux, 1983). Caine & Shoulson (1983) studied 30 patients with Huntington's disease and found that 22 were apathetic and 11 lacked insight into the nature and consequences of their disability. Chronic dysthymia, anxiety, and intermittent explosive disorder were also observed. They noted that apathy and loss of insight were more frequent among patients in the more advanced stages of the illness. Dewhurst *et al* (1969) investigated 102 patients with Huntington's disease and found that, among the 68 patients for whom behavioural information was available, 44 suffered from anxiety or personality alterations in the prodromal period, and the frequency of neuropsychiatric alterations increased as the disease progressed. Thus, apathy and inertia are common features of subcortical disorders and a variety of conduct and personality changes have also been reported. Few quantitative studies of this aspect of behaviour have been undertaken, and there has been little attempt to establish correlations between the presence and degree of intellectual impairment and the behavioural alterations.

Depression

Depression has received the most systematic study of all non-cognitive alterations in subcortical disorders and was recognised by Albert *et al* (1974) and McHugh & Folstein (1975) to be a cardinal feature of subcortical dementia.

Some degree of depression has been identified in 37–89% of patients with idiopathic Parkinson's disease (Mindham, 1970; Brown & Wilson, 1972; Ceesia & Wanamaker, 1972; Robins, 1976; Horn, 1974; Mayeux *et al*, 1981). Moderate to severe depression occurs in 35–50% of Parkinson's disease patients, and depression is significantly more common than among patients with other types of neurological disorders or comparably disabling medical conditions. The depression correlates imperfectly with the degree of motor impairment, suggesting that it is not merely a reaction to physical disability but is biologically linked to the underlying neuropathological changes. Depression is reported to occur more frequently among patients with intellectual impairment than among those without evidence of dementia (Mayeux *et al*, 1981, 1983).

Depression is also common in patients with Huntington's disease. Dewhurst *et al* (1969) reported that of 102 patients 42 were depressed, and Caine & Shoulson (1983) found that six of 24 patients had major depressive episodes, with an additional six

suffering from dysthymic syndromes. Schoenfeld *et al* (1984) noted that suicide is approximately four times more common among patients with Huntington's disease than among the general population, and is eight times greater in the 50–69 year-old age group.

Depression has also been observed among patients with PSP, subcortical infarctions, Wilson's disease, and spinocerebellar degenerations but prevalence studies are not available (Davies, 1949; Albert *et al*, 1974; Trimble & Cummings, 1981; Jackson *et al*, 1983; Janati & Apple, 1984; Cummings 1985a,c). In contrast, severe depression is uncommon in cortical degenerative processes such as Alzheimer's disease (Cummings & Benson, 1986).

Mania

Mania is less common than other neuropsychiatric conditions associated with subcortical disorders but occurs too frequently to be considered a chance association. McHugh & Folstein (1975) reported that two of eight patients with Huntington's disease had manic episodes. Dewhurst *et al* (1969) found that of 102 patients with Huntington's disease, 14 were euphoric, two had hypomania, and five had grandiose delusions.

Mania has also been observed in Wilson's disease and post-encephalitic Parkinson's disease (Fairweather, 1947; Pandey *et al*, 1981), and Cummings & Mendez (1984) drew attention to the fact that a majority of focal lesions producing secondary mania are located subcortically in hypothalamic and perithalamic regions.

Psychosis

Psychosis manifested by the endorsement of delusional beliefs is closely correlated with subcortical dysfunction and has been observed with unusual frequency among patients with Huntington's disease, post-encephalitic Parkinson's disease, Wilson's disease, idiopathic basal ganglia calcification, and spinocerebellar degenerations, as well as subcortical vascular, traumatic, and neoplastic lesions (Cummings, 1985a,c). The psychosis may precede the dementia or may occur concomitantly with it. In the series of Huntington's disease patients reported by Dewhurst *et al* (1969), 50% had delusions at the time of admission to hospital. Six of 30 Huntington's disease patients reported by Caine & Shoulson (1983) had schizophrenia-like, paranoid, or atypical psychoses. Of 46 patients reviewed by Rosenbaum (1941), 38 were delusional for some period during the course of this illness.

Thus, when the entire course of the disease is taken into account, it appears that a majority of patients with Huntington's disease will manifest psychotic ideation.

Psychosis is uncommon in idiopathic parkinsonism except when induced by anticholinergic or dopaminergic agents but was reported in 17–57% of patients with post-encephalitic Parkinson's disease (Fairweather, 1947; Mindham, 1970; Celesia & Wanamaker, 1972; Crow *et al*, 1976). Likewise, approximately 50% of all reported patients with idiopathic basal ganglia calcification have a schizophrenia-like psychosis (Cummings *et al*, 1983), and psychosis has also been reported among patients with Wilson's disease, spinocerebellar degenerations, and subcortical neoplasms and infarctions (Jackson & Immerman, 1919; Davies, 1949; Shepherd, 1955; Malamud, 1967; Keddies, 1969; Trimble & Cummings, 1981; Cummings 1985b,c). Cummings (1985b), in a comparative study of delusions in a variety of neurological disorders, found that patients with cortical dementing illnesses (Alzheimer's disease) had more severe dementias and manifested simple paranoid misbeliefs, whereas patients with subcortical dementias had less severe intellectual alterations and tended to have complex, highly structured delusions.

Pathology and pathophysiology

Correlations between the phenomenology of subcortical dementia and the anatomic structures and biochemical systems involved have not been fully established, but preliminary information derived from pathoanatomic investigations allow a tentative formulation of the structural and biochemical alterations involved in these conditions.

Histopathologic alterations

The pathological anatomy of the extrapyramidal syndromes is well established. In Huntington's disease, the greatest pathological alterations involve the caudate nuclei where Golgi type II interneurons are preferentially lost. The putamen and thalamus manifest less severe alterations (Corseillis, 1976; Dom *et al*, 1976; Cummings & Benson, 1983).

Lewy bodies and neuronal loss are the histopathologic alterations characteristic of idiopathic Parkinson's disease. The changes are most intense in the substantia nigra, ventral tegmental area, and locus coeruleus; less intense or variable alterations occur in the nucleus basalis, caudate, putamen, globus pallidus, and cerebral cortex (Greenfield &

Bosanquet, 1953; Den Hartog Jager & Bethlem, 1960; Oppenheimer, 1976; Javoy-Agid & Agid, 1980; Whitehouse *et al.*, 1983).

Progressive supranuclear palsy has its greatest impact on the structures of the mesencephalic-diencephalic junction including the red nuclei, subthalamic nuclei, globus pallidus, thalamus, substantia nigra, and cerebellar nuclei (Steele *et al.*, 1969). In Wilson's disease, the putamen is most involved, and the caudate, subthalamic nuclei, red nuclei, and thalamus show less intense alterations (Wilson, 1912; Smith, 1976). The pathologic geography of the spinocerebellar degenerations is variable but routinely includes the cerebellum and brainstem, and in many cases alterations are detectable in the basal ganglia, substantia nigra, and thalamus (Greenfield, 1954; Oppenheimer, 1976). Patients with idiopathic calcification of the basal ganglia have dense perivascular calcific deposits in the caudate, putamen, globus pallidus, and cerebellar nuclei (Oppenheimer, 1976; Cummings *et al.*, 1983). Thalamic degeneration involves primarily the anterior and dorsal thalamic nuclei with variable involvement of putamen, cerebral white matter, cerebral cortex, and inferior olives (Stern, 1939; Schulman, 1957; Katz *et al.*, 1984). A similar distribution of lesions was present in one case of post-herpetic thalamic dementia (Pilleri *et al.*, 1981); whereas one patient with sarcoid-related subcortical dementia had prominent granuloma formation in the hypothalamus, fornix, and infundibulum (Hier *et al.*, 1983). Multi-infarct syndromes with subcortical dementia have had lesions concentrated in the basal ganglia and thalamus (Cummings & Benson, 1983; Graff-Radford *et al.*, 1984; Guberman & Stuss, 1983). In summary, the disorders reported to produce subcortical dementia routinely involve the striatal nuclei and/or thalamus with more variable involvement of subthalamic nuclei, substantia nigra, red nuclei, locus coeruleus, cerebellar nuclei, and cerebral cortex (Benson & Cummings, 1985).

The extent and nature of cortical involvement associated with dementia in Parkinson's disease is controversial. Hakim & Mathieson (1979) and Boller *et al.* (1980) found an increased number of senile plaques and neurofibrillary tangles in the cerebral cortex of demented Parkinson's disease patients and suggested that the dementia was secondary to co-occurring Alzheimer's disease. Ball (1984), however, found no increase in Alzheimer-type pathology among Parkinson's disease patients with dementia, and epidemiological studies suggest that the two diseases co-occur no more frequently than predicted by chance in an elderly population (Heston *et al.*,

1981). As outlined above, the nature of the neuropsychological alterations occurring in most patients with Parkinson's disease are unlike those noted in Alzheimer's disease. Progress towards the resolution of these discrepancies depends on thorough documentation of neuropsychological deficits in Parkinson's patients and correlation with quantitative analysis of cortical and subcortical pathologic and neurochemical changes.

Investigations of the anatomical foundations of dementia have emphasised the possible role of the nucleus basalis of Meynert (NBM). Whitehouse *et al.* (1981) documented atrophy of the NBM in Alzheimer's disease and suggested that loss of these cholinergic neurons and their cortical projections could account for the cholinergic deficit and the dementia of Alzheimer's disease. Later, Whitehouse *et al.* (1983) demonstrated that NBM is also atrophic in Parkinson's disease patients with dementia and suggested that the two types of dementia might have a similar pathophysiology. More recent studies show that NBM cell loss occurs in a number of neurological illnesses including Parkinson's disease patients that lack neuropathological features of Alzheimer's disease, Wernicke-Korsakoff syndrome, and PSP (Arendt *et al.*, 1983; Nakano & Hirano, 1984; Tagliavini *et al.*, 1984). These observations suggest that atrophy of NBM is a feature common to several dementing disorders, occurs in diseases with and without Alzheimer-type neuropathological changes, and has been identified in disorders with a variety of types of neurobehavioural alterations. Atrophy of NBM cannot as yet be correlated with any specific neuropsychological or neuropsychiatric syndrome. No atrophic changes are found in NBM in Huntington's disease or in post-encephalitic parkinsonism (Clark *et al.*, 1983; Whitehouse *et al.*, 1983).

Biochemical alterations

Intellectual ability is no less a product of the activity of neurotransmitter systems than of neuroanatomical structures. In some cases the effects of structural alterations may be mediated through changes in transmitter function; in others, the biochemical alterations and resultant mental status deficits may occur in the absence of overt anatomical pathology. Consideration of the consequences of loss of neurotransmitter function is particularly important in subcortical disorders because several major transmitter systems (e.g., dopamine, norepinephrine, acetylcholine, serotonin) have their cells of origin in subcortical nuclei and project via subcortical tracts to diencephalic and telencephalic regions (Anden *et al.*, 1966; Mesulam, 1983).

In Huntington's disease the principal abnormality is a 50–90% reduction in γ -aminobutyric acid (GABA) and related enzymes in the striatum (Perry *et al*, 1973; Bird & Iversen, 1974). Substantial reductions have also been found in angiotensin converting enzyme, substance P, methionine-enkephalin, and cholecystokinin-like immunoreactivity (McGeer & McGeer, 1976; Arregui *et al*, 1977; Emson *et al*, 1980a; Emson *et al*, 1980b). Choline acetyltransferase (CAT) is variably reduced and other enzymes associated with metabolism of acetylcholine and of catecholamines are normal (Perry *et al*, 1973; Bird & Iversen, 1974; McGeer & McGeer, 1976). The neurotransmitter and neuromodulator deficits are confined to subcortical structures; cortical enzyme activities are normal. Likewise, neurotransmitter receptor studies have shown decreased muscarinic cholinergic and GABA binding in neostriatum, increased pallidal GABA binding, and normal cortical receptor function (Enna *et al*, 1976; Wastek & Yamamura, 1978; Penny & Young, 1982; Whitehouse *et al*, 1985). Thus, in Huntington's disease there is a preferential loss of GABA and a number of neuromodulators and related enzymes from subcortical structures, whereas neurochemical analyses of cerebral cortex (including the frontal lobe) reveal no abnormalities.

In idiopathic Parkinson's disease, the type and distribution of neurotransmitter alterations have been extensively studied. The most prominent and consistent abnormalities involve the dopaminergic system including dopamine, homovanillic acid, tyrosine hydroxylase, and dopadecarboxylase (Hornykiewicz, 1973). The dopamine losses are not confined to the basal ganglia but involve the midbrain ventral tegmental area, hypothalamus, and limbic structures as well (Price *et al*, 1978; Javoy-Agid *et al*, 1981, 1984). Less pronounced losses of dopamine and related metabolites have also been identified in the cerebral cortex (Scatton, 1982). Noradrenalin, serotonin, methionine-enkephalin, neurotensin, bombesin, and cholecystokinin immunoreactivity are also reduced in subcortical structures (Studler *et al*, 1982; Taquet *et al*, 1982; Scatton *et al*, 1983; Bissette *et al*, 1985). GABA and related enzymes have been found to be reduced in most but not all studies of patients with Parkinson's disease. Efforts to relate neurobehavioural alterations to these chemical changes have revealed significant correlations between impaired intellectual performance and decrease norepinephrine metabolites in the CSF (Mann *et al*, 1983; Stern *et al*, 1984); neuropsychological abnormalities also correlate with increased CSF homovanillic acid and acetylcholinesterase levels (Direnfeld *et al*, 1984); and

depression is associated with decreased CSF 5-hydroxyindoleacetic acid, the principal metabolite of serotonin (Mayeux *et al*, 1984). A correlation has also been found between severity of dementia and reduction of neurotensin in the putamen as measured at autopsy (Bissette *et al*, 1985). Studies of cholinergic system enzymes have raised several questions. Some investigators found no difference in cholinergic enzyme levels between parkinsonian and control subjects (McGeer & McGeer, 1971, 1976; Lloyd *et al*, 1975). Perry and colleagues (1983) however, found reduced levels of CAT in the cortex of demented Parkinson's disease patients. The latter cases also had widespread senile plaques and neurofibrillary tangles and may have been suffering from dementia of the Alzheimer type. Similarly, Ruberg *et al* (1982) and Epelbaum *et al* (1983) found that demented Parkinson's disease patients had decreased cortical acetylcholinesterase, but these patients also had Alzheimer-type histopathology and low somatostatin levels characteristic of Alzheimer's disease. Such observations along with the histologic studies described above, suggest that a number of patients with Parkinson's disease have dementia of the Alzheimer type with typical Alzheimer-type histopathology and chemopathology, other groups have cortical cholinergic deficits without Alzheimer-type histopathological changes, or more restricted subcortical, pathological and neurochemical alterations (Gaspar & Gray, 1984). The frequency with which dementia syndromes are noted in Parkinson's disease patients and the differences between the neuropsychological deficits of Parkinson's disease and Alzheimer's disease suggest that dementia is not confined to parkinsonians with cortical abnormalities, although Alzheimer's disease may account for a majority of severe dementias occurring among parkinsonian patients.

The serendipitous discovery that 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), a synthetic analogue of meperidine, produces the rapid onset of an irreversible form of parkinsonism has provided new insights into the biochemical basis of subcortical dementia (Ballard *et al*, 1985): MPTP produces a nearly pure hypodopaminergic state by selective destruction of dopaminergic neurons in the substantia nigra (Davis *et al*, 1979; Burns *et al*, 1983). Neuropsychological assessment of patients with MPTP-induced Parkinson's disease patients reveals abnormalities of attention and cognition consistent with subcortical dementia (Stern & Langston, 1985). These observations indicate that a pure dopamine deficiency is sufficient to produce subcortical dementia.

TABLE II
Characteristics of instrumental and fundamental functions

<i>Characteristics</i>	<i>Instrumental functions</i>	<i>Fundamental functions</i>
Neuropsychological activities	Language, memory, perceptual recognition, praxis, calculation	Timing, arousal, attention, motor programming, motivation, mood
Neuropsychological deficits	Aphasia, amnesia, agnosia, apraxia, acalculia	Slowing, forgetfulness, dilapidation of cognition, depression, apathy
Associated type of dementia	Cortical	Subcortical
Principal grey matter structures	Multi-layered cortex (neopallial association cortex and hippocampus)	Deep nuclei (basal ganglia, thalamus, rostral brainstem)
White matter connections	Discrete well myelinated intra- and interhemispheric tracts	More diffuse, shorter, less well myelinated projections
Organisational composition	Parallel connection of functional units; lateral dominance well developed	Serial connection of structures with overlapping functions; poorly lateralised
Phylogeny	Recent evolutionary acquisition, most well developed in humans	More primitive, present in triune structure of reptile brain
Ontogeny	Incompletely developed at birth, myelination and dendritic growth continue through childhood	Functional at birth or soon after
Major neurotransmitters	Acetylcholine	Dopamine, norepinephrine, serotonin, GABA, acetylcholine

Recently, the biochemical disturbances associated with PSP have been identified. There is a marked nigrostriatal dopamine deficiency with preserved mesocortical and mesolimbic dopamine levels (Kish *et al*, 1985; Ruberg *et al*, 1985). Ruberg and colleagues (1985) found 50% reductions of CAT in subcortical structures and a 20% reduction in the frontal cortex, whereas Kish *et al* (1985) found no CAT deficiencies even in two patients with severe dementia. Noradrenalin, serotonin, GABA, and aspartic acid levels were normal (Kish *et al*, 1985). Thus, PSP and its concomitant dementia correlate best with reduced dopamine levels and show little relation to cortical cholinergic system deficits.

Other subcortical disorders have not been adequately characterised neurochemically, but sufficient information is available to suggest that hypokinetic-rigid syndromes predictably reflect striatal dopamine deficiency (Hornykiewicz, 1973), and the dementias accompanying these disorders can be attributed to the dopamine loss (Bachman & Albert, 1984).

Metabolic investigations

Recent investigations using positron emission tomography (PET) and cerebral blood flow (CBF)

techniques have added a new dimension to understanding the pathophysiology of subcortical dementia. Studies of Huntington's disease patients using PET reveal a distinctive pattern of metabolism characterised by decreased activity of caudate and putamen and normal cerebral metabolism (Kuhl *et al*, 1982). This pattern correlates well with the pathological and neurochemical findings in identifying impaired subcortical function and preserved cortical function. It contrasts with the cortical hypometabolism and relatively intact metabolic function of subcortical regions observed in Alzheimer's disease (Benson *et al*, 1983; Foster *et al*, 1984).

Studies of hemiparkinsonism with PET reveal decreased metabolism in the putamen and globus pallidus contralateral to the parkinsonian manifestations (Garnett *et al*, 1984; Martin *et al*, 1984). Studying bilateral parkinsonism, Kuhl *et al* (1984) found a generalised decrease in brain metabolism involving cortical and subcortical structures. Five of these patients were restudied 3-4 years later: one with severe dementia exhibited a PET pattern characteristic of Alzheimer's disease, three of the remaining patients had more modest dementias and showed an 18-40% decrease in cortical metabolic activity in a pattern unlike that of Alzheimer's disease. Cerebral cortical blood flow is also decreased

in Parkinson's disease, but the magnitude of CBF reduction shows no correlation with the presence or severity of intellectual deterioration (Lavy *et al.*, 1979; Bes *et al.*, 1983; Globus *et al.*, 1985). This lack of association suggests that the two functions are separately mediated, with subcortical dysfunction (not reflected in CBF studies) accounting for much of the dementia. Positron emission tomography studies in PSP have demonstrated diminished metabolism in the frontal lobes (D'Antona *et al.*, 1985). Metabolic studies of other subcortical disorders with dementia are not yet available; when completed and correlated with the type and extent of neuropsychological abnormality, such investigations should broaden understanding of the physiology of subcortical dysfunction and its intellectual consequences.

Mechanisms of subcortical dementia

Investigations of the neuropsychology, anatomy, and biochemistry of subcortical disorders have provided sufficient information on which to base a tentative explanation of how subcortical dysfunction results in a dementia syndrome. Subcortical structures are not merely relay nuclei linking the cerebral cortex with brainstem and spinal cord; rather they have important analytic and synthetic functions that modify, focus, and direct incoming and outgoing impulses. The thalamus mediates sensory, cognitive, and limbic activities through specific thalamocortical circuits and establishes cortical arousal through non-specific reticulothalamocortical projections (Walker, 1937; Jasper, 1949; Krieg, 1966). Arousal, activation, and attention are dependent on the integrity of these connections. The striatal nuclei receive afferents from all cerebral cortical areas, and there are particularly dense connections between the frontal lobe cortex and the caudate nucleus (Kemp & Powell, 1970; Graybiel & Ragsdale, 1979). These projections provide an anatomic basis for the similarities between subcortical dementias and of frontal lobe syndromes such as perseveration, impersistence, distractibility, difficulty shifting sets, abnormal verbal fluency, poor abstraction, impaired categorisation, and difficulty with strategy generation and problem solving (Johnson *et al.*, 1968). Terms such as 'frontal systems disorder', 'frontal-subcortical systems disorder', or 'frontal-subcortical dementia' are acceptable alternatives to 'subcortical dementia' and might more accurately reflect the realm of anatomic, metabolic, and neurochemical dysfunction found in this group of conditions.

Important links have also been discovered between the basal ganglia and the limbic system. Limbic

structures have largely unreciprocated projections to nearly all parts of the striatum creating a limbic-striatal system that utilises dopamine as an important neurotransmitter (Nauta & Domesick, 1978, 1981; Nauta, 1982; Iversen, 1984). These connections subserve motivation, mood, and aspects of reality testing, and disturbances of such functions contribute to the apathy, mood abnormalities, and psychoses observed in subcortical disorders.

Conceptually, cortical and subcortical abilities can be categorised as instrumental functions and fundamental functions, respectively (Table II) (Albert, 1978). Instrumental functions include language, praxis, perceptual recognition, memory, and calculation. These faculties depend on the integrity of focal cortical regions and are subject to interruption by localised cortical lesions or by disconnection of relevant cortical-to-cortical white matter tracts. Abnormalities of instrumental functions produce aphasia, apraxia, agnosia, amnesia, and acalculia—deficits associated with the cortical dementias. Available neuropsychological instruments are most sensitive to detection of these cortical disturbances. Instrumental abilities are the most highly evolved of human activities and depend on phylogenetically recent and ontogenetically late developing structures (Yakovlev, 1948). Organisationally, instrumental abilities involve discrete cortical areas with specialised functions connected by well myelinated intra- and interhemispheric white matter tracts. Acetylcholine plays an essential role in cortical neurotransmission.

Fundamental functions, on the other hand, include arousal, activation, attention, sequencing, motivation, and mood. These functions are much less discretely organised and involve subcortical nuclei (basal ganglia and thalamus) that interconnect widely with the cerebral cortex. The major projections are reciprocal connections with the frontal lobe and afferent connections from the limbic system. Organisationally, subcortical nuclei are connected by shorter white matter projections, which are not so well myelinated (Yakovlev, 1948). A variety of neurotransmitters are utilised including dopamine, serotonin, noradrenalin, GABA, acetylcholine, and others. Fundamental functions are crucial to survival and emerge early in phylogenetic and ontogenetic development. Dysfunction of these fundamental abilities produces the cardinal features of subcortical dementia including slowed information processing, dilapidation of memory and cognition, and disturbances of mood and motivation. Direct neuropsychological assessment of fundamental functions is difficult, and subcortical dysfunction must often be inferred from the

behaviour of the patient and the pattern of effects on the more accessible instrumental abilities. The movement abnormalities that frequently accompany the subcortical dementias (rigidity, chorea, athetosis, dystonia, bradykinesia) are not coincidental but reflect the dependence of the extrapyramidal motor system on anatomic structures, neurotransmitters, and neurophysiological activities that also mediate fundamental functions.

Further investigation is needed to refine, modify, and extend the concept of subcortical dementia. Sufficient information is available, however, to demonstrate that subcortical dementia is characterised by a clinical syndrome that is identifiable and can be distinguished from the syndrome of cortical dementia. The clinical features of subcortical dementia correlate with dysfunction of striatum and thalamus and of specific neurotransmitter systems. The characteristics of subcortical dementia reflect disruption of fundamental functions associated with a phylogenetically and ontogenetically less advanced level of neurological organisation. Fundamental functions are essential components of human mental abilities, and their compromise has grievous consequences for human thought and emotion.

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CAMDEX

A Standardised Instrument for the Diagnosis of Mental Disorder in the Elderly with Special Reference to the Early Detection of Dementia

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A new interview schedule for the diagnosis and measurement of dementia in the elderly is described. The schedule named the Cambridge Mental Disorders of the Elderly Examination (CAMDEX), consists of three main sections: (1) A structured clinical interview with the patient to obtain systematic information about the present state, past history and family history; (2) a range of objective cognitive tests which constitute a mini-neuropsychological battery; (3) a structured interview with a relative or other informant to obtain independent information about the respondent's present state, past history and family history. The CAMDEX is acceptable to patients, has a high inter-rater reliability and the cognitive section has been shown to have high sensitivity and specificity.

Introduction

Attempts have been made in the past to assess and characterise patients suffering from dementia in late and middle life. They have aimed at resolving three related, but relatively distinct, problems:

- (1) To diagnose dementias reliably either by the use of operational criteria such as those set down in DSM-III (American Psychiatric Association 1980) or by using standardised methods of interview and examination such as the Present State Examination (PSE) of Wing *et al* (1974) or its derivatives.
- (2) To develop valid and reliable measures of the severity and extent of cognitive impairment which constitutes a central feature in established cases of dementia.
- (3) To devise reliable means of rating behaviour and adaptation in everyday life that are as objective and precise as the situation permits and independent of clinical diagnosis or severity of dementia as judged from performance on standardised tests.

As a result of these enquiries, a number of scales useful for certain specific purposes have been developed. For example, standardised diagnostic interviews such as the Psychogeriatric Assessment Schedule (PAS) of Bergmann *et al* (1975) are capable of separating groups of patients into 'demented' and 'non-demented' or 'functional' groups. This makes them valuable for certain kinds of scientific enquiry: for example, clinical trials or epidemiological studies

in the community to provide a basis for social and health care planning. A large number of cognitive tests have come into existence which are of two broad types. Mental state examinations are capable of differentiating between the presence or absence of significant cognitive impairment while tests which derive from the psychometric tradition such as the Kendrick Battery (Kendrick, 1972; Kendrick & Moyes, 1979) permit an assessment of severity of cognitive impairment. There are also many useful behaviour rating scales which can detect problems in the activities of daily living. One of these scales, the widely used Dementia Scale of Blessed *et al* (1968) shows a good correlation with Alzheimer neuropathology, while the rating scale of Roberts & Caird (1976) has been found to correlate with computed tomography (CT) scans.

However, one source of difficulty and confusion has been the failure to differentiate between the type of information which each of these three approaches can validly yield. In consequence, a scale or interview developed to answer one type of question has been inappropriately used to answer another. For example, behavioural scales alone have been employed as measures of dementia or the results of cognitive tests have been used to establish a diagnosis. Such misconceptions are liable to introduce error and confusion, into research endeavours and clinical practice alike.

We believe that the three approaches to characterising the demented patient are complementary. All three are required to obtain a

complete picture of the disorder. The central objective of the endeavour to be described in this paper was to incorporate these different kinds of measure within a single compact, integrated instrument that has an acceptable measure of reliability and validity.

Developments in the diagnosis of dementia

Although operational criteria for the diagnosis of dementia such as those provided in DSM-III have improved stringency in diagnostic practice, they are somewhat arbitrary. Moreover, these criteria do not serve to identify mild dementia which presents a problem of central importance in this field (Henderson & Huppert, 1984).

There is more promise in the structured and semi-structured interview schedule from which diagnosis of organic mental disorder can be derived. The two best known schedules are the Present State Examination (PSE) of Wing *et al* (1974) and the Diagnostic Interview Schedule (DIS) of Robins *et al* (1981). However, neither of these instruments provide sufficient information regarding the history of onset and the character and progression of cognitive impairment.

The Geriatric Mental State Examination (GMS) of Copeland *et al* (1976) which is largely derived from the PSE and uses the same hierarchical approach to the problem of diagnosis, incorporates more tests of cognitive function. It has been used in the influential US-UK Diagnostic Project for comparing mental disorder in the elderly in New York and London and has now been incorporated, with some modification, into the Comprehensive Assessment and Referral Evaluation (CARE) of Gurland *et al* (1977). It differentiates between six principal diagnostic categories of old age mental disorder. They are: affective disorders, schizophrenia/paranoid states, organic psychoses, alcoholism, neuroses and personality disorders, and other diagnoses. And in two small studies of inter-rater reliability (Copeland *et al*, 1976), the diagnostic agreement was of the order of 85% on a sample of 20 and 73% on a sample of 22 patients.

These scales represent an advance in that they standardise the examination of the present mental state and render the findings relating to the present state more objective and replicable. However, the entire psychiatric examination required to make a diagnosis has not been standardised.

This is of particular importance in the characterisation and measurement of dementia. The relevant data cannot be extracted from standardised examinations formed on the present mental state alone. Such

findings can lead to error if they are not combined with data relating to the development of the disorder. Information regarding adaptation during development and basic personality traits and observations on the behaviour of the patient over a period of time are also needed to determine the extent of his pre-morbid traits and competence in negotiating everyday tasks. For such information, the judgments of a relative or friend or observations conducted over a period in hospital, are indispensable. Such behaviour will sometimes be deranged and disorganised before evidence suggestive of dementia comes to light during a structured interview. To cite an example, in the course of a cross-sectional examination, a differential diagnosis between dementia and states of subacute clouding of consciousness can be difficult or impossible. Yet given a few items of information regarding the history and development of the condition and the background of the individual, the diagnosis rarely presents difficulties.

Differential diagnosis

Both in respect of therapy and scientific investigation, the failure to differentiate between forms of dementia attributable to different aetiological processes would serve to obscure important findings. Multi-infarct dementia requires to be differentiated from Alzheimer's Disease and the Ischaemia Score (Hachinski *et al*, 1975) has made a start in relation to this. Means of refining the Hachinski score have been suggested (Roth, 1981) and they have been incorporated in the interview described here. Again, clouded and delirious states need to be separated from the dementias. Doubts about diagnosis can be resolved by information relating to the development of the disorder, the previous medical history and evidence regarding drug and alcohol abuse. The record of previous psychiatric illness and family history can help in the differentiation of pseudo-dementia from true dementia. The family record can also assist in the differentiation of Alzheimer's disease of 'early' and 'late' onset; an excessive prevalence of dementia in first degree relatives has been found more commonly in 'early' cases (Heston, 1984).

Measurement of the range and severity of cognitive impairment

A scale for the objective assessment of cognitive function is an essential component of any systematic schedule for the diagnosis and precise delineation of dementia. Subjective complaints of poor memory and word finding difficulty have been shown to be

more influenced by depressed mood than by objective evidence of cognitive impairment (e.g. Kahn *et al*, 1960). The cognitive section of the GMS and the CARE have therefore to be interpreted with caution, since they contain a large number of self-report items. Nor are standard mental status questionnaires, of which there are a large number, adequate for this purpose. Although they employ objective tests, they are usually concerned only with memory and orientation and designed for use with a markedly impaired population. A cognitive function test must sample a broad enough range of functions to meet diagnostic criteria for dementia: DSM-III for example, requires the demonstration of a generalised loss of cognitive functions including language, praxis, perception, abstract thinking and constructional ability as well as memory. Both the older and some of the more recent schedules fail to meet these requirements.

The Mini Mental State Examination (MMSE) of Folstein *et al* (1975) appears to be the best brief objective cognitive test currently in use as part of a diagnostic schedule (it has been incorporated into the DIS), but even the MMSE fails to assess perceptual ability and abstract thinking, and permits only a rudimentary assessment of most other functions. For this reason, we have developed our own cognitive test as part of the diagnostic schedule. However, given the popularity of the MMSE and its use in the influential survey of elderly community residents currently being undertaken by the National Institute of Aging in the USA, we included the Mini Mental State Examination in our cognitive section to enable direct comparisons with the results of other investigators.

A further requirement for a research diagnostic tool, is the reliable grading of severity of dementia. A number of rating scales have been developed in recent years notably the Clinical Dementia Rating (CDR) of Hughes *et al* (1982) and the Global Deterioration Scale (GDS) of Reisberg *et al* (1982). The CDR rates dementia along a five-point scale (none, questionable, mild, moderate, severe) on the basis of the person's performance in six areas of daily living: memory, orientation, judgement and problem solving, community affairs, home and hobbies, and personal care. The criteria are not easy to apply in practice, but questions also arise regarding their validity. Patients may receive the same rating (e.g. CDR 1 or mild dementia) whether or not they have additional difficulty with language or praxis. Yet in a 12-month follow-up study, a poorer prognosis has been reported for patients who had such difficulty at the initial assessment. Patients who progressed to moderate (CDR 2) or severe dementia (CDR 3)

performed significantly worse when originally assessed on all tests of language and praxis as well as on most memory tests (Berg *et al*, 1983). Findings such as these make it imperative that all areas of cognition should be taken into account when arriving at a severity rating. The Global Deterioration Scale (GDS) of Reisberg *et al* (1982) grades cognitive impairment along a seven-point scale, but the criteria rely very heavily on the severity of memory disorder. While we acknowledge that grading the severity of dementia is not an easy task, we propose to show that a clinical assessment of severity based on a range of cognitive tasks and activities of daily living can be made in a reliable way.

In its present form, the CAMDEX focuses on the diagnosis of dementia, with particular reference to its mild forms and to the identification of specific types of dementia. This task requires the differentiation of dementia from non-dementing conditions which may masquerade as dementia. Hence items relevant to other diagnoses must be included. Such items, particularly those relating to depression and paranoid states, are also needed to complete the clinical picture in dementia and are of therapeutic importance. At a later stage when an adequate body of CAMDEX data for patients with a wide range of psychiatric diagnoses has been collected it may be possible to broaden the CAMDEX into a comprehensive diagnostic instrument for psychiatric disorders of the elderly.

Objectives in the development of the present schedule

We have attempted in the development of the schedule which we have called the Cambridge Mental Disorders of the Elderly Examination (CAMDEX), to remedy the gaps in the existing standardised interviews and scales of measurement. We understand that since we began work on this project additional sections are now being added to the GMS to provide this information (Copeland, personal communication).

In developing the CAMDEX we have sought to create a diagnostic schedule with the following main ingredients.

- (1) A structured psychiatric interview with the respondent incorporating questions regarding the present mental disorder and the past history and family history.
- (2) The objective evaluation of a broad range of cognitive functions.
- (3) A standardised schedule for recording observations of the present mental state together with appearance and demeanor.

- (4) A structured interview with a relative or other informant able to provide independent information regarding the respondent's present state, any changes in personality and activities of daily living, past history, and family history.
- (5) A brief physical examination including neurological examination.
- (6) A record of a range of laboratory findings and present medication where applicable.

The CAMDEX schedule

In its present form this schedule comprises a number of sections which are outlined below. It starts with a sheet on which basic demographic data are recorded.

Section A

This section covers items of enquiry regarding the patient's present physical and mental state and in particular seeks symptoms relating to organic psychoses, depression and functional paranoid psychoses. Enquiries regarding past history and family history are also made. The questions posed in the final part of this section are prompted by the preliminary evidence suggesting a relationship in Alzheimer's disease on the one hand, and Down's syndrome and leukaemia on the other (Heston & Mastri, 1977). The section starts with three simple questions; the patient's name, age and date of birth. If the patient fails to provide satisfactory answers to two out of three questions the interviewer abandons Section A and moves on to Section B.

Section B

This consists of the cognitive examination. The 19 items which comprise the widely used Mini Mental State Examination (MMSE) of Folstein *et al* (1975) are incorporated into this section. However, a number of additional items have been included to compensate for weaknesses in the MMSE. First, the MMSE does not sample certain cognitive functions e.g. abstract thinking and perception, which are relevant to diagnosis and are included in DSM-III operational criteria for primary degenerative dementia. Second, many functions are assessed by the MMSE in insufficient detail. For example, memory is assessed only by the repetition and recall of three words; we have added items covering remote and recent memory and the recall and recognition of new information. (For full details of the cognitive examination see Huppert *et al*, 1985.) Thus the

cognitive section of the CAMDEX provides a wider coverage of cognitive functions than the MMSE, as well as more information about most functions. The test assesses orientation, language, memory, praxis, attention, abstract thinking, perception and calculation. Parts of each interview have been recorded so that various aspects of spontaneous speech and the content of language can be systematically assessed later.

Section C

This consists of the interviewer's observations on the patient's appearance, behaviour, mood, speech, mental slowing, activity, insight, thought processes and level of consciousness, and any bizarre behaviour. The section is completed at the end of the interview.

Section D

This comprises a simple physical examination including blood pressure, superficial and tendon reflexes, gait, defects of hearing or sight, tremor and Parkinsonian features to provide some of the information needed for differentiating between 'primary' and 'secondary' dementias.

Section E

The results of laboratory and radiological investigations are recorded in this section. Blood count, B₁₂ and folate, urea and electrolytes, liver function tests, VDRL, skull X-ray and CT scan, are recorded whenever available.

Section F

A record is set down of any medication currently being taken by the patient, and a note of the approximate period during which drugs have been taken.

Section G

This provides for any additional items of information obtained in the course of the interview. The purpose of this section is to amplify the picture of the patient already obtained by the structured questions. Much information in this section is spontaneously offered and of interest on this account alone in addition to the help it may afford to formulate a diagnosis at the end of the interview, particularly in atypical and difficult cases.

Section H

This comprises the structured interview with a relative, or a carer who knows the patient well. Any personality change, difficulty in functioning in everyday life or indications of cognitive difficulty noticed by the carer are noted. Items which permit the Newcastle Dementia Score (Blessed *et al*, 1968) to be scored are incorporated in this section. Questions referable to the presence or absence of depressive or paranoid phenomenology are included. Family history and past history are investigated with the aid of questions similar to those asked of the patient.

At the end of the interview, the interviewer makes a psychiatric diagnosis based on all relevant and available information according to operational diagnostic criteria. Diagnoses are assigned to one of 11 categories: normal, four categories of dementia (senile dementia of the Alzheimer type (SDAT), multi-infarct dementia (MID), mixed SDAT and MID; dementia secondary to other causes), two categories of clouding or delirium (clouded state, clouded state with dementia), depression, anxiety or phobic disorder, paranoid or paraphrenic illness and other psychiatric disorder. Patients are also graded for severity of dementia and severity of depression each on a five-point scale.

Duration of administration

The administration of the respondent's part of the interview can be completed in about 60 minutes. The informant's section takes about 20 minutes.

Test procedure

Subjects

Forty patients (33 female and 7 male) over the age of 65 were interviewed for the reliability study. The main diagnostic categories are given in Table I. A further 52 patients were interviewed (R.G.) so that clinical diagnostic scales could be developed and the cognitive tests used on a larger population. There were 61 females and 31 males in all. They were recruited mainly from inpatients and outpatients from the Department of Geriatric Medicine at Addenbrooke's Hospital, Cambridge, and the Department of Psychogeriatrics, Fulbourn Hospital, Cambridge. The normal group consisted of both geriatric patients and community residents, chiefly from warden-controlled sheltered accommodation.

TABLE I

Inter-rater reliability of CAMDEX for major diagnostic groups

<i>Interviewer diagnosis</i>	<i>Observer diagnosis</i>			
	<i>Normals</i>	<i>Dementia</i>	<i>Clouding</i>	<i>Depression</i>
Normal	9			
Dementia		22		
Clouding		1	4	
Depression	1		1	2

Method

The 40 patients participating in the study of inter-rater reliability were rated at the same time by two psychiatrists; one acting as interviewer for the whole interview and the other as observer. Both psychiatrists completed the CAMDEX independently. One psychiatrist (E.T.) was present at the interview of all patients and was the interviewer in half the interviews. Three other psychiatrists (H. H., S. V., R. G.) took part in the reliability study. Two of the four psychiatrists were trained in the UK and two in the USA. For each patient the ratings of the two psychiatrists were compared for all items and for each section of the CAMDEX. The purpose of the item analysis was to identify the questions with a high rate of disagreement between interviewers so that the source of the disagreement could be ascertained and the question modified or discarded from the next edition of the schedule.

The agreement between interviewers was measured by means of the phi (ϕ) coefficient (Guilford & Fructer, 1981). Although ϕ can vary from -1 to $+1$, only under certain conditions can the coefficient be as extreme as either of these limits. It is only when the two means are identical that the coefficient equals 1. The degree of difference between means is reflected in the degree of reduction in the value of the ϕ coefficient.

Results

Inter-rater reliability

Diagnostic agreement

There was good agreement between pairs of psychiatrists in the main diagnostic groupings (Table I). There was complete agreement on cases diagnosed by the interviewer as normal or demented. One of the five cases diagnosed by the interviewer as clouded was classed as demented by the observer, while of the four cases diagnosed by the interviewer as depressed, the observer agreed on two, but classed one as normal and one as demented.

The ϕ coefficient for diagnostic agreement was reduced to 0.63 when dementia was subdivided into four diagnostic categories: SDAT, MID, mixed SDAT and MID and dementia secondary to other causes. There was complete agreement between raters on the ten cases diagnosed by the interviewer as SDAT and one case of secondary dementia. Two of the eight cases diagnosed as MID were classed as

TABLE II
Inter-rater reliability of sections of CAMDEX

Section	ϕ Coefficients (2 × 2 agreement/ disagreement on individual items)		Pearson correlations of total scores for each section	
	Median	Range	r	P
A Interview with patient	0.94	1.0–0.28	0.99	0.000
B Cognitive examination	0.90	1.0–0.30	0.97	0.000
C Observations	0.83	1.0–0.30	0.81	0.000
H Interview with informant	0.91	1.0–0.56	0.90	0.000

mixed by the observer, and one of the three cases diagnosed as mixed was classed as SDAT by the observer.

Agreement on subsections of the scales

The ϕ coefficient was calculated for each item in Sections A, B, C and H of the CAMDEX. Table II presents the median and range of these values.

It will be seen that all the median coefficients for sections are high. The lowest value obtained was derived from the record of observed behaviour (Section C) independently set down by the two psychiatrists, but at 0.83 this section may be judged satisfactory and to show an acceptable degree of inter-observer agreement.

Some idea of the reliability of the items can be obtained from the range of ϕ coefficients estimated for the different items in each section. The item with the lowest reliability in Section A was the judgement by the psychiatrists as to whether physical disability accounted for the patients' inability to deal with household tasks (ϕ 0.28). The item with lowest reliability in the cognitive examination (Section B) concerned the naming of as many different animals as possible in a minute (ϕ 0.30). This wide disagreement was traced to the fact that one of the raters had excluded fish or birds. The poorest item in the observation section (Section C) was "Speech very slow. Pauses between the words" (ϕ 0.33). In such cases, scoring instructions have been made more explicit to increase reliability in the revised version.

The answers recorded from relations or carers (Section H) showed very good agreement between raters. The item with the lowest ϕ coefficient was "Does he/she have difficulty in knowing where he/she is or in recognizing you?" (ϕ 0.53). This is probably too terse and ambiguous for lay persons and has been made more specific in revision.

However, the proportion of items with low ϕ coefficients proved relatively small. The great majority of items reached an acceptably high inter-rater reliability.

Correlations between the total scores obtained by the two psychiatrists for each section are also shown in Table II. The correlations are all high and very significant statistically.

Cognitive performance

The results presented here were for the whole sample of 92 patients. The diagnoses in the total group were 17

normals, 26 with SDAT, 13 with MID, seven mixed SDAT and MID, three with dementia secondary to other conditions, five clouded-delirious (hereafter called clouded state), nine clouded states superimposed on dementia, and 12 depressed patients. For the three cases where interviewer and observer disagreed about the diagnosis, the interviewer's diagnosis was used. There was no significant difference between diagnostic groups in respect of sex or age of the patients, except that the depressed patients were significantly younger (71.9) than the normal (79.5), SDAT (79.3) and MID (80.1) groups.

Sensitivity and specificity

From the cognitive section (Section B) we obtain two overall measures of cognitive function: (a) total score on the Mini Mental State Examination (MMSE) of Folstein *et al* (1975) and (b) total score on The Cambridge Cognitive Examination (CAMCOG) which consists of 14 of the 19 MMSE items plus 43 items covering additional aspects of cognitive function. Maximum scores are 30 on the MMSE and 106 on the CAMCOG.

Table III shows the cognitive scores in the four major diagnostic groups. The normal and depressed groups did not differ significantly from each other on either test but performed markedly better than either the demented or clouded groups ($P < 0.001$) which did not differ from each other.

The sensitivity and specificity of the two tests for detecting organic mental impairment have been calculated. For the MMSE a cut-off value of 21/22 was recommended for those aged 60 or over (Anthony *et al*, 1982). Using this cut-off we obtain values of 96% sensitivity and 80% specificity for our population. This means that 96% of cases with an MMSE score of 21 or less had a clinical diagnosis of organic mental disorder (dementia or clouded state) while 80% of cases with an MMSE score of 22+ had been diagnosed as normal or depressed. Conversely, 20% of those scoring 22+ had a clinical diagnosis of organic mental disorder and hence were misclassified using this cut-off. We found that the optimal cut-off for our sample, was 23/24 which yielded 94% sensitivity and 85% specificity.

Sensitivity and specificity of the CAMCOG were also calculated. Examination of the distribution of scores showed the optimal cut-off to be 79/80. This yields 92% sensitivity and 96% specificity. Only one organically

TABLE III
Total scores for the Cambridge Cognitive Examination (CAMCOG) and the Mini-Mental State Examination (MMSE) in the major diagnostic groups

	Mean and range of CAMCOG scores (maximum = 106)		Mean and range of MMSE scores (maximum = 30)	
Normal (n = 17)	90.0	(72–101)	26.7	(22–29)
Demented (n = 49)	44.2	(8–82)	13.4	(1–25)
Clouded (n = 14)	52.3	(38–79)	15.3	(9–27)
Depressed (n = 12)	83.9	(68–98)	24.8	(14–30)

impaired patient was misclassified by this instrument compared with four who were misclassified by the MMSE.

Several patients obtained maximum or near maximum scores on the MMSE. Sixteen patients scored 27+, including five depressed and one clouded patient. Five patients scored 29+ (three normals, two depressed), the highest score (30) being obtained by a depressed patient. In contrast, only two patients, both normals obtained a CAMCOG score of 100+ while an additional two (one normal, one depressed) scored within 10 points of the maximum, and a further five (two normal, three depressed) scored within 20 points of the maximum. These findings demonstrate that CAMCOG can discriminate between individuals even at the high end of the ability range.

Severity of dementia and cognitive function

The relationship between cognitive performance and severity of dementia was examined by correlating both the dementia score (Blessed *et al*, 1968) and the clinical rating of severity of dementia with scores on the cognitive tests. For the group as a whole, all correlations were highly significant ($P < 0.000$). The dementia score correlated -0.70 with the CAMCOG and -0.66 with the MMSE. The clinical rating of severity of dementia correlated -0.78 with the CAMCOG and -0.76 with the MMSE. There was a correlation of 0.94 between the two cognitive tests and 0.68 between the dementia score and the clinical rating of severity.

The dementia score correlated significantly with each of the eight subscales of the Cambridge Cognitive Examination (orientation, language, memory, praxis, attention, abstract thinking, perception and calculation). The clinical rating of severity of dementia also correlated highly with the cognitive subscales ($P < 0.000$ for all comparisons).

Within the demented group as a whole ($n = 49$) there was very good agreement between cognitive performance and the clinician's estimate of severity of dementia. The correlation was -0.83 for the total score on the CAMCOG and -0.69 for the score on the MMSE. The correlation was highly significant for each of the eight cognitive subscales, the highest correlation being obtained for language (-0.77) and the lowest for attention (-0.40 , $P = 0.004$).

Subjective assessment of cognitive function

Cognitive function was reported subjectively by the patient in Section A of the CAMDEX and reported by the informant in Section H. We examined the relationship between this type of information and objective measures of cognitive performance. Three questions in Section A were concerned with the patient's self-assessment of memory; a general question about memory difficulty, a question about misplacing objects and a question about losing one's way in familiar surroundings. None of these correlated significantly for the group as a whole, with performance on the memory items or any other measures of cognitive performance. Questions about difficulty with concentration, slowing of speech or slowing of thoughts also failed to correlate significantly with cognitive performance. On the other hand, the responses of

informants to five questions about memory, concentration and muddled thinking, all correlated highly ($P < 0.001$) with total score on the CAMCOG and ($P < 0.01$) with seven of the eight cognitive subscales (orientation, language, memory, praxis, attention, abstract thinking, perception) while all failed to correlate with the calculation subscale and a measure of reading ability.

A question about word-finding difficulty was also asked of both the patient and the informant. Patient's self report did not correlate significantly with objective measures of naming or verbal fluency although it correlated significantly with measures of verbal memory ($P < 0.01$). The informant's evaluation of the patient's word-finding difficulty correlated significantly ($P < 0.01$) with both naming and fluency, and with each of the main cognitive subscales except memory.

In general it may be concluded that the patient's self report of cognitive function bears no relation to cognitive performance whereas the informant's judgment provides a reliable guide. Consistent with this is the absence of any significant correlations between patients' and informants' reports of the patient's of cognitive function.

Depressed mood and cognitive function

In addition to making a clinical diagnosis of depression and rating its severity, CAMDEX permits depressed mood to be assessed in all patients. This is done by; (a) self report on 20 items from Section A relating to the signs and symptoms of depression which were added together to provide a depression severity scale, and (b) the informant's response to the question, "Do you think he/she is depressed?". We examined the relationship between these measures and cognitive performance.

For the group as a whole, scores on the depression severity scale are significantly related to orientation and memory performance ($P < 0.01$) but not to any other measure of cognitive performance. The informant's assessment of depressed mood correlated significantly ($r = 0.30$, $P < 0.01$) with scores on the depression severity scale but not with any measure of cognitive function.

Responses to a key item in the depression severity scale ("Do you feel sad, depressed or miserable?") were found not to be related to cognitive performance, but correlated 0.64 ($P < 0.001$) with informant's assessment of depression. These findings indicate that the presence of the signs and symptoms of depression is related to impaired memory functioning but complaints about depressed mood are not.

Development of the clinical diagnostic scales

The number of patients in the SDAT, MID and depressed groups was sufficiently large to consider the development of a diagnostic scales from items in the CAMDEX. The first stage was to select items considered, on clinical grounds, to be of possible use in making differential diagnoses. The frequency distribution of these items was drawn up for all diagnostic groups recorded on the CAMDEX and those items which proved to differ in frequency between diagnostic groups were selected for the development of three diagnostic scales. The first intended

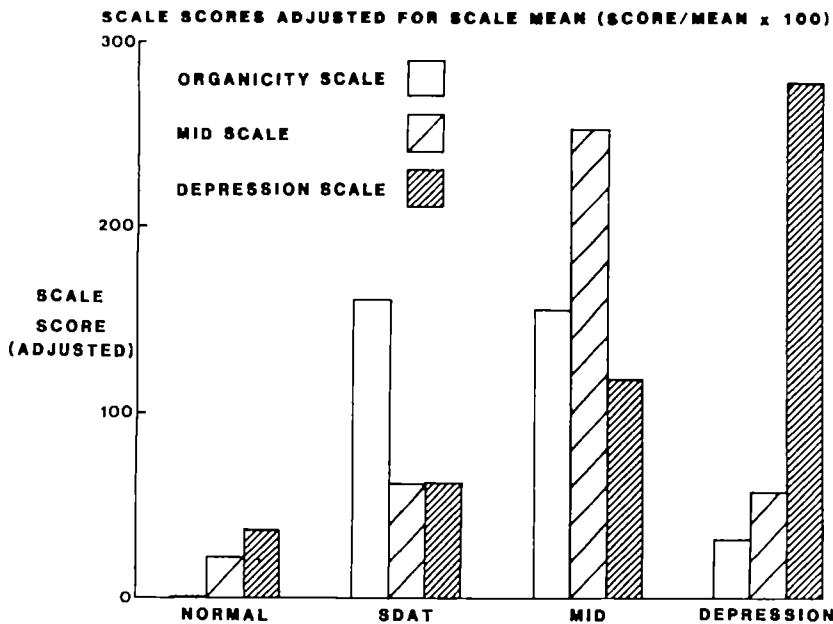


FIG. 1. Mean scores on diagnostic scales for four diagnostic groups. (Scores adjusted for scale score mean)

as a scale for SDAT consisted of 18 items and proved to be a scale of organicity in general. The second was a scale for MID which consisted of 12 items, and the third for depression consisting of 14 items. The scores on these subscales plotted by the diagnosis made on the basis of operational diagnostic criteria is illustrated in Fig. 1.

The cumulative frequency curves of the scores in each scale were plotted for SDAT, MID, depressed and normal groups, (Figs 2, 3, 4). These graphs were used to determine optimal cut-off points for diagnosis. The proportion of correctly classified patients using these optimal cut-off points is shown in Table IV.

DISTRIBUTION OF ORGANICITY SCORES - 4 DIAGNOSTIC GROUPS

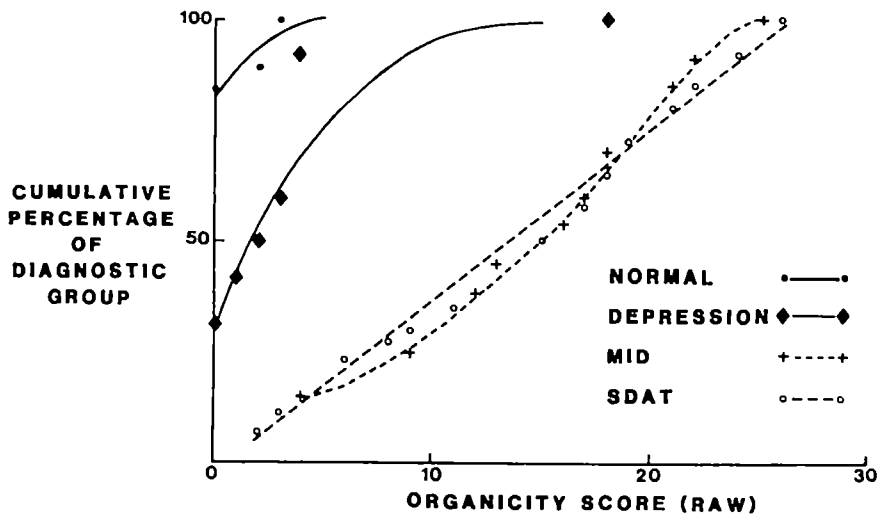


FIG. 2. Distribution of scores on organicity diagnostic scale for normal, depressed, SDAT and MID groups.

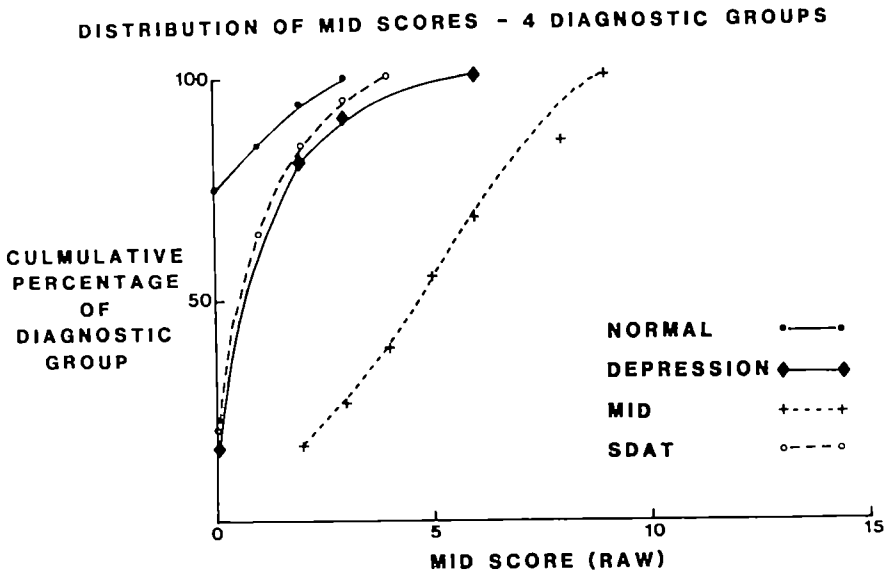


FIG. 3. Distribution of scores on MID diagnostic scale for normal, depressed, SDAT and MID groups.

The statistical significance of the diagnostic differentiation between the groups at these cut-off points was tested, using χ^2 tests and Fisher's exact test. Using a cut-off point of 4, the organicity scale differentiated between SDAT patients and normals ($P < 0.0001$) and depressed patients ($P < 0.0005$) but did not differentiate

between SDAT and MID. A cut-off point of 2 on the MID scale produced significant differences between MID cases and normals ($P < 0.01$), SDAT ($P < 0.0001$) and depression ($P < 0.001$). A cut-off point of 10 on the depression diagnostic scales produced significant differences between depressed patients and normals ($P < 0.0001$), SDAT

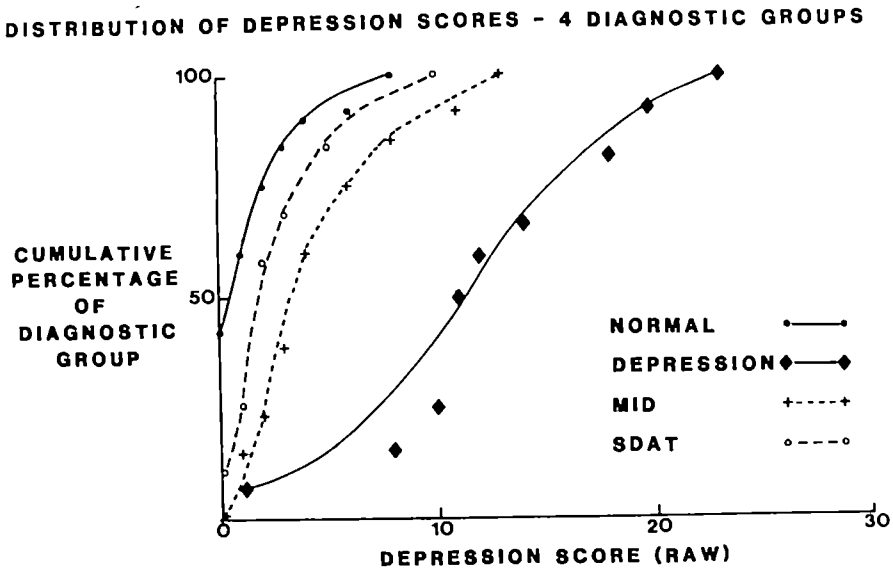


FIG. 4. Distribution of scores on depression diagnostic scale for normal, depressed, SDAT and MID groups.

TABLE IV
Classification of diagnoses by clinical diagnostic scales using approximate cut-off points

	Cut-off score	% Correctly classified			
		Normal	SDAT	MID	Depression
Organicity scale	4	100	85	85	92
MID scale	2	95	85	85	83
Depression scale	10	100	100	85	75

($P < 0.0001$) and MID ($P < 0.01$). It has to be borne in mind of course that there can be no complete differentiation since the conditions are not mutually exclusive in reality as is probably reflected in cases of depression and MID.

The independence of the three scales was tested by calculating their inter-correlations. These showed that there was a high degree of independence between the depression scale and the other two scales ($r = -0.15$ and 0.18 for organicity and MID scales respectively) although a significant relationship between the organicity and MID scales was found.

A test of reliability of the scales was made by an odd-even-split-half method. The reliability coefficients were corrected to full length and are shown below. A particularly high level of reliability is shown for the organicity scale (0.95). The other two scales for depression (0.90) and MID (0.77) seem quite acceptably reliable.

The problem of validity is difficult. A clinical diagnosis made under these circumstances cannot be fully independent of responses to items in the scale since the items are based on similar considerations to those on which clinical judgement is based. This is, of course, substantially why the items are included in the scale at the outset. Equally the expectations resulting from a clinical diagnosis probably create some bias in the evaluation of responses to the scale items, where the scale is administered by the diagnostician. Difficulties of this kind are incapable of complete resolution and validation becomes a progressive process of iterations, in which many varied sources of information must be used. In the present research the scales described are derived only from clinical data and, therefore, it is possible to turn to the cognitive section to look for validation. A positive correlation would show a relationship between a scale and some aspect of cognitive performance such as to indicate

TABLE V
Correlations between clinical diagnostic scales and factors derived from cognitive items

	Factor 1 (66.5%)	Factor 2 (9.5%)	Factor 3 (6.7%)	Total score on CAMCOG
Organicity	-0.61***	0.19	0.13	-0.63***
MID	-0.27**	-0.10	-0.01	-0.25*
Depression	0.21*	-0.12	0.02	0.22*

(Figures in brackets are variance explained by each factor)

* $P < 0.05$

** $P < 0.01$

*** $P < 0.001$

the scale was a rational measure. It is, of course, to be noted that the absence of a correlation simply would leave the validity question open since the test does not propose a relationship of any hypothetical kind.

Items from the cognitive section were subjected to a principal component analysis and the resulting factor scores of the first three factors were also correlated with the three scale scores. The pattern of results for Factor 1, a general factor, was strikingly similar to that obtained for the total score on CAMCOG as a whole (Table V). Both correlated highly with the organicity scale giving substantial support for its validity. There is also a smaller but statistically significant correlation with the MID and depression diagnostic scales.

The organicity and MID scales also correlated significantly with the psychiatric global rating of dementia though not with global ratings of depression. The reverse was true of the depression diagnostic scale which correlated highly with the clinical global rating of depression (Table VI).

TABLE VI
Correlation coefficients between clinical diagnostic scales and global ratings of severity

Clinical scale	Dementia	Depression
Organicity	0.73***	-0.11
MID	0.28**	0.15
Depression	-0.25	0.74***

** $P < 0.01$

*** $P < 0.0001$

Eight of the 18 items in the organicity diagnostic scale are items which also appear in the Newcastle Dementia Scale for severity of dementia (Blessed *et al*, 1968) and there is therefore a high correlation between the two scales (0.95) although the Blessed scale did not differentiate satisfactorily between diagnostic groups.

Discussion

In this paper we have described a diagnostic and assessment schedule that seeks to evaluate all parameters of present state, history, observation and measurement that may be relevant for the purpose of diagnosis and quantitative gradation of dementia.

Each section of the CAMDEX has been found to have an acceptably high measure of inter-observer reliability and this holds also for the great majority of individual items. The measure of agreement between different psychiatrists in respect of broad categories of diagnosis has also proved satisfactory and compares favourably with figures for inter-observer agreement in published schedules directed at the problem of diagnosis alone (Henderson *et al*, 1983). Data relating to test and re-test reliability are for the present limited; there are special problems attaching to the administration within a short period

of a long assessment schedule to elderly individuals. However, agreement between test and re-test within 3 weeks in a small number of cases is good.

The CAMCOG score in the cognitive section proved to have a high sensitivity and specificity in differentiation between organic and non-organic cases and was highly correlated with the Blessed Dementia Scale and the psychiatrist's clinical rating of severity.

Two principal advantages which the CAMCOG has over the widely used Mini Mental State Examination (Folstein *et al*, 1975) are (i) it covers a broader range of cognitive functions (ii) it detects mild degrees of cognitive impairment and (iii) it avoids ceiling effects.

Comparisons between information obtained from patients and their relatives indicates that relatives may be unaware of depressed mood but are far better at assessing cognitive impairment than are patients themselves.

The diagnostic scales derived from the sample show a relatively good classification rate; are reliable as shown by the split half reliability and have some evidence of validity, inferred from their correlations with global clinical ratings and with cognitive performance. These scales must now be tested on a new population so that their utility can be assessed. In the longer term it is planned to use these as the basis for computerised diagnosis, along the lines of the CATEGO classification (Wing *et al*, 1974) and the AGE CAT classification newly developed by Copeland and his colleagues for the GMS, in which organic psychosis is treated as a single category.

It is intended to investigate the validity of the CAMDEX schedule in a number of different ways, including long-term follow-up studies and correlation with pathological and biochemical measures. Examination of cognitive change in the follow-up studies will be of particular importance for the derivation of valid criteria in the diagnosis of early and mild dementia and depressive pseudo-dementia. Post mortem studies of the brain should also prove valuable to this end in addition to shedding light on the structural and neurochemical changes associated with the early stages in the development of dementia which are for the present largely unknown.

For the present the CAMDEX has proved itself of value as an instrumental aid in clinical diagnosis. When determined with the aid of CAMDEX, diagnoses have shown an acceptably high measure of inter-observer correlation. To develop the CAMDEX to the next stage will require additional data derived from the independent methods of validation which have been suggested in this section of the paper.

Acknowledgements

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Assessing and Treating Attention Deficit Disorder in Adolescents

The Clinical Application of a Single-Case Research Design

S. P. KUTCHER

Psychiatric assessment strategies that integrate research findings with daily practice are necessary and clinically useful. This paper uses the example of an attention deficit disorder adolescent to illustrate how double-blind placebo controlled and multi-dose adjustment designs can be successfully implemented in routine clinical practice. These designs are especially valuable in evaluating the therapeutic effect of methylphenidate because of biases that may lead to clinical misuse of this stimulant medication.

It has recently been appreciated that children with attention deficit disorder $\pm$ hyperactivity (ADD) do not outgrow their difficulties at puberty but may continue to experience psychiatric symptoms in adulthood (Menkes *et al*, 1967; Borland & Heckman, 1976; Wood *et al*, 1976; Weiss & Hechtman, 1979; Weiss *et al*, 1979; American Psychiatric Assoc., 1980; Weiss, 1983; Gauthier, 1984). These difficulties often lead to medical referral from parents, schools or correctional institutions and many attention deficit disorder adolescents may be seen by general psychiatrists. Although ADD is relatively common (Bloomingdale, 1984; Taylor, 1985), standard psychiatric textbooks (Hill *et al*, 1979; Kendell & Zealley, 1983) including one specifically concerned with adolescence (Evans, 1983) devote little space to the adolescent aspects of this disorder and low general awareness of adolescent ADD may lead to its underappreciation in this age group.

Significant numbers of childhood hyperactives have adolescent years characterised by continued motor restlessness, deficient problem solving skills, impulsive cognitive styles, delinquency, drug abuse, poor peer relationships, school failure and personality styles of immaturity, recklessness and impulsivity (Weiss *et al*, 1971; Minde *et al*, 1972; Cohen *et al*, 1972; Huessey *et al*, 1972; Stewart & Mendelson, 1973; Mendelson *et al*, 1974; Satterfield, 1981; Hechtman, 1984). Intrapsychically they tend to have low self esteem (Stewart & Mendelson, 1973; Mendelson *et al*, 1974; Weiss, 1983) and those adolescents with learning disabilities or conduct disorders have poorer outcome when concurrently

suffering from ADD (Ackerman *et al* 1977; Offord *et al*, 1979). Symptomatic ADD may prevent appropriate completion of adolescent maturational tasks (Erickson, 1950; 1968) leading to adult vocational, interpersonal and intrapsychic difficulties (Bellak, 1979) and successful clinical intervention during the adolescent years may have long-term benefits (Gittleman, 1983; Weiss, 1983; Cantwell, 1984).

Although long-term studies have yet to be reported, recent pharmacologic research has identified positive short-term effects of stimulants in the treatment of adolescents with ADD, and has not shown evidence of previously postulated excitatory switch effects at puberty (McKay *et al*, 1973; Safer & Allen, 1975; Lerer & Lerer, 1977; Barkley, 1977; White, 1981; Varley, 1983).

These results suggest a potentially helpful intervention strategy in the treatment of the ADD adolescent for whom the various psychotherapies have not been shown to be effective.

Pharmacological treatment studies, however, carried out in groups of patients with outcome data presented as population means offer few helpful guidelines for routine clinical practice. Individual physiologic, intrapsychic, social and interpersonal factors may all affect drug response and clinician bias towards stimulant therapy may influence decisions about instituting or evaluating treatment (Taylor, 1985). The use of the single-case research design (Kazdin, 1983) offers a potentially valuable tool in routine clinical assessment and treatment of ADD adolescents. It provides an objective measure of

stimulant effectiveness in the individual case, discouraging both overenthusiastic prescription or *a priori* rejection of drug therapy.

This paper uses the example of an adolescent assessed with this design to provide a practical guideline that could be readily applied in the routine clinical assessment of ADD adolescents, by adolescent specialists and general psychiatrists alike.

The Patient

D. S. a 14-year-old boy living at home with his mother and two younger brothers was admitted to a psychodynamic milieu orientated treatment centre. He had a history of thieving from cars, running with a gang, poor academic performance, anti-authority behaviour and poor peer relationships. School difficulties led to his placement in a 'Summerhill'-type residential school which had then for 2 years encouraged his free expression before finding his behaviour intolerable. The educational psychologist who referred him following his expulsion from that school described him as "a severely emotionally disturbed child locked in a constant battle with his mother". His mother stressed D. S.'s impulsiveness, anti-authority stance and his inability to learn from his mistakes.

D. S. was born of an unplanned pregnancy when his mother was 16 years of age. There were no natally associated difficulties and developmental milestones were reportedly normal. As a child, he was described as 'hard to discipline', 'full of excess energy', 'on the go', 'impulsive', 'accident-prone', and 'unable to wait his turn at games'. He had major school difficulties with reading and mathematics, paying attention in class and had failed grades.

His family life had been characterized by violent disagreements between his parents which culminated in their divorce when he was 8 years of age. His maternal grandfather had a history of excessive gambling and a maternal uncle was described as having low self-confidence and difficulties relating to people. His paternal grandfather was an alcoholic and his biological father had been jailed for assault and was prone to impulsive, angry, violent outbursts.

Method

Mental state

On examination, D. S. presented as a 'cocky lad' with a sense of bravado. He was easily engaged but was restless and fidgety. He spoke reasonably about what he thought his difficulties were: acting without thinking, fighting, not getting on with others, an overly quick temper, trouble

sitting still, trouble concentrating and feeling badly about himself (low self-esteem). He was not depressed nor thought-disordered. He thought little of the future and had no vocational plans, feeling that schooling was a "waste of time".

Physical

Physical examination showed a healthy adolescent; however, he had difficulties with rapid alternating movements, fine motor movements and balance. Drawing tests showed visuospatial difficulties. An EEG showed no seizure focus but excessive artifact secondary to his motor restlessness. Routine serology was within normal limits.

Educational

Educational assessment carried out by the Unit's teacher gave a reading age of 10.4 and a comprehension age of 11.4 on the Neale Reading Test. A Burt Spelling Test gave an age score of 10.0. Mathematics Operations were at the 9 year level and he was unable to apply basic arithmetic skills to problem solving.

Psychological

A Wechsler Intelligence Scale for Children (WISC) showed a performance IQ of 83 and a verbal of 86, although the testing psychologist felt these were underestimations secondary to 2 years of minimal academics. The coloured version of the Standard Progressive Matrices gave a score at the 11.5 year level and the Embedded Figures Test showed 20% correct responses to the house figure. A Rorschach was interpreted as within normal limits.

Pharmacological

Following a 2-day baseline assessment period a double-blind placebo cross-over trial (ABAB design) implemented, with the co-operation of the hospital pharmacologist, of methylphenidate 10 mg orally (0.16 mg/kg) three times daily was begun. The trial was maintained throughout periods of weekend leave at home.

D. S. was rated on an 18-item five-point scale (Appendix 1) derived from the Conners Scale (Conners, 1969) to which were added items related to the diagnostic criteria of Attention Deficit Disorder + Hyperactivity of DMS-III (1980). Ratings were independently made by nursing staff, physicians and teacher, and the scores for each day, were totalled and averaged for nurses, doctors and teachers separately, to determine the daily score by discipline.

Because this trial demonstrated a positive effect, a blind multi-dose trial of methylphenidate at 10 (0.6 mg/kg), 15 (0.9 mg/kg) or 20 (1.2 mg/kg) mg orally three times was then instituted. During this trial, both D. S. and his mother participated in the ratings. For each dose block (4 days per block) the daily scores were totalled and averaged to determine the block total score.

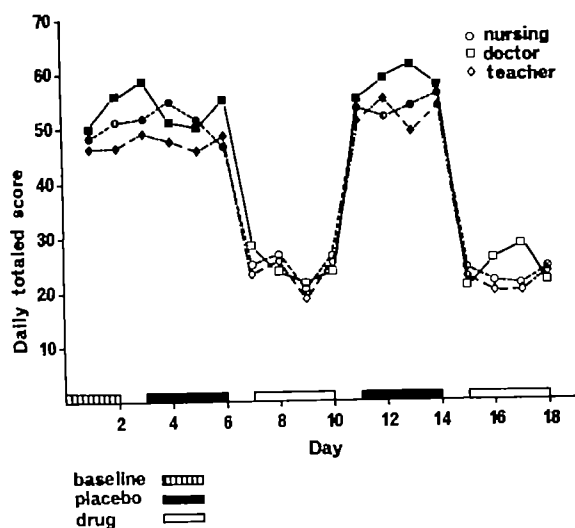


FIG. 1 ABAB, single case design, methylphenidate trial treatment response ($P < 0.01$).

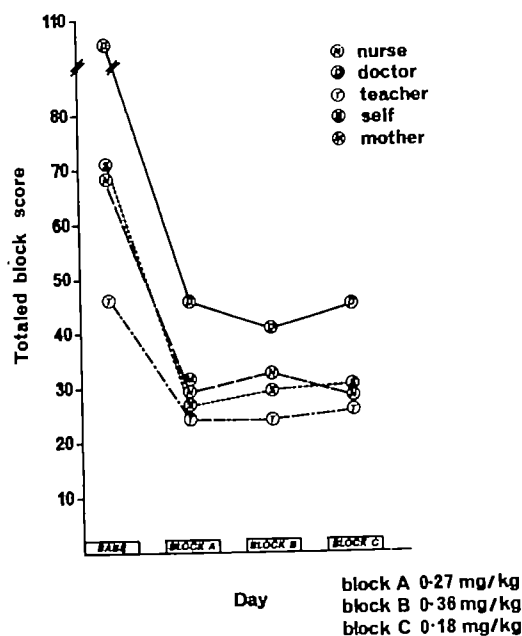


FIG. 2. Methylphenidate blind multi-dose trial: treatment response.

Results

For the double-blind placebo cross-over design (see Fig. 1), the baseline score gave a mean value of 50.0 ± 3.2 . The scores for the two medication periods were 24.1 ± 2.3 and 22.6 ± 2.4 . Each medication period was associated with a

greater than 50% symptom reduction in comparison with the two placebo periods and baseline assessment ($P < 0.02$, student's *t*-test).

The baseline score for the blind multi-dose trial gave a mean of 71.2 ± 22.6 (Fig. 2). The scores for the medication blocks were: block A (0.9 mg/kg) 30.2 ± 8.7 ; block B (1.2 mg/kg) 30.0 ± 7.7 and block C (0.6 mg/kg) 31.5 ± 8.4 . Although symptom scores of all three medication blocks differed by more than 50% from the non-treatment baseline, there were no significant differences amongst blocks ($P > 0.5$, student's *t*-test) (Fig. 2). Nursing and teacher observations regarding side-effects and D. S.'s self report noted the presence of drowsiness and lethargy during the block B (1.2 mg/kg) medication period.

Therapeutic intervention was tailored to D. S.'s needs as identified by the assessment studies, on a multimodal basis. Methylphenidate was begun at a dose of 10 mg orally three times daily (0.6 mg/kg). Individual remedial education, social skills training and insight oriented group therapy were implemented. Attempts were made to involve D. S. in pottery and woodworking, two areas in which he showed aptitude, to help improve self-image. Family therapy was continued.

The institution of methylphenidate treatment was associated with noted changes in D. S.'s behaviour. Staff found him to be less disruptive and more amenable to direction, decreasing conflict with authority figures. His group skills improved and fighting diminished. His mother reported a positive change in weekend home visits and individual meetings with him about his medications helped develop insight into his difficulties and hope about the future.

Discussion

This single-case study design illustrates many of the fundamental issues of clinical assessment in an impulsive, inattentive and overactive adolescent who presented with intrapersonal, social, educational and family difficulties. First, an appropriate diagnosis of attention deficit disorder with hyperactivity was made—because of an awareness of this presentation in the adolescent age group. It would have been possible to explain D. S.'s problems solely in terms of sociopathy or psychodynamic conflicts which may have precluded pharmacologic, behavioural or social interventions.

In this case the diagnosis of attention deficit disorder with hyperactivity was made using DSM-III criteria (1980). These criteria highlight the inability to sustain attention as the central feature of the syndrome, thus shifting the emphasis from overactivity as described in both DSM-II and ICD-9 in which the terms 'hyperkinetic reaction' and 'hyperkinetic syndrome of childhood' are used. This change in focus, although not universally acknowledged may have greater diagnostic utility than behavioural overactivity (Cantwell, 1984) and may

improve a clinician's ability to differentiate this syndrome from age-appropriate overreactivity or overactivity secondary to a chaotic environment.

Further, the diagnostic criteria as found in the DSM-III (1980) are more clearly defined than previously, thus potentially providing greater consistency in clinical diagnosis. Also, by permitting more than one diagnosis to be entered on Axis I, DSM-III (1980) supports concurrence of this syndrome with conduct disorder (in this case conduct disorder, socialized, non-aggressive). Previous comparisons of diagnostic practices between the United Kingdom and North America have reported similarly high rates of hyperkinetic behaviours but up to a 20-fold difference in the syndromal diagnosis of hyperkinesis (Taylor, 1985). The effect of the DSM-III diagnostic system, particularly its focus on attention deficit and the coding of multiple non-hierarchical diagnosis on future epidemiological comparisons remains to be fully studied.

It should be noted that a positive response to methylphenidate both in terms of diminished activity or an increase in attention maintenance does not of itself confirm a diagnosis of ADD. Stimulants may improve symptoms of restlessness and scores on attention-related tests in both overactive and non-hyperactive children with conduct disorder (Taylor, 1983), and have been reported to decrease motor activity and improve vigilance in both hyperactives and normal controls but more so in the hyperactives (Rapoport *et al*, 1980). Sustained attention as measured by reaction time tasks however, improved selectivity in the hyperactives only. Thus although methylphenidate treatment may improve the symptoms of ADD, symptomatic improvement is not diagnostically specific.

The drug trial design illustrated in this case provided objective evaluation of two related treatment issues. First, use of a double-blind cross-over methylphenidate trial during the assessment period provided objective data about the potential value of pharmacological intervention, showing a significant treatment response to methylphenidate. Secondly, the blind multi-dose treatment trial allowed clinical titration of drug effect that balanced behavioural control against overmedication and side-effects.

This second trial showed slightly higher scores both at baseline and at treatment perhaps reflecting the influence of environmental factors, as some major changes including changeover of treating physician had occurred in the Unit between the two trials. However, the initial treatment response was replicated. There was no significant difference in

symptom reduction amongst methylphenidate doses of 0.6 mg/kg, 0.9 mg/kg and 1.2 mg/kg thus identifying a minimum dosage for effective behavioural control without missing potential higher dose responders and reducing the possibility of dose-related methylphenidate side-effects previously described by Sprague and Sleator (1977), and Greenhill *et al* (1984).

These designs proved easy to incorporate into ward routine. Total staff time required was estimated at less than 2 hours and treatment effect of methylphenidate was apparent within 2 weeks of beginning the trial. Inpatient stays of 3-6 months are not uncommon in adolescent treatment units and the objective rapid evaluation of methylphenidate effect as determined by these assessment designs may potentially decrease admission length, providing more cost-effective service without sacrificing comprehensiveness of care.

The use of these assessment studies had beneficial effects for the treatment unit as well. Initial staff scepticism about the use of drugs in adolescents was modified by the objective data derived from the trials leading to an increased vigour in D. S.'s therapy. The assessment model provided a 'live' experiment illustrating that pharmacologic and psychodynamic approaches can successfully complement each other in the treatment of the disturbed adolescent.

Double-blind cross-over and multi-dose treatment trials of methylphenidate should be an integral part of the clinical assessment of the ADD adolescent. Their routine use allows the clinician to bridge the gap between research studies reported in the literature and the multiple presenting problems of the individual adolescent. They provide objective measures of pharmacologic effect and also have psychologic treatment utility. These designs are cost-effective and provide immediate objective verification of clinical response relatively free from placebo effect and negative or overly optimistic attitudes of adolescents, parents and treatment staff towards stimulant medications.

With the involvement of a responsible parent this design can be modified for outpatient assessment. In the adolescent undergoing long-term methylphenidate treatment future changes in stimulant dosage can be similarly assessed, and behavioural responses compared to initial presentation thus increasing the effectiveness of long-term treatment monitoring. Regular clinical use of these assessment strategies may lead to more appropriate pharmacotherapeutic interventions in adolescent ADD and should prevent stimulant drug misuse.

APPENDIX 1

Rating Scale						
Name	Date	Time				
Category	Never	Continuously				
Restless	1	2	3	4	5	
Excitable	1	2	3	4	5	
Inattentive	1	2	3	4	5	
Difficulty concentrating	1	2	3	4	5	
Disturbs others	1	2	3	4	5	
Quarrelsome	1	2	3	4	5	
Temper outbursts	1	2	3	4	5	
Attention seeking	1	2	3	4	5	
Fails to complete tasks	1	2	3	4	5	
Easily distracted	1	2	3	4	5	
Impulsive	1	2	3	4	5	
Needs supervision	1	2	3	4	5	
Trouble with others	1	2	3	4	5	
Fidgety	1	2	3	4	5	
Difficulty staying seated	1	2	3	4	5	
On the go	1	2	3	4	5	
Speaks out of turn	1	2	3	4	5	
Disorganised	1	2	3	4	5	
Comments	Rater.....					

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Computed Tomographic Density Numbers A Comparative Study of Patients with Senile Dementia and Normal Elderly Controls

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Cranial computed tomography (CT) scans of 48 patients with senile dementia of the Alzheimer type (SDAT) and 40 normal elderly volunteers were compared. The demented group were found to have significantly larger lateral ventricles assessed planimetrically and by using a linear measure of the ratio of the maximum width of the frontal horns of the lateral ventricles to the maximum intracranial diameter. Attenuation density was computed for 15 brain regions. The demented group did not differ significantly from the normal controls with respect to attenuation density in any of the regions studied.

Reports on computed cranial tomography (CT) generally concur in the finding that patients with senile dementia have larger lateral and third ventricles than normal elderly controls (Jacoby & Levy, 1980; Gado *et al*, 1982). Similarly patients with senile dementia have been shown to have more severe sulcal widening as rated by an experienced neuroradiologist, than normal elderly controls (de Leon *et al*, 1980; Jacoby & Levy, 1980). There is, however, substantial overlap between groups both with respect to estimates of ventricular size and cortical atrophy and were one to make a diagnosis of senile dementia solely on the CT finding then at least 17% of subjects would be misclassified (Jacoby & Levy, 1980).

It is less clear whether patients with senile dementia differ from normal elderly controls with respect to CT attenuation density (Brinkman *et al*, 1981). Some workers including our own group, have reported patients with senile dementia as having lower brain attenuation density than controls (Bondareff & Levy, 1981; Naeser *et al*, 1982), others reported no difference (Wilson *et al*, 1982; Gado *et al*, 1983) and one group found density to increase with the severity of dementia (George *et al*, 1981). Comparison between studies is difficult for several reasons. Different scanners have been used which may not produce comparable density measurements and in one study results obtained using several scanners were amalgamated (Wilson *et al*, 1982). Studies have used differing criteria for subject selection and attenuation density has been computed for regions that are not comparable in site or size.

In view of the conflicting findings concerning CT brain attenuation density in senile dementia, and the paucity of replicative studies using comparable methods, it was decided to use the same CT scanner and method as in the study of Bondareff *et al* (1981) to examine a new, larger, series of elderly demented patients and controls. This method was chosen as it had fewer drawbacks, than others. The use of a very large brain sample area increases the probability of the inclusion of unwanted areas such as sulci and it might be argued that the finding of lower attenuation density in demented patients by Naeser *et al* (1982) reflected the inclusion of atrophic sulci in the whole and quarter slices which they sampled. Conversely the use of very small brain sample areas might be expected sometimes to result in unrepresentative areas of a particular region being sampled. Furthermore, a small shift in the cursor could result in a quite different reading. The use of high convexity and centrum semi-ovale slices, as in the study by George *et al* (1981), introduces the possibility of obtaining elevated density readings in patients with dementia, where with ventricular enlargement these slices selected would tend to be pushed higher. It is recognised that as one approaches the apex, brain density readings increase (De Chiro *et al*, 1978). Similarly brain density readings taken at the convexity of the brain as in the study by Gado *et al* (1983), might be expected to be raised through the inclusion of partially volumed bone although this would affect both controls and patients with dementia. The method chosen for the present study

utilises a brain sample size of intermediate area and avoids high slices and convexity readings.

Method

Subjects

Subjects consisted of a group of 48 patients who were diagnosed as having senile dementia of the Alzheimer type (SDAT) and were hospital inpatients, outpatients, day-patients or attending a day centre. Controls were a group of 40 normal elderly volunteers who were living independently in the community and showed no evidence of dementia or psychiatric disorder. Informed consent was obtained from all participants or, where not possible, the closest relative. The diagnosis of dementia was made on the basis of progressive global deterioration of higher cerebral function, of at least 6 months duration excluding those patients who had histories of stroke, head injury, alcoholism or major psychiatric illness. Other exclusion criteria were the presence of focal neurological signs, a measured blood pressure greater than 160 mmHg systolic or 100 mmHg diastolic and evidence of an underlying cause of dementia such as vitamin B12 or folate deficiency, endocrine disorder, metabolic disturbance or syphilis. All subjects were interviewed using a semi-structured interview [the Geriatric Mental State Schedule (GMSS) (Copeland *et al.*, 1976)], underwent a full neurological examination, were given a test of memory and orientation, The Mental Test Score (MTS) (Hodkinson, 1973) and performed the Digit Copying Test (DCT) (Kendrick, 1965).

Computed tomography

Subjects were examined using the Maudsley Hospital EMI CT 1010 head scanner. Eight slices were made parallel to the orbitomeatal line. An EMI Independent viewing centre (IVC 464) was used to read the scans. Those scans with no more than minimal movement artefact were selected, displayed on a television screen and, by means of a tracker ball, an approximately circular area of 37 pixels was superimposed on anatomical regions of interest in appropriate slices. The mean attenuation density in Hounsfield units (HUs) was then calculated using a computer programme. Further details of the method are described by Bondareff *et al.* (1981). In addition the ventricular-brain ratio (VBR), that is the ratio of maximum ventricular area to internal skull area was determined using a planimeter. The correlation between the VBR of the same scans by independent raters is high ($r=0.98$, $P<0.001$; Jacoby *et al.*, 1980). A linear measure of the maximum width of the frontal horns of the lateral ventricle to the maximum internal skull diameter (bifrontal/intracranial ratio) was also determined for each subject.

TABLE I
Correlation between cognitive measures and those of lateral ventricular size

	VBR	Bifrontal-intracranial ratio
MTS	-0.70	-0.61
DCT	-0.67	-0.55

All significant at $P<0.001$

TABLE II
Mean attenuation density in HU's and s.d. in different brain regions and analysis of variance

Region	Hemisphere	Patients with senile dementia (n = 48)	Normal elderly controls (n = 40)	F (d.f. = 1)	P ²
Cerebellum	L	42.24 ± 3.91	43.37 ± 4.54	2.47	NS
Cerebellum	R	42.77 ± 4.11	43.36 ± 3.88	0.73	NS
Pons		35.45 ± 4.41	37.29 ± 4.87	2.87	NS
Medial temporal	L	38.08 ± 5.11	37.43 ± 9.15	0.82	NS
Medial temporal	R	37.54 ± 5.23	39.59 ± 4.89	1.62	NS
Anterior frontal	L	39.90 ± 4.09	40.02 ± 3.71	0.18	NS
Anterior frontal	R	39.63 ± 4.24	39.24 ± 3.37	0.08	NS
Caudate	L	38.20 ± 3.86	38.89 ± 2.60	2.35	NS
Caudate	R	37.57 ± 4.73	38.47 ± 2.72	0.84	NS
Thalamus	L	38.28 ± 2.78	39.03 ± 1.80	1.16	NS
Thalamus	R	37.54 ± 2.96	37.83 ± 1.91	0.12	NS
Parietal	L	39.15 ± 2.85	38.84 ± 3.43	0.61	NS
Parietal	R	38.12 ± 2.70	38.73 ± 3.91	0.26	NS
Occipital	L	41.95 ± 3.44	42.55 ± 3.58	0.48	NS
Occipital	R	41.53 ± 3.54	42.28 ± 3.50	0.63	NS

1. NS Indicates not significant analysis of variance.

2. HU indicates Hounsfield Units.

Results

The mean age of the demented subjects (77.9 ± 8.4) did not differ significantly from that of the control group (75.0 ± 8.3). The preponderance of females was similar in both groups (38 of 48 vs 31 of 40).

Demented subjects performed significantly worse than normals both on the Mental Test Score (10.3 ± 5.8 vs 31.8 ± 3.0 , $P < 0.001$) and the Digit Copying Test (28.8 ± 22.7 vs 82.2 ± 34.3 , $P < 0.01$).

Ventricular size was larger in the demented patients than in the control subjects both as assessed by the VBR (18.7 ± 4.2 vs 9.5 ± 4.1 , $P < 0.001$) and by the linear bifrontal/intracranial ratio (38.7 ± 4.1 vs 31.9 ± 4.3 , $P < 0.001$).

Cognitive measures were found to correlate negatively with those of ventricular size when the total sample of normal controls and demented patients was examined but not for the separate groups (Table I).

Although attenuation density was generally lower in SDAT subjects this was not significant in any region when demented and normal subjects were compared (Table II).

Correlations between regional density measurements and cognitive tests were in general low and not statistically significant with the exception of those between the MTS and pontine density ($r = 0.31$, $P > 0.004$), MTS and left thalamic density ($r = +0.20$, $P > 0.03$) and between the DCT and density in the right occipital region ($r = +0.23$, $P > 0.04$).

Discussion

The finding that patients with SDAT have significantly larger lateral ventricles than a group of normal control subjects of a comparable age is consistent with reports by other workers (de Leon, *et al*, 1960; Jacoby & Levy, 1980; Brinkman *et al*, 1981; Gado *et al*, 1982). Cognitive measures were found to

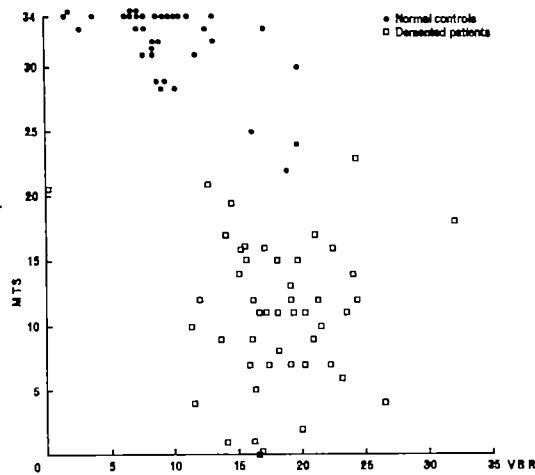


FIG. 1. Scattergram of Mental Test Score (MTS) against ventricular-brain ratio (VBR) for normal controls and demented patients ($r = -0.70$, $P < 0.001$).

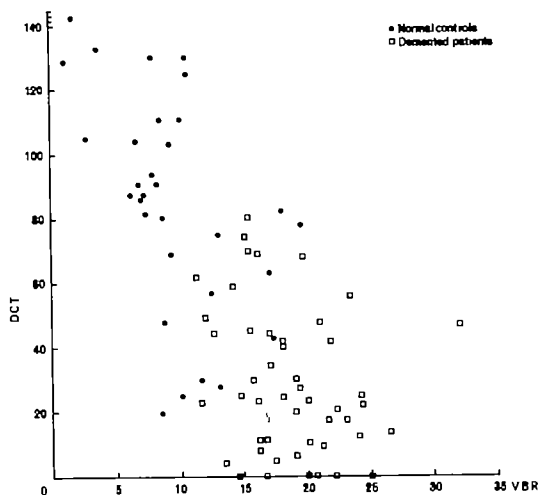


FIG. 2. Scattergram of scores on the Digit Copying Test (DCT) against ventricular-brain ratio (VBR) for normal controls and demented patients ($r = -0.67$, $P < 0.001$).

correlate with those of ventricular size only when the demented and normal control groups were amalgamated as was found by Jacoby & Levy (1980). Scattergrams are provided of scores on the cognitive measures against VBR for normal controls and demented patients combined (Figs 1 and 2). That there were few large or statistically significant correlations between regional attenuation densities and cognitive measures is in keeping with a report by Naguib & Levy (1982) who used the same EMI scanner but a different group of patients. Higher correlations between CT density and cognitive measures have been reported by other workers (de Leon *et al*, 1980) but this may reflect the use of a more advanced scanner or to differences in sampling technique of regions of interest. Unfortunately de Leon & George (1983) do not state what size area of each region of interest was sampled in their study.

The present findings do not suggest that patients with SDAT differ significantly from normal elderly with respect to CT mean attenuation density in any of the regions examined. Our findings do not, therefore, replicate those previously reported by Bondareff *et al* (1981) working with our group. He found significantly lower attenuation density bilaterally in the anterior frontal, medial temporal and caudate regions of a group of patients with senile dementia when they were compared with a group of normal elderly controls. We used the same EMI CT 1010 scanner and similar methodology to that of Bondareff *et al* (1981). It is not likely that the sampling of regions of interest differed greatly in that

the density measurements by the present investigators on the same scans correlated highly ($r=0.93$, $P<0.001$). Scanner drift does not appear to be a likely source of variation in that densities were not found to correlate with date of scan over a 6 month period and a plot of density readings from a phantom over time did not suggest any systematic drift. A possible, although speculative, explanation for the difference in findings of the present study and that of Bondareff *et al* (1981) might be that the samples of demented patients studied were not comparable. Whilst both studies selected patients whose symptoms conformed to the ICD 9 criteria for senile dementia they differed in that those patients in the present study whose blood pressure exceeded 100 mmHg diastolic or 160 mmHg systolic were excluded. The 25 scans used by Bondareff *et al* (1981) were selected from the series of 40 patients reported by Jacoby & Levy (1980). Although in this series patients with hypertensive disease were not included those with high blood pressure alone were deliberately not excluded. Jacoby & Levy (1980) reported 10 patients with silent infarcts and these were significantly more likely to have had a diastolic blood pressure greater than 100 mmHg. It is possible that were many of these patients with silent infarcts to have been included in the sample studied by Bondareff *et al* (1981) this may have predisposed the finding that the demented group had lower attenuation density in some regions, alternatively it may be that patients with raised blood pressure have different CT attenuation densities from those who are normotensive.

In conclusion, it would appear that attenuation density does not significantly differentiate senile dementia subjects from age-matched controls although this method appears to show differences within the dementia group in that low attenuation in the parietal regions is associated with a more rapid demise (Naguib & Levy, 1982; Colgan, 1985). Further studies should therefore concentrate on the use of this method to isolate subgroups with different clinical courses rather than attempting to employ it as a diagnostic procedure.

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Cognitive Functioning in Parkinson's Disease: In Relation to Prevalence of Dementia and Psychiatric Diagnosis

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Forty-three neurologically and psychiatrically assessed patients with idiopathic Parkinson's disease (PD) underwent detailed cognitive assessment. Cognitive deficits typical of senile dementia of Alzheimer's type (SDAT) were found in 7% but the majority showed definite impairments not typical of SDAT. Cognitive impairment was significantly more likely in those with more severe PD symptoms. There was substantial agreement between psychiatric diagnosis and psychological picture of SDAT and some links were found between other diagnostic categories and nature of cognitive functioning. However, cognitive deficits were also found in two-thirds of patients with no psychiatric diagnosis.

The prevalence of dementia in Parkinson's disease has been a longstanding subject of speculation and investigation. It is recognised that it is important to have factual information on the issue for both theoretical and practical reasons, but it is also acknowledged that accurate information is extremely difficult to obtain. Recent reviews (Boller, 1980; Mayeux, 1982; Brown & Marsden, 1984) highlight the wide variation in estimates of prevalence from 20% to 81%.

There are a number of factors which may contribute to such a degree of variation, including the definition of dementia adopted, the means of assessing presence of dementia and the composition of the PD sample.

Definitions of dementia adopted in studies of PD patients have varied from an inclusion of all patients with evidence of cognitive impairment to a narrower definition more consistent with the DSM-III criteria for diagnosis of dementia (American Psychiatric Association, 1980). A useful definition is one whose operational criteria are clearly defined and which is narrow enough for useful theoretical conclusions to be drawn, e.g. for the purposes of the present study our interest is in the presence of a defined constellation of deficits seen as being typical of senile dementia of Alzheimer's type (SDAT).

The method of assessment needs to be able to distinguish changes due to dementia from those caused by other factors, including normal ageing, depression, acute confusional states and physical disability, all of which have an increased risk of being present in patients with PD.

Finally, aetiology of PD in the sample assessed needs to be clearly stated. The most useful group to study from a theoretical point of view are patients with idiopathic PD.

Aim

The central aim of the study reported here was to clarify whether there is an increased risk of patients with idiopathic PD having cognitive deficits typical of senile dementia of Alzheimer's type (SDAT). Secondary aims were to ascertain the proportion of patients with idiopathic PD having cognitive impairments not typical of SDAT; and to examine how the nature and severity of these deficits and impairments related to severity of PD, psychiatric diagnosis and types of treatment being received by subjects. Deficits typical of SDAT were defined as evidence of impairment in secondary memory (shown through impaired performance on a test of secondary memory or on an information/orientation scale); and of a significant decline in general intelligence (shown by a discrepancy of more than one standard deviation between current and pre-morbid intelligence).

Subjects

All patients with a diagnosis of idiopathic PD attending the Regional Neurological Centre, Newcastle General Hospital, between September 1981 and March 1985 were invited to participate in a research study on complications of PD. Two patients declined but a cohort of 81 was established

and all were assessed by neurologists and psychiatrists over a 3-year period to establish the severity of their PD as well as the presence of any degree of dementia or depression, or other significant psychiatric disorder.

Appointments were sent to 63 patients drawn from the cohort of 67 patients still living, for psychological assessment between July 1984 and March 1985. Twenty did not attend (two refused, five moved away, five had temporary ill health, four because of miscellaneous reasons, four for reasons unknown) and four were not sent appointments for practical reasons e.g. they were living a great distance from the hospital. The attenders comprised 21 women (mean age 63.6 years, range 59–77) and 22 men (mean age 66 years range 53–71 years). Their mean duration of disease was 14 years (± 13) and ratings on the Webster Parkinson Disability Scale (Webster, 1968) showed that at the time of psychological assessment 18 had mild impairment (Webster 0–9), 20 showed moderate impairment (Webster 10–19) and five showed severe impairment (Webster 20+). Of those with predominantly unilateral symptoms nine had left-sided disabilities and eight had right-sided disabilities.

All patients except one were receiving medication; (31 levodopa preparations; one bromocriptine, two anticholinergic medication and eight a

combined drug therapy). Three had had stereotaxic surgery.

Age-matched control subjects included spouses of patients and attenders at Newcastle Age Concern Pop-In. None had any severe physical disability or history of psychiatric illness.

Method

All control subjects and 35 patients were seen in a quiet, well lit room. Seven patients were seen at their place of residence and one during a short-stay hospital admission.

Administration of tests took between 45 and 90 minutes and subjects were given time to rest between tests as appropriate. An attempt was made to assess all patients in the areas listed below, with the exception of the visual-spatial imagery test which was administered to a subgroup of 13 patients.

Full descriptions of the tests are not given but brief notes give cogent information on why the tests were chosen for use with this population.

1. *Pre-morbid intelligence: New Adult Reading Test (NART)* (Nelson & O'Connell, 1978). This test has been standardised on British population samples up to age 70 and has proved to give valid results with patients diagnosed as demented.

2. *Current intelligence: Raven's Progressive Matrices (PM)* Standard or Coloured Version, as appropriate (Raven *et al* 1977). This was chosen in preference to the more

TABLE I

Number of patients with idiopathic PD showing normal and impaired performance on each of the series of tests

Area of cognitive functioning	Test	Criteria for impairment	% (n) results in normal range	% (n) results showing impairment
Intelligence	NART PM	Discrepancy of one s.d. or more between PM and NART	82% (28)	18% (6)
Information/orientation	CAPE I/Q scale	Score ≤ 7 shown in validation studies to be strong indicator of the presence of dementia	93% (37)	7% (3)
Primary memory	Digit-Span forwards	Below age-matched norms (i.e. < 5)	86% (33)	14% (5)
	Digit-Span backwards	Below age-matched norms (i.e. < 4)	72% (26)	28% (10)
Secondary memory	Two-choice verbal recognition	Below range scores of 12 age-matched control subjects	71% (24)	29% (10)
Retrieval from long-term memory	Benton Word Fluency Test (Total)	a) Below range scores b) Below 25%ile scores c) Above 25%ile scores of 12 age-matched controls	c) 22% (8)	a) 33% (12) b) 78% (28)
Visuospatial imagery	Block counting test	Below range scores 12 age-matched controls	31% (4)	69% (9)

TABLE II
Cognitive performance of 43 patients with idiopathic PD

<i>Cognitive Performance</i>	<i>Number</i>	<i>% of sample</i>
Cognitive deficits typical of SDAT	3	7
Definite evidence of deficits not typical of SDAT	28	65
Question raised of deficits (at least one mildly impaired score)	8	19
No cognitive deficits	1	2
Insufficient data	3	7

commonly used Wechsler Adult Intelligence Scale since movements are not required to complete the items and it is untimed. In addition, British norms are available up to age 89 years.

3. *Knowledge of current information/orientation: Clifton Assessment Procedures for the Elderly (CAPE) Information/Orientation Scale* (Pattie and Gilleard, 1979). This test has been validated against diagnosis of dementia and standardised on British samples over age 60 years.

4. *Primary Memory: Digit span* forwards and backwards. The standard subtest from Wechsler Adult Intelligence Scale (Wechsler, 1958) was used.

5. *Secondary Memory: 2 choice verbal recognition memory* The test used was an extended version of that described by Morris *et al* (1983) and found to be sensitive to memory deficits of patients with mild to moderate SDAT. The recognition format appears to be equally informative, yet it is less threatening to patients than a recall test.

6. *Retrieval from long-term memory: Benton Word Fluency Test* (Benton, 1968).

7. *Visuospatial imagery: modification of block counting test* from Stanford-Binet Intelligence Scale (Terman & Merrill, 1973). Each item requires mental reconstruction of a three-dimensional object from a line drawing and it thus tests an aspect of visuospatial functioning without requiring a motor response.

Results

All, or the majority of tests, were completed by 36 out of 43 patients with the remainder carrying out such tests as their disabilities and circumstances would allow. Table I shows results with regard to performance on each test and Table II gives the results in a manner related to the aims of the study.

A majority of patients showed deficits on the tests of retrieval from long-term memory (Benton Word Fluency Test) and visuospatial imagery (Block Counting Test).

On the word fluency test significant differences were found between patients and control subjects in the number of words produced on each letter and in total (see Table III). A number of perseverative intrusions (i.e. production

of a word from a previous letter category) were given by 10 (32%) of patients but no control subjects. There was no significant difference in the number of repetitions (i.e. of a word already given) the majority of subjects giving at least one repetitive response.

TABLE III
Average number of words recalled on each letter and in total on Benton Word Fluency Test

	<i>Letter 1</i>	<i>Letter 2</i>	<i>Letter 3</i>	<i>Total</i>
Age-matched Control subjects ($n=12$)	16.4	15.6	15.1	47.1
Idiopathic PD patients ($n=36$)	10.4	9.9	9.9	30.3
Significance of difference	$P<0.01$	$P<0.02$	$P<0.01$	$P<0.001$

On the test of visuospatial imagery control subjects scored significantly higher than the patient group (controls: average 12.5/14; patients: average 7.45/14; $t=3.75$, $P<0.01$).

In all other areas assessed, a minority of patients showed impairment in comparison with age-matched norms drawn from British standardisation studies, or control data.

Relationship of cognitive dysfunction to other measures

A significant, though fairly low, positive correlation was found between length of illness and severity of PD as measured by Webster scale ratings ($r=+0.35$; $P<0.05$). Those with mild symptom ratings were significantly less likely to show cognitive impairment than those with moderate symptom ratings—Webster score 0–9: 61% definite cognitive impairment; Webster score 10–19: 95% cognitive impairment; $\chi^2=4.6$, $P<0.05$). Of five patients with severe symptom ratings, two had deficits typical of SDAT and two had severe communication problems which limited assessment. The fifth showed mild deficits. There was a tendency for a greater proportion of patients who had a lengthy history to have more severe cognitive impairment in comparison with those with a short history but this trend was not significant.

As symptoms of PD became more severe there was a tendency for word fluency to be poorer ($r=0.3$; $P<0.1$). In unilateral cases, side of symptoms had no influence on cognitive deficits found.

The three patients who had undergone thalatomy all had severely depressed word fluency, an effect which has been shown to occur after bilateral ventral thalamic lesions. Effects of medication on cognitive functioning could not be isolated but two patients with a history of drug-induced confusional states showed specific cognitive deficits indistinguishable in character from the general sample.

The relationship between psychiatric diagnosis and psychological assessment is shown in Table IV. For the purposes of this study patients found at psychiatric

TABLE IV

Relationship between psychiatric and psychological assessment of cognitive functioning in 43 patients with idiopathic PD

<i>Psychiatric diagnosis: number (%) with each degree of cognitive impairment</i>						
<i>Cognitive functioning</i>	<i>Dementia</i>	<i>Cognitive deficits</i>	<i>Depression</i>	<i>Anxiety</i>	<i>None</i>	<i>Insufficient data</i>
SDAT deficits	2 (100%)	1 (10%)				3 (7%)
Other deficits		9 (90%)	5 (56%)	1 (100%)	12 (63%)	28 (65%) ¹
? deficits			2 (22%)		6 (32%)	8 (19%)
No deficits					1 (5%)	1 (2%)
Insufficient data			2 (22%)			3 (7%)
	2 (5%)	10 (23%)	9 (21%)	1 (2%)	19 (44%)	1 (2%)

¹ Includes one patient with diagnosis of combined depression and mild cognitive deficits

assessment to have cognitive impairment are divided into two diagnostic groups. The first includes those with SDAT, the second includes those with mild or doubtful dementia and those given a diagnosis of dementia qualified as not typical of SDAT (mild or atypical cognitive impairment). Where a psychiatric diagnosis of SDAT was made this predicted that the patients would show SDAT deficits at psychological assessment. In all other psychiatric categories, the majority of patients showed specific cognitive impairments, this being also true where there was no psychiatric diagnosis though in this group over 33% were found to have no definite evidence of cognitive impairment.

The nature of cognitive impairment showed some significant differences between diagnostic groups. Those diagnosed at psychiatric assessment as having mild or atypical cognitive impairment showed a significant lowering of intelligence (NART: average 109.25, PM: average 94.625; $P < 0.01$) whereas neither the depressives nor those with no psychiatric diagnosis showed any change in average level of intelligence. Those with a diagnosis of depression produced significantly fewer responses on the third letter of the word fluency test (depressives: average 8.1; non depressives: average 11.4; $P < 0.05$).

However, deficits in information/orientation, secondary memory and total production of words in the word fluency test were equally likely whether psychiatric diagnosis was of mild or atypical cognitive impairment, depression or no disorder.

Characteristics of patients not seen

Among those patients who died before they could be psychologically assessed a greater number had severe PD and a greater number received a psychiatric diagnosis of dementia than among the sample seen: 9.3% of all patients given a comprehensive psychiatric assessment received a diagnosis of SDAT with a further 2.7% receiving a diagnosis of dementia (atypical). Among those who did not attend or were not sent an appointment a greater proportion had moderate symptoms of PD and fewer had mild symptoms, but the proportion in the various psychiatric categories of diagnosis did not differ from the sample seen except for a greater number of cases with combined depression and mild cognitive impairment.

Discussion

Prevalence of dementia

Deficits typical of SDAT were found in 7% (three) of the current sample. This figure is lower than any estimate reported prior to 1985 except for a rate of 0 found by Patrick & Levy (1922).

Brown & Marsden (1984) present clearly the reasons why figures produced in many past studies may overestimate the prevalence of dementia. When studies of only those with idiopathic PD were examined the reported prevalence rate fell to 24.5%; and a correction for the use of inappropriate assessment measures led them to suggest that the true prevalence may be 15–20%. Less (1985) recently re-worked data in seven studies to include only patients whose diagnosis of dementia complied with DSM-III criteria and produced figures between 7 and 15%.

Thus re-examination of previous studies to correct for composition of sample, criteria for diagnosis and use of inappropriate assessment techniques produces a dramatic lowering in estimated prevalence to a similar level to that found in the prospective study reported here.

Since Brown & Marsden's critique was published, two short reports of equally low figures (15% and 8%) of prevalence for dementia have been given in published letters (Lees 1985; Taylor *et al* 1985). Thus clear evidence is emerging that the true prevalence rate for a clinical picture of dementia of Alzheimer's type in PD is substantially lower than was previously thought.

Two further points concerning these conclusions need to be made. Firstly we re-emphasise that the definition of dementia adopted influences the prevalence rate found. The term dementia, even defined as strictly as in the DSM-III criteria has disadvantages where an attempt is being made to distinguish between different patterns of cognitive

impairment, since it leads to the assembling of a heterogeneous sample of cognitively impaired individuals.

Operational definitions, which group together individuals who are impaired in stated aspects of cognitive functioning are more informative in this respect, and may clarify distinctions in this situation between patients who at psychiatric assessment appear to have SDAT and those who are felt to have an 'atypical' dementia.

Secondly, in this study despite using an operational definition of cognitive impairment we have tied our definition to a neurological level of explanation by using the term SDAT which implies the presence of certain cognitive pathology. The underlying pathology, however, cannot be assumed since identical cognitive impairments may be due to differing underlying pathology. Perry *et al* (1985) recently found that the pathological changes in a group of clinically demented PD patients differed from those in patients having SDAT in the absence of PD.

We therefore conclude that a lower proportion of PD patients have impairments typical of SDAT than was previously thought; but that the level of prevalence is nevertheless above that expected in the population of this age. Kay *et al* (1970) reported a prevalence rate of 2.4% among their population sample aged 65–69 years. This increased level of prevalence does not necessarily imply an increased risk among PD patients of developing SDAT but may indicate that a number of patients show cognitive deficits usually associated with SDAT but in their case consequent upon other cortical changes that may occur as a complication of PD.

Prevalence of other types of cognitive impairment

The results of the present study emphasise that many PD patients with no global dementia face difficulties in cognitive functioning. Taylor *et al* (1985) and Lees & Smith (1983) also found that a majority of patients with idiopathic PD showed subtle cognitive deficits not amounting to dementia. Risk of cognitive dysfunction appeared to increase to some degree with length and severity of illness, confirming some past findings (reviewed by Mayeux, 1982) but being contrary to the view recently put forward by McCarthy *et al* (1985) that there is correspondence between incidence of cognitive deficits in early and late PD. This issue may only be clarified by a longitudinal study of a patient cohort.

Interlink between psychiatric and psychological assessment

The results of this study suggest that where a psychiatric diagnosis of dementia has been made, the

patient is also likely to show SDAT deficits at psychological assessment. However, in all other cases, degree of cognitive dysfunction is almost independent of psychiatric diagnosis. This is such that the patient who receives no psychiatric diagnosis is almost as likely to show definite impairments as the patient who receives a diagnosis of affective disorder or mild or atypical cognitive impairment.

The dissociation, however, is not complete since the nature of the cognitive dysfunction may vary in some respects. Where a psychiatric diagnosis of mild or atypical cognitive impairment is made it seems that the psychiatrist is responding to a decline in general intelligence in the absence of clear-cut memory impairment, but more subtle impairments in word fluency and secondary memory may pass unnoticed. It should therefore be useful during psychiatric assessment of a patient with PD to include a simple test of word fluency in the absence of a full psychological assessment of cognitive functioning.

Where a patient is depressed there may be typical cognitive deficits as a result of the depression but these do not account for all the impairment found among this diagnostic group.

Conclusions

Results are now emerging from a number of sources, including the present prospective study, to suggest that the prevalence of deficits typical of SDAT in PD is lower than was previously thought, affecting only 5–15% of patients. More subtle forms of cognitive impairment may be much more common but can be missed during psychiatric examination. Their nature and course needs to be clarified through longitudinal follow-up and clearly designed psychological investigations.

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Interactions between Procyclidine and Neuroleptic Drugs Some Pharmacological and Clinical Aspects

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Procyclidine was administered to 25 chronic psychotic inpatients, stabilised on chlorpromazine, haloperidol or fluphenazine decanoate injection. We observed a significant reduction in mean serum levels of all three neuroleptic drugs which was reversed on stopping procyclidine and was inversely correlated with mean serum procyclidine levels. No significant alterations occurred on the BPRS scores or in serum prolactin levels throughout the study in any of the three groups. Possible mechanisms of this interaction and its clinical relevance are discussed.

Extrapyramidal side-effects are a frequent accompaniment of neuroleptic treatment and anticholinergic agents used to reduce these symptoms are the commonest form of medication concurrent to neuroleptic therapy (Johnson, 1983). Apart from the observation that anticholinergics may not be superior to placebo in the treatment of drug-induced parkinsonism (Brumlik *et al*, 1964; Mindham *et al*, 1972), it has also been claimed that their use can result in an exacerbation of psychosis (Stephens, 1967; Shader & Greenblatt, 1971; MacVicar, 1977), a delay in symptom improvement (Singh & Kay, 1975, 1979; Johnstone *et al*, 1983) and a worsening of tardive dyskinesia (Crane, 1973; Klawans & Rubovits, 1974).

The possibility that these effects may be due to alteration in the blood levels of neuroleptics has been the subject of many studies. The results, however, are by no means unequivocal. Rivera-Calimlim *et al* (1973, 1976), Loga *et al* (1975), Gautier *et al* (1977) and Morselli *et al* (1980) reported that co-administration of anticholinergic drugs resulted in lower blood levels of neuroleptics. In several other studies no differences were observed (El-Yousef and Manier, 1974; Hansen *et al*, 1979; Linnoila *et al*, 1980; Simpson *et al*, 1980; Johnstone *et al*, 1983) and an increase in chlorpromazine levels was found by Kolakowska *et al* (1976).

We re-examined our data from a study relating neuroleptic dosage to serum level in chronic schizophrenics (Krska *et al*, 1986), dividing patients into two groups according to whether an anticholinergic was prescribed. For most neuroleptics, there were no differences in the dose, serum level or in the serum level/dose ratio between the two groups. In patients

the two groups. In patients treated with fluphenazine decanoate, however, those patients receiving anticholinergics had significantly lower serum level/dose ratios than those on fluphenazine alone ($P=0.04$, Mann Whitney U Test).

This study was designed to investigate the effect of administration of an anticholinergic drug on neuroleptic blood levels and clinical parameters in patients stabilised on different antipsychotic agents. The neuroleptics studied were fluphenazine decanoate, as an example of a parenteral drug and chlorpromazine and haloperidol, both oral drugs, but with differing chemical structures and metabolism. Procyclidine was used as the anticholinergic drug and a method of measuring its serum levels was developed to establish compliance.

Method

Clinical procedures

Subjects

Male and female inpatients in Prestwich Hospital, diagnosed as chronically schizophrenic, who had no physical illness and had been stable on one neuroleptic for at least 4 weeks, were selected for the study. Of these 12 were receiving chlorpromazine, 12 haloperidol and nine fluphenazine decanoate.

Design of the study

(Fig. 1). The protocol was passed by Salford Health Authority Ethical Committee and was carried out between June 1983 and July 1984. Patients entering the study continued to receive the same dose of the neuroleptic on which they had been maintained for several weeks, or in

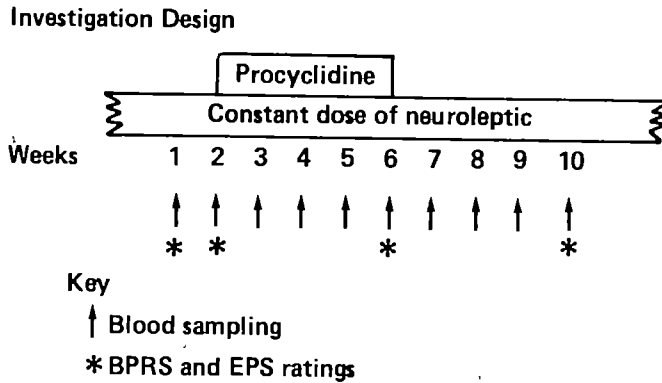


FIG. 1. Design of the study. Blood samples were collected every week; clinical assessments done at weeks 1, 2, 6 and 10.

most cases, months prior to the study. This dose did not change in any patient throughout the 10 weeks of the study. Procyclidine 5–15 mg/day (depending on the severity of extrapyramidal symptoms) was added to the neuroleptic therapy from the end of week 2 for exactly 4 weeks. It was then discontinued abruptly and the patients maintained on neuroleptic alone until the end of the study.

The only non-study drug administered was in patient no. 20 who received sulthiame 50 mg twice daily throughout. Blood samples were taken weekly for the whole 10 weeks and clinical assessments made on weeks 1, 2 (baseline), 6 (after 4 weeks of procyclidine) and 10 (4 weeks after stopping procyclidine). Occasional blood samples were omitted in a few patients for various reasons; where this was the case, it is noted in the legends to the figures.

Rating procedures

Psychiatric status was assessed using the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1962) and extrapyramidal side-effects using the Extrapyramidal Scale (EPS) of Simpson and Angus (1970). Assessments were made prior to blood sampling on four occasions throughout the study. Each patient was assessed by the same rater throughout.

Biochemical procedures

Laboratory personnel responsible for measurements were blind to all treatments.

Neuroleptic levels

Serum levels of all neuroleptics were measured by the radioreceptor assay method (Krska *et al.*, 1986) using haloperidol as internal standard. The results are expressed in terms of 'neuroleptic units'.

Prolactin levels

Serum prolactin was measured by radioimmunoassay using a kit obtained from Chelsea Hospital for Women (Dovehouse Street, London). The precision of this method was 4.8% within assay and 6.2–8.2% between assay over the range 150–850 mIU/litre. Prolactin levels in nine

normal males and 14 females were 88–215 and 99–250 respectively.

Procyclidine levels

A method of measuring procyclidine in serum by antiparkinsonian radioreceptor assay was developed for this study, based on the method of Tune & Coyle (1980). Samples were assayed after precipitation of proteins so values obtained represent total drug (i.e. free and bound to serum proteins). Procyclidine was used for the standard curve and results are expressed as nmol/litre. Recovery of procyclidine from spiked serum was $95 \pm 13\%$, $95.5 \pm 12.4\%$ and $98.6 \pm 7.9\%$ for concentrations 500, 1000 and 1500 nmol/litre respectively.

Statistical analysis

All data were divided according to neuroleptic drug and subjected to analysis of variance for differences among week numbers. If this was significant, paired *t*-tests were performed for selected week numbers. Relationships between variables were studied by Pearson's product moment correlations.

Results

General

Of the 12 patients who were in the chlorpromazine group, two dropped out because of uncooperation or aggression. One patient receiving haloperidol was excluded because of antiparkinsonian side-effects; one patient in the fluphenazine group refused to give blood samples and a second patient developed an increase in psychotic behaviour before the introduction of procyclidine; both were excluded. The demographic data for the remaining patients (Table I) showed no significant differences in any of these parameters between groups.

When the samples were analysed, one patient in the chlorpromazine group (no. 10) had very low neuroleptic levels and was judged to have been non-compliant. One patient in the haloperidol group (no. 21) had detectable

TABLE I
Demographic data and medication of all patients included in the study

Patient number	Age (years)	Sex	Weight (lb)	Duration of illness	Duration of time in hospital	Medication	Dose (mg/day) ¹
1	61	M	153	24	24	Chlorpromazine	500
2	28	M	149	2	2	"	600
3	58	M	161	31	31	"	300
4	54	M	133	35	32	"	200
5	60	M	131	29	29	"	400
6	64	M	134	38	14	"	200
7	53	M	140	3	1	"	400
8	58	F	120	8	4	"	250
9	66	M	133	44	43	"	400
10	52	M	127	18	18	"	300 ²
11	62	F	144	28	28	Haloperidol	15
12	53	M	91	23	20	"	15 ³
13	61	F	142	26	20	"	10
14	56	F	161	23	7	"	30
15	45	F	162	29	29	"	10
16	22	M	147	3	1	"	20
17	58	M	148	28	28	"	45
18	53	M	131	28	28	"	20
19	54	M	168	12	1	"	15
20	47	M	133	29	23	"	30 ⁴
21	59	M	109	36	6	"	30 ⁵
22	58	M	126	37	34	Fluphenazine	18.75
23	54	M	185	11	8	"	25
24	46	F	120	26	1	"	18.75
25	48	F	126	21	1	"	25
26	55	F	131	26	26	"	6.25
27	56	M	146	11	10	"	8.3
28	69	F	115	37	37	"	6.25 ⁶

1. Dose in mg/week for fluphenazine decanoate

2. Data excluded from analysis as patient was found to be noncompliant

3. Data from weeks 7-10 excluded from analysis as procyclidine not discontinued

4. Patient received sulthiame 50 mg twice daily throughout the study

5. Data excluded from analysis as patient had detectable procyclidine levels throughout the study

6. Data excluded from analysis as no procyclidine was detected in any sample

procyclidine levels. All three patients were excluded from statistical analysis.

Serum neuroleptic levels

The administration of procyclidine resulted in a significant fall in serum neuroleptic levels in 19 of the remaining 25 patients. In three patients on chlorpromazine and two on haloperidol there was little change in the neuroleptic level. One patient in the chlorpromazine and another in the fluphenazine group had higher levels (+14% and +27% respectively) whilst also receiving procyclidine.

In the chlorpromazine group, the mean neuroleptic level fell gradually from the baseline level after initiation of procyclidine, reaching a minimum after 3 weeks. The level then increased again, despite continuing procyclidine and continued to rise back to baseline after stopping the drug. In haloperidol treated patients, the mean neuroleptic level

fell significantly after only 1 week on procyclidine and remained low until the drug was stopped, after which the level returned to baseline. The pattern in patients on fluphenazine was similar to haloperidol; mean neuroleptic levels were low on all 4 weeks of procyclidine treatment, then returned to baseline after stopping the drug. The mean neuroleptic levels of all groups are shown in Figs 2, 3 and 4.

Prolactin levels

Prolactin was elevated in six out of nine patients on chlorpromazine, five out of six on fluphenazine and all on haloperidol. Among the patients on haloperidol and fluphenazine, those who had high neuroleptic levels tended to have higher prolactin levels but this was not the case for chlorpromazine. In most patients, prolactin varied only slightly from week to week, but in a few patients large swings were seen. There were no overall trends in prolactin

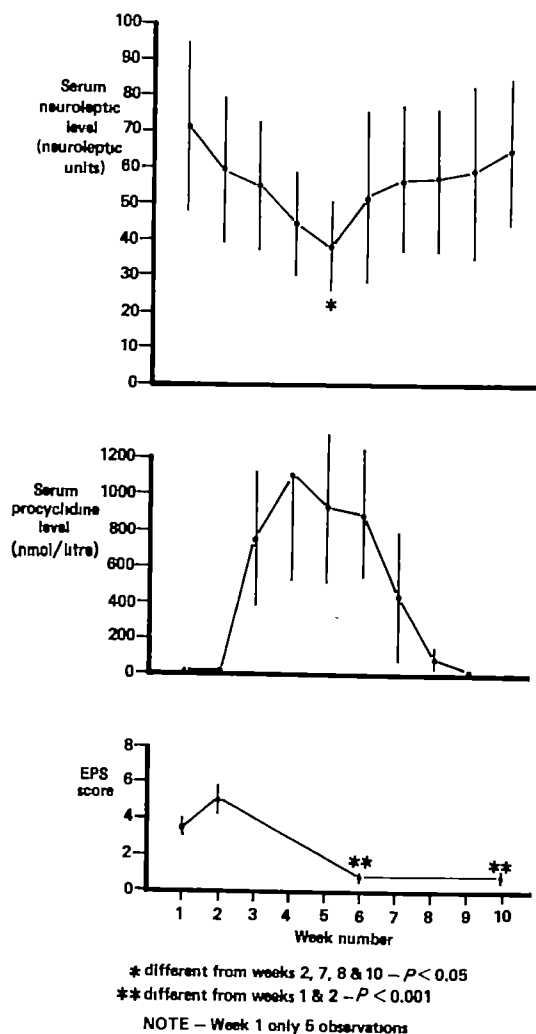


FIG. 2. Effect of procyclidine, administered from weeks 3 to 6, on serum neuroleptic level, serum procyclidine level and extrapyramidal symptom score in nine patients receiving chlorpromazine. Points are mean $\pm$ standard error.

levels and no significant differences between weeks in any of the three treatment groups. Mean prolactin levels were not related to mean neuroleptic levels.

Serum procyclidine levels

Procyclidine was detected in weeks 3 to 6 in all patients except one from the fluphenazine group whose data were excluded from analysis. The levels were fairly constant within each patient and declined gradually on stopping the drug. Levels varied widely between patients and were not related to the daily dose of procyclidine. Mean neuroleptic levels inversely correlated with mean procyclidine levels for all three groups: chlorpromazine $r = 0.805$, $P < 0.005$;

haloperidol $r = 0.907$, $P < 0.0005$; fluphenazine $r = 0.773$, $P < 0.01$. Mean procyclidine levels for all groups are shown in Figs 2, 3 and 4.

Clinical state

The three groups were the same with respect to clinical state as measured by the BPRS score before procyclidine was given (week 2). There were no changes in the mean BPRS score in any group after 4 weeks on procyclidine (week 6) or after a further 4 weeks on neuroleptic alone (week 10). However, in one patient (no. 12) there was a substantial fall in the total BPRS score (9 points) at week 6, which was due to a reduction in positive symptoms. A second patient (no. 13) became considerably more psychotic (increase of

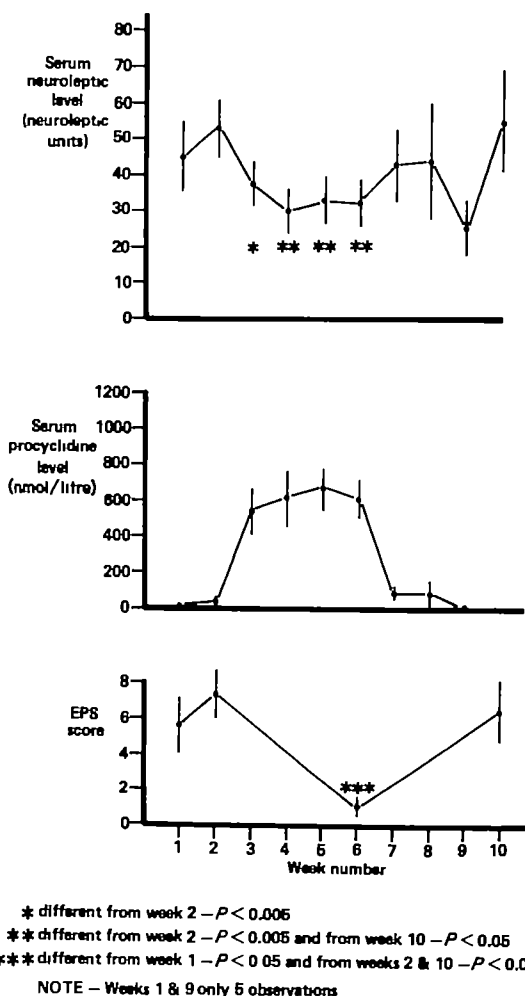


FIG. 3. Effect of procyclidine, administered from weeks 3 to 6 on serum neuroleptic level, serum procyclidine level and extrapyramidal symptom score in ten patients receiving haloperidol. Points are mean $\pm$ standard error.

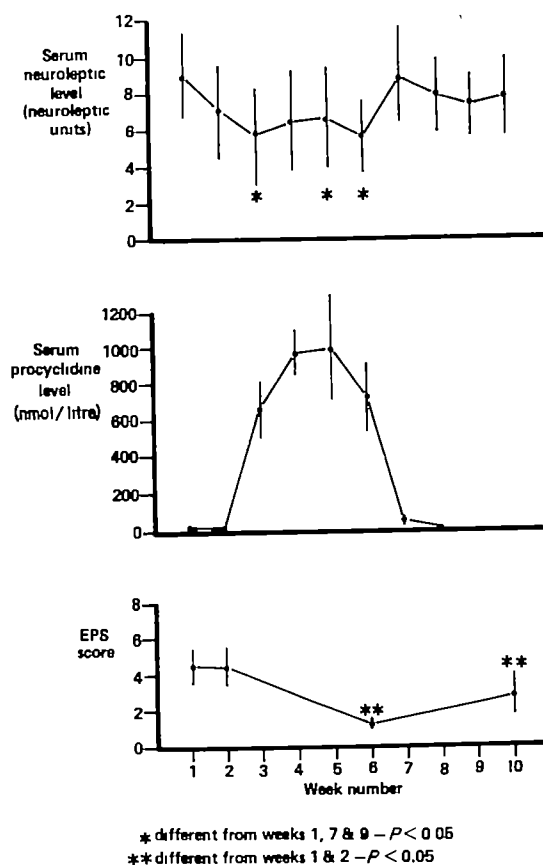


FIG. 4. Effect of procyclidine administered from weeks 3 to 6 on serum neuroleptic levels, serum procyclidine level and extrapyramidal symptom score in six patients receiving fluphenazine decanoate. Points are mean $\pm$ standard error.

15 points on the BPRS score) whilst receiving procyclidine. Both patients were in the haloperidol group. These changes were reversed after stopping procyclidine; no further changes in neuroleptic dose were required.

Side-effects

There were no significant differences in the EPS scores between groups before procyclidine treatment was started. In all three groups the score was greatly reduced after 4 weeks on the anticholinergic ($P < 0.05$ at least, paired *t*-test). However, only in the haloperidol group did the score return to its previous level 4 weeks after stopping procyclidine. In patients receiving either chlorpromazine or fluphenazine, EPS scores remained lower than at week 2 despite the return of the neuroleptic levels to baseline and the absence of detectable procyclidine in the serum. Mean EPS scores are shown in Figs 2, 3 and 4.

One patient in the fluphenazine group had an apparent increase in tardive dyskinesia during procyclidine treatment

which had improved by 1 week after its discontinuation. As no rating scales for tardive dyskinesia were performed this effect was not quantified.

Discussion

The main finding of our study is that anticholinergic antiparkinsonian medication significantly reduces the serum levels of neuroleptic drugs. This is in agreement with the studies of Rivera-Calimlim *et al* (1973, 1976) and Loga *et al* (1975). Discontinuation of procyclidine resulted in a reversal of this effect on neuroleptic levels. This is also in keeping with the results of Gautier *et al* (1977) and Morselli *et al* (1980). Although control groups were not included in our study, several factors suggest that these changes were not due to either temporal variation or placebo effects. Patients entered the study at different times, independently of their neuroleptic treatment; it is therefore unlikely that all would have had a change in serum level at the same time from any other cause. The changes in serum levels are related temporally to both administration of procyclidine and to measurable levels of this drug in serum but not to the dose of procyclidine as reported by Chouinard *et al* (1977).

Despite the overall effect of procyclidine on neuroleptic levels, there were no differences in both the mean percentage fall in serum level and in the proportion of patients showing a fall between the three neuroleptic groups. These differences may help explain the conflicting results found by other workers such as El-Yousef & Manier (1974) and Hansen *et al* (1979), who showed no changes in serum levels of butaperazine and perphenazine respectively.

In the chlorpromazine group, only five out of nine patients had reduced serum levels, three showed no changes and one had an increased level while receiving procyclidine. Similar observations have been reported by Rivera-Calimlim *et al* (1976), who found three of 15 patients had higher chlorpromazine levels while on benzhexol and by Simpson *et al* (1980), whose patients had lower, higher or unchanged levels. A significant difference in mean chlorpromazine levels did not emerge until the third week of procyclidine therapy, which is similar to the results obtained by Loga *et al* (1975) using orphenadrine. This is in contrast to the lack of change after 15 days in the study reported by Simpson *et al* (1980) and an increase in levels found by Kolakowska *et al* (1976) after 7 days of benzhexol.

The interaction of anticholinergic drugs with haloperidol has been studied rather less than with chlorpromazine. Eight out of ten patients had a dramatic reduction in serum level which was

maintained throughout the 4 weeks of procyclidine treatment. A reduction in haloperidol serum levels in two out of three patients when given benzhexol has also been reported by Morselli *et al* (1980). However, Linnoila *et al* (1980) in a rather complicated study, found no differences in serum levels of either haloperidol or thioridazine in behaviourally disturbed, mentally retarded patients whether or not they also received biperiden. Studies of large populations of neuroleptic-treated patients with or without long-term anticholinergic drugs, such as those of Forsman & Ohman (1977) and Itoh *et al* (1980) cannot be directly compared to the results of the present study. Nevertheless, the lack of differences in serum levels between these groups of patients, which we have also found, may suggest that tolerance develops to this effect of anticholinergics.

The reduction in fluphenazine serum levels in five out of six patients was surprising because of the depot characteristics of fluphenazine decanoate. However, lower relative serum levels had been found previously in patients on long-term anticholinergic drugs. We have been unable to find any published data with which to compare these findings. Although Hansen *et al* (1979) did find that intravenous biperiden did not affect the blood levels of intramuscularly administered perphenazine, this is not comparable to longer-term anticholinergic administration.

Our results are particularly interesting with regard to the possible mechanism(s) by which anticholinergic drugs could lower neuroleptic levels. The reduction in gastric and intestinal motility caused by these drugs is dependent on many factors but may lead to prolonged intestinal transit time. In the case of chlorpromazine, this could result in increased intestinal metabolism and consequently reduced absorption (Rivera-Calimlim, 1976). Since the serum levels of a parenterally administered drug (fluphenazine) were also lowered by the anticholinergic in our study and the patterns of reduction in neuroleptic levels were more or less similar in the three groups, effects on gastric motility may be only of minor importance in the interaction. The induction of liver metabolising enzymes has been proposed as a possible mechanism (Loga *et al*, 1975) and orphenadrine has been shown to be a powerful enzyme inducer both in animals (Conney and Burns, 1959; Conney *et al*, 1960) and in man (Loga *et al*, 1975). Procyclidine may have a similar effect, which at least in haloperidol and fluphenazine treated patients, significantly alters serum neuroleptic levels within a week.

In the majority of patients, prolactin levels remained unaltered throughout the study, indicating

that dopaminergic function in the tubero-infundibular pathway is not affected by anticholinergic agents. Other workers have also found these drugs to have no effect on serum prolactin, both in patients (Kolakowska *et al*, 1976; Lindholm *et al*, 1978; Johnstone *et al*, 1983) and in normal volunteers (Halbreich *et al*, 1980). On the other hand, Lal *et al* (1979) showed that benztropine slightly increased prolactin response to haloperidol in normal volunteers, while de Rivera *et al* (1976) found that some chronic patients on neuroleptics and anticholinergics had higher prolactin levels than patients on neuroleptics alone. Our present data do not support these findings. However, in two groups of 25 patients on chlorpromazine, haloperidol or fluphenazine decanoate, matched for sex and dose of neuroleptic, we found the group which was receiving anticholinergic drugs did have higher serum prolactin levels (unpublished data). We feel the data from our present study are more likely to reflect the true effect of anticholinergic drugs on prolactin, since each patient acts as his own control, thereby eliminating the large interpatient differences which occur in the prolactin response to neuroleptics.

The adverse effects of anticholinergic drugs on psychotic symptoms found by many workers in acute patients (Hanlon *et al*, 1966; Singh & Smith, 1973; Singh & Kay, 1979; Johnstone *et al*, 1983) were not evident in the present study. Only one patient showed clinical deterioration during procyclidine administration and this was not associated with any marked changes in neuroleptic levels. Other studies involving similar, chronically ill patients in hospital also report no adverse clinical effects of anticholinergics (Kolakowska *et al*, 1976; Gautier *et al*, 1977; Simpson *et al*, 1980). Overall, this would suggest that effects such as delay in symptom improvement are more significant in the acute population.

All patients had a reduction in EPS score during procyclidine treatment. Discontinuation of procyclidine led to a return of extrapyramidal symptoms in only two patients on fluphenazine and five on haloperidol, and in only one of these was it found necessary to reinstate therapy. This observation is in agreement with studies of Orlov *et al* (1971) and McClelland *et al* (1974) and supports the recommendation that antiparkinsonian drugs should not be routinely prescribed with neuroleptic therapy (DiMascio & DeMirgian, 1970).

An increase in tardive dyskinesia seen in one patient appeared to be related to procyclidine, as the symptoms were reduced when the drug was discontinued. This effect of anticholinergics has been reported in the literature (Klawans & Rubovits, 1974).

In conclusion, our results show that procyclidine causes a significant reduction in the neuroleptic levels in chronic psychotic patients, without any significant adverse clinical effects. This reduction is inversely proportional to the serum procyclidine level, but is unrelated to the dose. The findings in our study suggest that the effect is due to enzyme induction by procyclidine, which would support the findings of delayed symptom improvement in acute patients treated simultaneously with neuroleptics and anticholinergics. Prolactin levels were not found to be affected by procyclidine. However, the drug did significantly alleviate extrapyramidal symptoms, which did not recur in the large majority of patients on discontinuation. This implies that antiparkinsonian drug administration is, in general, unnecessary with chronic neuroleptic therapy, but that adverse clinical effects are not as important as in acute patients.

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The Epidemiology of Deliberate Self-harm The Ibadan Experience

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In a 6-month prospective study of the three main general hospitals in Ibadan, Nigeria, 39 cases of deliberate self-harm were reported out of a total 23, 859 subjects attending the sections of hospital studied. Thirty (76.9%) were under 30 years of age and 36 (86.3%) were under 34 years of age. The male to female ratio was 1.4.1: more than half of the population (51.3%) were students and 25.6%, manual workers. The methods used were mainly ingestion of chemicals (24 patients) and psychotropic drugs (11 patients). Disturbed interpersonal relationships, especially with parents, were mainly found to have motivated the acts. The implications of these findings are discussed.

In the last three decades, reports on the epidemiology of deliberate self-harm (DSH) in Western European and North American countries have shown many similarities (Weissman, 1974). Studies found that most subjects are aged between 20 and 30 years; there are more females than males involved; also more single persons and more persons from the lower social classes; self-medication is the most common method of self-harm, and depression is the most common diagnosis (Edwards & Whitlock, 1968; Aitken *et al*, 1969; Stanley, 1969; Birtchnell, 1970; Hetzel, 1971; Jacobson & Tribe, 1972; Murphy, 1972; Patel *et al*, 1972; Weissman *et al*, 1973; Whitehead *et al*, 1973). Moreover DSH occurs quite frequently in the context of recent interpersonal conflicts (Ellis *et al*, 1966; Venkoba, 1971); and the intensity of hopelessness in depression correlates with suicide intent (Pierce, 1977; Dyer & Kreitman, 1984).

From the few reports on DSH in Nigeria, the prevalence rate ranged from 1 to 7 per 1 000 000; males predominated and major psychiatric disorders were found to be associated with the act (Asuni, 1967; Eferakeya, 1984). Regarding the methods used for DSH, Asuni (1967) found physical self-harm (slashing of the throat etc) to be more common, although a more recent report showed that the ingestion of psychotropic drugs and chemicals was more prevalent (Eferakeya, 1984).

While both studies shed some light on the epidemiology of DSH in Nigeria, Asuni (1967) was unable to interview most of his cases and Eferakeya (1984), in using a retrospective design, limited the extent of the information obtainable. Yet, more than before, the frequency of reports in the news media of fatal and non-fatal DSH suggest an increase in its prevalence in Nigeria.

The need for more current epidemiological information on the subject prompted the present study, which is part of a larger study of fatal and non-fatal DSH in Ibadan. Our operational definition of a case was based on an episode of non-fatal, deliberate self-harm by means of physical injury, drug overdosage or poisoning carried out in the knowledge that it was potentially harmful and in the case of drug overdosage, that the amount taken was excessive (Morgan *et al*, 1975).

Method

The study was conducted in Ibadan, Nigeria. Ibadan, the capital of the present Oyo State of Nigeria is one of the largest cities in Africa. It is a sprawling city, with a projected population from the latest census, of 1.5 million people of the Negroid race. It is located about 150 km from Lagos, the capital of Nigeria. Within the city, health services are provided by government and private institutions. However, our preliminary study of the private practitioners in this city revealed that cases of DSH are hardly ever referred to such health services but to government specialist health institutions (unpublished data). This observation guided us in selecting the three main general hospitals in the city for the study. These are the Adeoyo State Hospital, Ring Road Specialist Hospital (both controlled by the Oyo State Government) and the University College Hospital (a Federal Institution). The areas of the University College Hospital covered throughout the study were the accidents and casualty or emergency sections and, medical and surgical inpatient services. At the State hospitals, the accident and emergency sections only were studied.

Patients population

All patients who fulfilled the operational definition of DSH and attended the above-mentioned sections of these

hospitals over a 6-month period (August 1984 to January 1985) were included in the study.

Interview procedure

A questionnaire was designed to collect information in the following areas; demographic data, reasons for deliberate self-harm, the method used, hospital diagnosis, psychiatric diagnosis, previous history of physical or mental illness, life events preceding the incident, history of substance abuse and treatment outcome. Experienced nursing sisters and a psychologist administered the questionnaire to the patients. Prior to the commencement of the study, the interviewers were trained in the use of the questionnaire and their joint interview ratings reached a satisfactory level of reliability before the main project started.

The interviewers ensured that the patients were interviewed as soon as possible after they had recovered from the major adverse effects of the attempts (usually within 24–72 h). The information obtained from the interviews were not made available to the physicians responsible for the patients' treatment or the psychiatrist consulted to assess the patient.

Apart from the questionnaire designed specifically for this project, the supplementary classification section of the International Classification of Diseases, ninth edition (ICD-9) was used to standardise the diagnosis given by the hospital doctors in non-psychiatric divisions of the hospital. Also, the clinical psychiatric diagnosis was based on Section V of the ICD-9 using the WHO glossary of definition of symptoms.

The data collected was manually analysed; *t*-test and *z* scores were used for test of significance with $P < 0.05$ as the level of statistical significance.

Results

Population studied and rate of deliberate self-harm

During the 6 months of the study, the total number of patients attending the accident and emergency units of the Adeoyo State Hospital and the Ring Road Specialist Hospital was 13 368. In the same period, 10 491 patients attended either the accident and emergency unit or were admitted to non-psychiatric wards of the University College Hospital, Ibadan. The 6-month incidence rate of DSH for the 1.5 million population of Ibadan was calculated to be 2.6 per 100 000. Of these, 39 patients were cases of deliberate self-harm; 18 of these 39 patients were seen at the State hospitals while 21 reported at the University College Hospital. On all the variables, we found no statistical difference between the population of State hospitals and that of the University College Hospital.

Age distribution

The mean age was 23.3 ± 7.3 years, median 21 years. There were 23 males and 16 females, the male to female ratio being 1.4:1. The male mean age was 24.8 ± 7.3 years. The female mean age was 21.1 ± 7.2 years. There was no statistical difference in age between the sexes ($t = 1.6$; $P > 0.05$; d.f. = 37). Thirty-three (84.61%) of the 39 patients were aged between 15 and 34 years. When the cohort was divided

into age groups, the peak of DSH (11 of 39, i.e. 28.2%) was in the age group 15–19 years.

Religion

Twenty-six (66.67%) of these patients were Christians whereas 13 (33.33%) were Muslims. The ratio of Christians to Muslims is therefore 2:1. This is in spite of the fact that Ibadan is mostly populated by Muslims.

Occupation

When the 39 patients were classified according to their occupations, 20 (51.3%) were students, 10 (25.6%) were manual workers, four (10.3%) were semi-skilled workers, three (7.7%) were skilled workers while only two (5.1%) were unemployed. Of the 20 students, 11 were females (i.e. 68.8% of the total 16 females) and nine were males (i.e. 39.1% of the total 23 males). There were more manual workers among the males than the females (39.1% and 6.3% respectively).

Marital status

Twenty-eight (71.8%) patients were single, ten (25.6%) were married, while only one (2.6%) was divorced.

Sibling position

In three patients, the sibling position was not stated by the interviewers. Of the remaining 36 subjects 18 (50%) are first borns; 25 (69.4%) of all patients are either first or second borns; only three (8.3%) had sibling position above fourth. There were no last borns.

Methods used in DSH

Table 1 shows the methods of deliberate self-harm used. Thirty-six (92.3%) of the thirty-nine patients used solid or liquid substances; 24 (61.5%) used non-ingestant chemicals amongst which insecticides, battery water and gamallin-20 were the most common. A total of 12 (30.8%) of the patients used psychotropic drugs, with diazepam being the commonest. The females showed a tendency to use a wider variety of psychotropic drugs than the males. Only three (7.7%) of the patients used physical means, namely, cutting throat with a blade (one case), setting one's self on fire (one case) and jumping from a height (one case).

Diagnoses

Acute reaction to stress was the diagnosis in 16 (41%) of the 39 cases. Five (21.7%) males and eleven (68.8%) females had this diagnosis. The diagnosis of acute reaction to stress was significantly more common ($P < 0.05$) among females than males. Ten (25.6%) cases were diagnosed as reactive depression; among these were seven males and three females. Four patients (10.3%) had a diagnosis of adjustment reaction. Other diagnoses are depressive psychosis, schizophrenia (two cases each, all males), sexual deviations (two cases, one male, one female); and psychic factors associated with other diseases (one case each, both males). Only in one subject was there insufficient information to make a psychiatric diagnosis based on the ICD-9.

Reasons for deliberate self-harm

Disturbed relationship with parents accounted for eight cases (20.5%); interpersonal discord with significant others, excluding spouses six cases (15.4%); marital discord, five cases (12.8%); frustration or shame resulting from failure in examinations, eight cases (20.5%); disappointment due to unwanted pregnancy, one case (2.6%); being reproached for impregnating a girl, two cases (5.2%); mental illness, five cases (12.8%); unresolved physical illness, unemployment, job dissatisfaction and financial problem, one case each.

Outcome of treatment

Seventeen (48.6%) of the 39 patients were referred to the psychiatrists while 15 (38.5%) were discharged home without any planned follow-up or even referral to psychiatrists. One patient (2.6%) was transferred to a peripheral, non-psychiatric hospital.

Fatal deliberate self-harm patients

Five patients died, three females (ages 15, 15, 16 years) and two males (aged 21 and 26 years old). All three females and the younger male had interpersonal conflicts with their parents, while the remaining male was unemployed. None of them took psychotropic drugs. Apart from the female who poured kerosene on herself before setting herself ablaze, the other four cases used non-ingestant chemical agents namely caustic soda, battery water, gamallin-20 and insecticide.

Discussion

Our results support some of Eferakeya's (1984) previous observations that a large proportion of DSH are students under the age of 30, and that males predominate. This is in contrast to findings in developed countries. However, the DSH incidence rate of 2.6 per 100 000 found in this study falls within the range previously reported. A more extensive epidemiologic study is therefore required to test the hypothesis that DSH is on the increase in Nigeria.

In addition, this is the first prospective study of DSH in this part of the world for 20 years. The strong association found between DSH and the diagnosis of acute reaction to stress in females has not been previously reported and one of the contributing factors is the diminishing social and emotional support for women in the face of rapid urbanisation, severe socio-economic constraints and cultural changes.

The prospective design of this study gave us the opportunity to explore the motives for deliberate self-harm. Disordered interpersonal relationships, especially between the patient and a significant person in his/her life stand out prominently as a causal factor. Such significant persons were invariably parents, spouses or very close associates

TABLE I
Methods of deliberate self-harm

Methods	Male	Female	Total	Percentage (%)
Non-ingestant Chemical				
Battery water	6	—	6	15.4
Gamallin 20	4	2	6	15.4
Insecticides	2	2	4	10.3
Kerosene	1	1	2	5.1
Caustic soda	1	1	2	5.1
Other chemicals	2 ¹	2 ²	4	10.3
Subtotal	16	8	24	61.6
Drugs				
Diazepam	3	2	5	12.8
Phenobarbitone	—	2	2	5.1
Other drugs	2 ³	3 ⁴	5	12.8
Subtotal	5	7	12	30.7
Physical	2	1	3	7.7
Total	23	16	39	100

1. Detergent: 1, concentrated acid: 1

2. Paraquat: 1, dettol: 1

3. Ephedrine: 1, clinitest: 1

4. Flunitrazepam: 1, piriton: 1, alcohol: 1

such as boyfriends. This finding agrees with the observation that distress drives people to self-poisoning acts and that distress is not the exclusive preserve of the mentally ill (Kessel, 1965). Morgan *et al* (1975) also found a high degree of psychological distress in the cases of attempted suicide that he studied. From our present report, acute stress reactions, with feelings of hostility and hopelessness rather than specific psychiatric disorders seem to account for most of the acts. This supports current reports of the association of hostility with suicidal attempts (Weissman *et al*, 1973; Hetzel, 1971; Dyer & Kreitman, 1984).

The finding of a high ratio of fatal to non-fatal DSH (1:7:8) is considerably higher than the ratio of 1:47 reported by Eferakeya (1984). In this study, 66.7% of the subjects used non-ingestant chemicals as opposed to 20.4% in Eferakeya's study; in addition four of the five subjects who died, ingested chemicals. The high fatality can be accounted for by the method employed and inadequate medical resources for resuscitation and management of complications after the attempt. Most of our patients committed their attempts impulsively. None of them had made previous attempts and factors related to high suicidal intent (Gelder *et al*, 1984) were absent apart from the predominant use of dangerous methods.

It is noteworthy that 50% of our cohort were first borns. Though the small size of the population would not permit a categorical statement, it is our impression that the adult role which young persons are expected to play prematurely in the Nigerian culture (such as being responsible for the stressful socio-economic and moral needs of their younger siblings) contribute to the sense of failure and frustration when not able to cope with these demands. This observation supports Cochrane's (1975) view of the role of stress in the lives of DSH patients.

One of the implications of the findings and observations reported in this paper is the need for further research into the epidemiology of DSH in Nigeria with a view to identifying factors that contribute to its apparent increasing prevalence. It is also essential that we develop better methods of preventing it and reducing the high fatal outcome.

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Comparison of the Nutritional State of New and Long-term Patients in a Psychogeriatric Unit

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Nutritional deficiencies have been implicated in the pathophysiology of various mental disorders. Life in Institutions has been associated with development of nutritional deficiencies, the elderly being most at risk especially those with senile dementia. In this study nutritional status, particularly vitamin and essential mineral status was assessed in a group of 64 new admissions to a psychogeriatric unit and 49 long-term patients.

The results indicated that psychogeriatric patients were at risk of experiencing nutritional deficiencies and those with senile dementia were likely to have more abnormalities than those with functional illnesses. Those patients in long-term care would appear to have a better nutritional state than those on admission.

Nutritional deficiencies have been implicated in the pathophysiology of various mental disorders. Hunter *et al* (1967) have reported two cases of patients presenting with psychiatric disorders, one with depressive symptoms, the other with psychotic symptoms and personality change, who had low levels of vitamin B₁₂ which when treated resulted in a marked improvement in their mental state. Thiamine (vitamin B₁) deficiency has long been associated with Wernicke's encephalopathy which if untreated may lead to Korsakoff's psychosis. More recently Carney *et al* (1982) have associated affective changes with vitamins B₂ and B₆ deficiency.

Folic acid has long been known to be associated with psychiatric disorders. Strachan & Henderson (1967) described an improvement in the mental state of two patients with dementia following folic acid treatment and indeed Reynolds (1967) has suggested that many of the symptoms of dementia may be the result of folic acid deficiency rather than the dementia causing the folic acid deficiency. More recently studies have shown a high incidence of folic acid deficiency in geriatric patients (Sneath *et al*, 1973, Vir & Love 1979) and those with senile dementia are particularly at risk (Sneath *et al*, 1973).

Studies on both acute and chronic psychiatric patients have indicated that zinc deficiency is associated with senile dementia, schizophrenia and chronic alcohol abuse (Srinivasan, 1984) and other trace elements such as vanadium, lead and aluminium are topical areas of research. Institutional care has been implicated as a cause of malnutrition and has been highlighted by Anderson (1968) and Bender (1984).

Thus three risk factors which predispose to malnutrition are:

- (1) the elderly
- (2) those with senile dementia
- (3) those in long-term care.

It was therefore the purpose of this study to assess the nutritional status of new admissions to a psychogeriatric unit, looking for any differences between those suffering from dementia and those with functional illnesses and to compare them with long-term patients.

Method

Group 1 consisted of 64 new admissions, median age 78 years (range 65-92 years). They were divided into those with functional illnesses, Group 1a (*n*=21) and those with dementia, Group 1b (*n*=43). Group 2 consisted of 49 long-term patients, median age 79 years (range 67-91 years) who had been in hospital for a minimum of 1 year, median 4.5 years (range 1-52 years). Group 2 was also divided into those with functional illnesses, Group 2a (*n*=12) and those with dementia, Group 2b (*n*=37).

Blood from each patient was assessed within 72 h of admission for serum albumin, transferrin, calcium, phosphate, magnesium, iron, zinc, copper, vitamins A, E, C, B₁₂, folate and red cell folate. Vitamins B₁, B₂ and B₆ status was assessed by red cell enzyme studies. Other routine haematological and biochemical studies were also carried out. Reference normal values were derived from 100 elective admissions for orthopaedic surgery, with no evidence of organic disease or nutritional deficiency. Where age was found to affect the reference range, reference values were derived from 40 patients aged 51-80 (median 62). Groups were compared using Wilcoxon Rank Sum Tests for non-paired data and χ^2 analyses.

TABLE I
Vitamin and mineral results in Groups 1 and 2

	Reference range	Group 1			Group 2		
		n	median	range	n	median	range
Vitamin A	1.0–3.5 $\mu\text{mol/litre}$	63	1.8	0.9–3.8	48	2.2	1.1–6.4**
Vitamin E	14–39 $\mu\text{mol/litre}$	63	26	15–47	48	23	9–48
Vitamin C	11.4–114 $\mu\text{mol/litre}$	55	18	9–90	38	20	9–76
Vitamin B ₁	< 25% activation	39	13	1–37	49	8	1–25**
Vitamin B ₂	< 60% RBC	58	15	1–110	49	10	1–54
Vitamin B ₆	< 150% enzyme ¹	58	63	4–311	49	47	3–100**
Vitamin B ₁₂	150–730 $\mu\text{g/ml}$	42	289	38–836	47	340	99–600
Serum folate	2.2–11.4 ng/ml	42	3.4	1.4–11.6	48	3.5	1.6–30
Red cell folate	106–614 ng/ml	44	169	76–795	47	201	85–708*
Mg	0.7–1.0 mmol/litre	60	0.85	0.67–1.08	49	0.84	0.67–1.0
Zn	12–18 $\mu\text{mol/litre}$	61	12.5	9.5–18.5	48	13.2	9.5–20
Cu	11–24 $\mu\text{mol/litre}$	61	22.5	13.5–31	48	20.5	10–31.5**
Fe	9–32 $\mu\text{mol/litre}$	59	12.5	3–29.5	42	14.5	2–27.5

* $P < 0.02$

** $P < 0.002$

1. High levels of activation of rbc enzymes are indicative of poor vitamin status.

Results

Comparison of Group 1 (acute) with Group 2 (chronic)

Significant differences were found between the two groups with respect to vitamin B₁ and B₆ status, red cell folate, serum retinol, vitamin E and copper. The results are shown in Table I. With the exception of copper, the results indicate poorer status in Group 1.

No significant differences were observed between the groups with respect to vitamin B₂ status or serum vitamin B₁₂, folate, vitamin C, magnesium, zinc, iron, albumin or transferrin.

There was also a significant difference in the number of patients in each group who had abnormalities of their vitamin or essential mineral status with Group 1 having more patients with abnormal results.

In Group 1 abnormally low values were found in 7 out of 58 patients for vitamin B₂ status, 13 out of 55 patients for serum vitamin C, 17 out of 61 patients for serum zinc and 13 out of 59 patients for serum iron. In Group 2 the respective frequencies were 0 out of 49, 5 out of 38, 10 out of 48 and 6 out of 42 patients. The difference in frequency for vitamin B₂ status was significant ($P < 0.01$, χ^2 test).

Comparison of Groups 1a (acute functional), 1b (acute dementia), 2a (chronic functional) and 2b (chronic dementia)

There was no significant difference in the number of patients in each subgroup with abnormally low values although the trend was for the dementia subgroups to have more patients with abnormally low values. Thus the percentage of patients in each subgroup with one or more abnormalities was 57% in Group 1a, 79% in Group 1b, 33% in Group 2a and 62% in Group 2b. The mean number of abnormalities for each patient was 1.28 in Group 1a, 1.8 in Group 1b, 0.67 in Group 2a and 1.08 in Group 2b.

Comparison of Groups 1a (acute functional) and 1b (acute dementia)

On admission Group 1b had significantly lower serum concentrations of zinc and vitamin B₁₂ than Group 1a. Also in Group 1b abnormally low values were found in 12 out of 39 patients for serum iron, 14 out of 40 patients for serum zinc, 7 out of 35 patients for serum vitamin C and 6 out of 39 patients for vitamin B₂ status. The respective frequencies in Group 1a were 1 out of 20, 3 out of 21, 6 out of 20 and 1 out of 19 patients. The difference in frequency for serum iron was significant ($P < 0.02$, χ^2 test).

Comparison of Groups 2a (chronic functional) with 2b (chronic dementia)

There was no significant difference between Groups 2a and 2b with respect to their vitamin and essential mineral status but there was a significantly higher number of patients in Group 2b (10 out of 36 patients compared with 0 out of 12) who had abnormally low values for serum iron ($P < 0.05$, χ^2 test).

Comparison of Groups 1a (acute functional) with 2a (chronic functional)

Comparing Group 1a with Group 2a, significantly more patients in Group 1a had abnormally low vitamin B₁ status (3 out of 11 patients compared with 0 out of 12). Otherwise there were no differences between the groups.

Comparison of Groups 1b (acute dementia) with 2b (chronic dementia)

Group 2b had significantly higher levels of vitamins A, B₁, B₂, B₁₂, and red cell folate than Group 1b as shown in



TABLE II
Significant differences between acute and long stay demented patients

	Group 1b			Group 2b		
	n	median	range	n	median	range
Vitamin A ($\mu\text{mol/litre}$)	42	1.7	1-3.2	36	2.1	1.1-6.4*
Vitamin B ₁ (% activation)	28	13	1-27	37	7	1-25*
Vitamin B ₂ (% activation)	39	15	1-110	37	10	1-54**
Vitamin B ₁₂ ($\mu\text{g/ml}$)	30	247	38-480	35	313	99-600**
Red cell folate (ng/ml)	30	176	76-795	35	201	106-708**

* $P < 0.02$

** $P < 0.05$

Table II. Group 1b also had significantly more patients with abnormally low vitamin C levels (7 out of 35 patients compared with 1 out of 27, $P < 0.05$, χ^2 test).

Folic acid, vitamin C, vitamin B₂ and vitamin B₆ results

The incidence of low serum folic acid in Group 1 was 12% with the dementia group having an incidence of 10%. There was no significant difference between the dementia group and the functionally ill group. In Group 2 the incidence was 8.5%, all the patients being in the dementia group.

The incidence of vitamin C deficiency was 24% in Group 1 and 13% in Group 2. In both groups the functionally ill had a higher incidence of vitamin C deficiency. With respect to vitamins B₂ and B₆, deficiencies were found only in Group 1 but there was no significant difference between Groups 1a and 1b.

Discussion

This study has shown that there are significant differences in nutritional status between new admissions to a psychogeriatric unit and those already in long-term care. Also, demented patients within both groups tend to have significantly more abnormalities, with those in long-term care having better status than those on admission.

Significantly more patients on admission had abnormally low values for vitamins and essential minerals with 57% of those diagnosed as functionally ill and 79% of those suffering from dementia showing abnormalities. For those in long-term care the incidence was 33% and 62% respectively but the mean number of abnormalities per patient was much less being 0.67 for the functionally ill in long-term care compared with 1.28 for those on admission and 1.08 for those with dementia in long-term care compared with 1.8 for those on admission.

This finding that patients with dementia are more at risk of nutritional deficiencies is in agreement with the work of Sneath *et al* (1973) who found that demented elderly patients were more at risk of folic

acid deficiency than elderly patients suffering from other illnesses. The incidence of 8.5% with folic acid deficiency, in long-term patients is much lower than the 25% incidence found by Vir & Love (1979) in institutionalised elderly patients. These lower values perhaps indicate that the diet of patients living in the East End of Glasgow is better nutritionally than in some comparable areas. Patients in long-term care would, however, appear to improve their folic acid status on hospitalisation.

New admissions were more at risk of having lower vitamin B₁ and B₆ status and serum vitamin A levels, and also more at risk of having abnormally low vitamin B₂ status than long-term patients. No relationship was found between those diagnosed as functionally ill and their B₂ and B₆ status, as suggested by Carney *et al* (1982). New admissions with dementia however, had significantly lower values for serum zinc and vitamin B₁₂ and were more at risk of having abnormally low serum iron than new admissions with functional illnesses.

A lower serum zinc in dementia patients has been noted by other authors and is reviewed by Tiggelen (1983).

In this study many patients were found, on admission, to be deficient in vitamin B₂, serum vitamin C, zinc and iron. The incidences were 12%, 24%, 28% and 22% respectively. The corresponding values for those in long-term care were 0%, 13%, 21% and 16%. With respect to the vitamin C levels, these figures are again lower than those quoted by other authors; 57% in senile dementia patients (Shaw *et al*, 1984), 29.2% in institutionalised elderly patients (Vir & Love 1979) and 34% of all new admissions to a psychogeriatric unit (Hancock *et al*, 1985).

Conclusions

This study has shown that psychogeriatric patients, especially those with senile dementia are at risk of nutritional deficiencies. In view of the evidence

linking such deficiencies with mental disorders these may well contribute to the psychological morbidity of this group.

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A Survey of Practising Psychiatrists' Views on Treatment of the Depressions

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The views of practising psychiatrists on treatment of the depressions were investigated as part of a Quality Assurance Project. A one in six random sample of all Australian psychiatrists was mailed a questionnaire. This asked for treatment recommendations for each of five case descriptions of patients with depression. Respondents were asked to code their treatment plans from a glossary listing possible treatments for depression: 85% of the sample responded. Tricyclic antidepressants were the treatment of choice for two cases of endogenous depression, electroconvulsive therapy (ECT) being recommended when psychotic features were present or when drug therapy had failed. Psychotherapies were the treatment of choice for cases with neurotic features, drugs being recommended when improvement with psychotherapy did not occur.

The Australian Quality Assurance Project (QAP, 1982b) has prepared treatment outlines for the commoner psychiatric conditions: agoraphobia (QAP, 1982a), the depressions (QAP, 1983), the schizophrenias (QAP, 1984), and anxiety states, obsessional compulsive disorder and somatoform disorder (QAP, 1985a,b,c).

Three sources of information were used: the results of controlled trials, reports from a sample of practising psychiatrists, and the opinions of psychiatrists believed to be expert in the treatment of the disorder under study.

Information from the literature is limited as, almost without exception, single treatments only are explored, while the opinions of experts may be limited by personal bias. Information about treatment practices of experienced psychiatrists is potentially of value for their management plans make it clear that seldom are single treatments used in isolation, while the sampling procedure should limit the effect of personal bias.

This paper is a report of a sample of practising psychiatrists' views about the treatment of five persons suffering from various depressive syndromes.

Method

The problems confronting those who would survey the treatment methods of practising clinicians have been discussed by Hall *et al* (1982). These authors used a questionnaire to investigate the views of practising psychiatrists on the treatment of agoraphobia. The questionnaire employed two strategies. The first required respondents to describe treatment plans for each of three

agoraphobic patients described in vignette. The three cases varied in treatment history and personality characteristics. The second strategy had practitioners select preferred treatments for 'agoraphobia' from a comprehensive list, and note indications and contraindications for each of the recommended treatments.

Not surprisingly, the strategies employed by Hall and his colleagues were found to produce different response biases. Hall concluded that a combination of the two approaches, including a glossary of treatment options, would have ensured more "... agreement on terms between those who ask and those who answer the questions: an advantage that would probably offset any bias produced in the responses." In the present study it was decided to persist with the questionnaire method of survey, but to include the glossary and certain other modifications that appeared desirable from the experience of Hall and his colleagues.

The questionnaire

The questionnaire included five case descriptions of patients referred to a psychiatrist by general practitioners for the treatment of depression. Attached to the questionnaire was a glossary of 54 possible treatments, defined where necessary. Inclusion of the glossary of treatments meant that respondents could code their own answers to the questionnaire by using the numbers identifying treatments (for example: '01' specified the tricyclic antidepressants; '34' specified 'psychotherapy, supportive/counselling'). The five case descriptions were based on patients for whom long-term follow-up data were available. Diagnoses were given, using the ICD-9 and DSM-III classifications, and were to be regarded as correct by respondents. The histories were chosen to illustrate a broad spectrum of depressive illnesses. For mnemonic convenience the five cases have been identified in the text as follows: Case 1—ED (endogenous depression); Case 2—PD (psychotic depression); Case 3—ND (neurotic

depression); Case 4—ED/ND; Case 5—Adj Rn (adjustment reaction).

Following each case history were rows of coding boxes in which the clinician could specify his treatment plan using the code numbers from the glossary of treatments. Treatment components introduced at the same time were able to be distinguished from those introduced later in the therapy sequence. Space was provided following coding boxes for each respondent to describe details of treatments identified by particular code numbers, for example: [01] "Amitriptyline 75 mg nocte. I would vary this dose depending on patient's tolerance, but with probable eventual aim of 150 mg nocte." [34] "Supportive psychotherapy to aid coping with practical difficulties: side-effects of medication, family concerns etc. Initially twice per week plus telephone contact and/or extra consultations as necessary."

Seven questions were asked about each case history. They were the location of treatment, therapy plan, critical component of that plan, extra historical information required, duration of therapy before discharge to general practitioner if therapy successful, duration of therapy before choosing an alternative treatment plan if therapy unsuccessful, alternative treatment plan. Data will be presented as percentage of respondents making particular recommendations, and for the treatment duration items as medians and semi-interquartile ranges.

The sample

The 1154 psychiatrists practising in Australia in January 1981 were identified from the list of College members, from the Department of Health list of psychiatrists in private practice, and from a commercial mailing list verified using the Medical Directory of Australia (1980). A one in six random sample stratified by state was chosen and approached to participate in the surveys associated with this project. Following exclusion of refusers and persons overseas, 185 practitioners were approached to complete a structured questionnaire on the treatment of the depressive states: 157 codeable responses were received (85% response rate).

Data coding and analysis

Only those coding boxes in which an average of more than 1% of the sample had made responses were analysed. For each case, individual therapies were summed across respondents irrespective of in which box the therapy had originally been coded, to give the total number of respondents employing a particular therapy (say, tricyclic antidepressants). Therapies employed in a particular case are thus presented for all respondents.

Results

Sample characteristics

The present sample is virtually identical with that surveyed by Hall *et al* (1982). Since these authors have presented demographic and other data in some detail, this information is not duplicated here.

Questionnaire results:

Case vignettes and treatment recommendations

Case 1: Vignette

Mrs A is a 47-year-old deserted wife with a 19-year-old son and a 15-year-old daughter. Her husband left her 7 years ago and since then she has coped well bringing up her children. She was described by her brother as 'normally bright and lively . . . gentle, home loving, quite a capable person'.

Over the last year she has become increasingly depressed and agitated. She has lost interest in her home, feels that her actions are slowed, and has great difficulty in doing any housework. She has started to "worry about everything . . . the children . . . their future . . . the financial part of it . . . about accidents, about death". She feels that she is a failure, that she is lazy and unworthy, and suspects that in some way she has brought the illness upon herself. She says that things feel changed, unreal ". . . as if I am in another world". She is anorexic, has a dry mouth and has lost 1.5 stone in weight over the last few months. She sleeps very patchily, wakes at 3 a.m. and gets up and paces the floor. Her depression is at its most severe during these early hours.

Mrs A's mother suffers from recurrent endogenous depressions and has had numerous admissions to psychiatric units; on one occasion she cut her throat in an attempt at suicide. Mrs A's oldest brother (she is the youngest of four sibs) died some years ago in a car accident. He is said to have been "heading for a nervous breakdown . . . depressed . . . talking about driving into another car . . ."

Mrs A was referred to you for treatment by her general practitioner. She had been taken to see him by her brother whom she had telephoned after taking a small overdose of sleeping tablets. She has not previously been referred for psychiatric treatment nor has she experienced any previous depressive episodes. At interview she was preoccupied with her depression but realised that she was ill and was grateful at the prospect of effective treatment.

DIAGNOSTIC CLASSIFICATION:

ICD-9 [296.1] Manic-depressive psychosis, depressed type.

DSM-III [296.23] Major depression, single episode, with melancholia.

Recommendations

Seventy-two per cent of respondents felt this woman should be treated as a voluntary inpatient, a further 1.3% noting that her stay in hospital should be involuntary if necessary; 21% considered that she should be treated as an outpatient.

Almost 99% recommended antidepressant drugs (tricyclics 92.4%, tetracyclics 4.5% and MAOIs 1.9%) which smaller numbers combined with other physical treatment methods including electroconvulsive therapy (ECT) (17%), neuroleptics (14%), and benzodiazepines as anxiolytics (11%) or hypnotics (19%). Essentially all respondents recommended some form of psychotherapy: individual, group, or family, as a concurrent or later element of therapy.

Therapy for this patient was advanced with a great deal of confidence, only 7.6% of the sample failing to advocate a particular therapeutic element as critical. More than eight out of ten saw antidepressants as "most critical" (tricyclics 79%, tetracyclics 3.2%); the remainder mentioned ECT 4.5%, some form of psychotherapy (total for glossary codes 32-35) 2.4%, referral to inpatient unit for treatment 1.3%, lithium 0.6% and family/marital therapy 0.6%.

Therapy occupied a median of 12.2 h (semi-interquartile range [SIQR] 5.3 h) of the psychiatrists' time. If therapy proved unsuccessful an alternative plan of management was considered after 3.5 weeks (SIQR 0.9). At this stage 69% said that they would introduce a new treatment plan, 21% continued on an essentially similar plan, 5% said that they would refer for a second opinion and 3% that they would refer the patient on for further management.

Of those who proposed a new treatment plan, all included ECT; the only other marked differences from originally proposed patterns of treatment were increased proportions recommending MAOIs (20%) and tetracyclics (11%).

Case 2: Vignette

Mr B is a 32-year-old immigrant of British stock. The sixth child in a working class family of ten children, he left school at 14 to work in a textile factory. As a child, he said, he had been shy and very close to his parents, both of whom are alive and well. He continued working in the same factory until at the age of 19 he decided to emigrate to Australia "because of the greater opportunities".

On arrival in Australia he lived in a hostel for the first 12 months, working as a surgical dresser in a large hospital. Several months after his arrival he developed a serious depressive illness which was successfully treated with ECT. A similar illness recurred 2 years later but was treated, he thinks, with "pills and injections". He remembers little of either illness "... looking back they just seem like bad dreams".

He subsequently enrolled in and completed nursing training and has been working as a registered general nurse, a job which he enjoys, for several years. He remains a quiet self-effacing man given to periods of self-doubt. His life has improved over the past 5 years since the advent of a steady girlfriend with whom he now lives. "Her confidence and energy are important to me." They plan to marry and "start raising a family" in about a year's time.

Five weeks ago, without apparent cause, Mr B again started to become depressed. He lost interest in his work, in his girlfriend, and in life generally. He had no appetite and had lost more than a stone in the last month. He was waking after only 2 hours' sleep. He became convinced that his body was "an empty shell ... that nothing exists ... the world and all the people in it are doomed".

Mr B was referred for treatment by his general practitioner whom he had seen at his girlfriend's insistence. On initial interview he was withdrawn, retarded, and preoccupied with his delusions.

DIAGNOSTIC CLASSIFICATION

- | | | |
|---------|----------|--|
| ICD-9 | [296.1] | Manic-depressive psychosis, depressed type. |
| DSM-III | [296.34] | Major depression, recurrent, with psychotic features (mood congruent delusions). |

Recommendations

Eighty-eight per cent of respondents recommended that this man be treated as an inpatient (included in the 88% were 16% who felt that he should be held involuntarily, half of this latter group noting that involuntary status should be invoked only if necessary). Interestingly 9.6% suggested that they would manage this man as an outpatient; a further 1.3% suggested management as a day patient.

Physical methods of treatment predominated, nearly nine out of ten practitioners prescribing antidepressant medication (tricyclics 87.3%, tetracyclics 1.3%). These drugs were combined with ECT by slightly more than half (53.5%), and/or neuroleptics (phenothiazines 27.4%, butyrophenones 2.5%). Benzodiazepines were included as hypnotics by 17.8% and as anxiolytics by 5.1%. Lithium prophylaxis was initiated by 26.8%. As in Case 1, the majority of practitioners saw some form of psychotherapy as an important element of therapy, one in three considering that therapy for significant others or marital therapy should be included.

Respondents advanced treatment plans for this man with as much confidence as in Case 1, only 7.6% of the sample failing to label a particular element of therapy as 'critical'. Electroconvulsive therapy and tricyclics were proposed as critical elements approximately equally as often (42.7% and 41.4% respectively). Other treatments considered 'critical' were phenothiazines 2.5%, supportive psychotherapy 2.5%, referral for inpatient treatment 1.3%, brief analytically oriented psychotherapy 0.6%, family/marital therapy 0.6% and a period in hospital without treatment 0.6%.

Therapy occupied a median of 15.5 h (SIQR 9.7) of the psychiatrists' time. If unsuccessful the therapy was persisted with for a median of 3.5 weeks (SIQR 1.0) before a change in management was considered. At this stage 62% introduced a new treatment plan, 24% persisted with an essentially similar therapy, 11% asked for a second opinion and 2% referred the patient on for further treatment.

Of those psychiatrists who introduced a new treatment plan the most striking differences in therapy were the proportion now recommending ECT (93%), tetracyclics (12%), MAOIs (13%), phenothiazines (35%) and lithium (35%). There were reductions in the proportion including any form of psychotherapy as part of their new treatment plan.

Case 3: Vignette

Mrs C, an attractive 25-year-old, was the younger child and only daughter of a respected and "highly moralistic" father who was "active in many church organisations". Her mother was "soft, sensitive, too emotional".

At school she was an above average student, popular, but more so with boys than with girls. She enjoyed dancing and wanted to take it up as a career. Becoming pregnant at 15 she decided to conceal the pregnancy from her parents and left home, subsequently marrying the father. When this young man deserted her after the birth of a second child she took an overdose of nitrazepam. Following counselling, and with the support of her parents she began to achieve some acceptance of her situation.

Four years later she was working as a dancing teacher, supporting her two children, and living in a home unit which

she was paying off with her parents' help. She had a boyfriend she had been seeing for 1 year and hoped to marry. During the 4 years she had protracted periods of feeling "down in the dumps", separated by short periods of no more than a few months when she would feel like her "usual self". During the depressive times she felt tired and lacking in energy, but continued to work. Her dancing would give her less pleasure; and she would have occasional spells of weeping.

Three weeks before referral Mrs C again became depressed. She had been irritable with her children and was pessimistic about the future. At initial interview she was keen for help and the therapist was able to establish a good rapport. She said that she was depressed because her responsibilities were "... just too great". She compared herself unfavourably with married schoolfriends and said that she had "... always felt inadequate as a mother" ... "that things always go wrong for me no matter how hard I try". She had been referred for treatment by her general practitioner to whom she had gone after an episode of "uncontrollable crying" at work.

DIAGNOSTIC CLASSIFICATION:

ICD-9 [300.4] Neurotic depression.

DSM-III [300.40] Dysthymic disorder.

Recommendations

An overwhelming majority suggested that this lady be managed as an outpatient (96.8%); the remainder recommended voluntary inpatient (1.3%), day patient (0.6%) or were uncertain (1.3%).

The most commonly recommended therapies were the psychotherapies: psychoanalytically oriented (31.2%), brief psychoanalytically oriented (33.1%) and supportive (35.7%). "Other" psychotherapies were suggested by 10.8%, behaviour therapy by 7% and cognitive therapy by 6.4%. Counselling for significant others or family/marital therapy were advised by 18.4%. Antidepressants were included as an element of therapy by one in three, tricyclic antidepressants being favoured twice as often as MAOIs. Benzodiazepines were included as anxiolytics by 10.8% and as hypnotics by 3.8%.

In sharp contrast to the first two cases, less than two-thirds of the respondents were willing to label any particular element of therapy as 'critical' (34.4%, no recommendation). Curiously those psychiatrists recommending more intensive psychotherapies were less confident about labelling such therapies as 'critical' than those recommending supportive therapy. Those proposing psychoanalytically oriented therapy as 'critical' (12.1%) represented little more than one in three of those employing that therapy, brief psychoanalytically oriented therapy (15.3%) about half of those using that therapy, while supportive therapy (22.3%) approached two-thirds of those advocating its use. Other therapeutic methods labelled as 'critical' included cognitive therapy (5.1%), 'other' forms of psychotherapy (3.8%), family/marital therapy (1.3%), MAOIs (1.9%), tricyclics (1.3%), and each of the following 0.6%: psychoanalysis, hypnotherapy, 'other' group therapy and lithium.

A median of 29.6 h (SIQR 16.9) of the psychiatrists' time was anticipated as necessary for successful outcome. When

the initial treatment plan was unsuccessful, an alternative plan of management was considered after a median of 9.6 weeks (SIQR 3.4). At this stage 61% introduced new treatment, 15% continued essentially similar treatment, 12% sought a second opinion and 10% referred the patient on for further treatment.

Of those introducing new treatment, the most notable change was in the proportion who now advocated antidepressants: tricyclics (54%), MAOIs (25%) and tetracyclics (4%); other therapies more often proposed in the revised treatment plan included supportive psychotherapy (42%), cognitive therapy (10%), psychodynamically oriented group therapy (6%), 'other' group therapy (5%), hypnotherapy (6%) and psychoanalysis (3%).

Case 4: Vignette

Mr D, a 65-year-old solicitor, has been living alone in an inner city unit for the last 4 months.

His wife of 25 years died from asthma 9 years ago. After what seems to have been a natural mourning process, he continued his customary business, social and sporting life. A keen yachtsman, he is a popular but reserved member of his local club. Six months ago he began living with a woman he had been courting for 3 years with a view to marriage. She went to live with him in his original home, but this development was not a success. He became very anxious and the relationship broke up in a matter of weeks. In rather a panic Mr D sold this home and purchased his present unit. He says he made the decision to sell the family home because, "... she wanted to change everything that I was comfortable with ... it just wasn't right, and I'm not happy in the house any more."

Until this time Mr D feels that he led a "charmed life", with a "good marriage, loving and diligent children and enough time and money to enjoy the good things of life." He was the only child of a doting mother and a solicitor father who were very close. His mother died from heart failure when Mr D was 32 years old; his father died of pneumonia the next year. Mr D had successfully risen to full partnership in the family law firm.

In the last months he has lost interest in yachting and in his work. He has been avoiding living in his new unit, feeling terrified of being alone at night and complains of disturbing dreams and waking 1-2 hours earlier than his normal time. It seems that he can sleep better if staying with relatives or friends. In fact his daughter says that when staying with them, provided there are no outside demands, he is nearly his old self. He now laments his haste in selling the family home; he feels quite unable to make any decisions about his future, let alone act on them. He has begun to miss his wife again and says "it's as if she has just died", and "I really don't see any point in struggling on." He complains of persistent feelings of depression, of constant fatigue unrelated to effort, and feels he is only eating from habit.

He was referred to his GP by his married children with whom he was normally in frequent contact. They had become concerned at his withdrawn and pessimistic behaviour.

At interview he quickly developed rapport and made it clear that he felt that the psychiatrist could be of great help. He described himself as a "yacht without a rudder ... needing

direction and guidance." He appeared depressed, mildly anxious, talked freely and showed no psychotic phenomena or suicidal ideation.

DIAGNOSTIC CLASSIFICATION:

ICD-9 ? [300.4] Neurotic depression.

DSM-III ? [296.22] Major depression without melancholia.

Recommendations

Respondents advised treatment locations as follows: outpatient (85.4%), inpatient (8.3%), day patient and night patient each 1.9%, and uncertain 2.5%.

In the treatment recommendations for this clinically complex case psychotherapies and pharmacotherapies were given roughly equal weight. The most frequently proposed form of psychotherapy was supportive (59.2%); other psychotherapies included brief psychoanalytically oriented (29.9%), "other" (8.3%) and psychoanalytically oriented psychotherapy (5.7%). Counselling for significant others or family/marital therapy was recommended by 11.5%. Antidepressants were exhibited by 56.6% (tricyclics 47.1%, tetracyclics 5.7% and MAOIs 3.8%), and a benzodiazepine was included as an anxiolytic (22.9%) or hypnotic (17.8%).

Nonetheless psychotherapies were considered 'critical' very much more frequently than were pharmacotherapies. Psychotherapies proposed as critical included supportive (36.9%), brief psychoanalytically oriented (15.9%), 'other' (4.5%), psychoanalytically oriented (3.8%). Antidepressants were considered critical by 12.7% (tricyclics 10.2%, MAOIs 1.9%, tetracyclics 0.6%). The proportion of the sample unwilling to label any treatment as critical was 23.6%.

Therapy was expected to consume a median of 15.2 h (SIQR 4.9) of the psychiatrists' time, while unsuccessful therapy was persisted with for a median of 5.6 weeks (SIQR 2.2). New treatment was introduced by 61%, similar therapy was continued by 21%, a second opinion was sought by 12% and 5% referred the patient on for further treatment.

Of those proposing new treatment, the major change was in the proportion now advocating antidepressants, alone or in combination (tricyclics 79%, MAOIs 28%, tetracyclics 9%).

Case 5: Vignette

Miss E is a 20-year-old nurse in the last year of training. She was the oldest in a family of three and described her parents as "strict, religious and kind". She attended a convent school and was intending to matriculate when increasing arguments with her father made her decide to leave home and commence nursing at a Catholic hospital.

She enjoyed nursing and was well regarded by her superiors. She lived in the nurses' home until a year ago when she was persuaded to share a flat with friends. She enjoys this arrangement, since she has "far more freedom" than at home or in the nurses' home. She has had a steady boyfriend for the past three years. During the first two years the relationship was sexually conservative as befitted their religious background. Since moving into the flat it has become overtly sexual. She now feels that this change was unwise and that her boyfriend is losing interest in her.

A month ago she learnt that because of illness in the earlier part of her nursing career she will not be eligible to complete her finals with the rest of her class.

In the past month she has been withdrawn, unable to study at nights, sleeping poorly and not wanting to see anybody. She says, "I only find satisfaction in nursing my patients" and that nothing else seems to matter. She has become depressed, is often discovered weeping quietly and while she admits that she may be "blowing things out of all proportion", she is unable to behave differently.

Her flatmates took her to see her local GP after she had spent the weekend crying in her room. At interview she attributed her depression to her "unwise" sexual behaviour and to her pending separation from her classmates. She offered information readily and concluded by saying, "I'm usually so sensible, I don't understand why I can't cope!"

DIAGNOSTIC CLASSIFICATION:

ICD-9 [309.0] Adjustment Reaction, Brief Depression

DSM-III [309.00] Adjustment Disorder with Depressed Mood.

Recommendations

The majority of the practice sample (86.6%) recommended that this young woman be treated as an outpatient; of the remainder 8.9% thought that she should be treated as an inpatient, 1.3% as a day patient and 3.2% were uncertain.

Nearly all respondents recommended some form of psychotherapy, either as a concurrent or later element of therapy. These psychotherapies included brief psychoanalytically oriented (47.8%), supportive (47.1%), 'other' (7%) and psychoanalytically oriented (6.4%); some form of group therapy was proposed by 3.8%, and cognitive or behavioural therapy by 3.2%. Counselling for significant others or family/marital therapy was recommended by 8.3%. Pharmacotherapeutic treatments were advised sparingly, 19.1% suggesting tricyclics and 3.2% MAOIs, either alone or in combination with a benzodiazepine as an anxiolytic (17.2%) or hypnotic (15.3%).

The practice sample were least certain in this case about which element of treatment was most likely to be 'critical', 38.9% being unwilling to note as such any treatment. Of those who did propose 'critical' treatments, supportive psychotherapy (26.8%) was favoured slightly more often than brief psychoanalytically oriented therapy.

It was anticipated that a median of 14.6 h would be devoted to therapy; unsuccessful treatment was persisted with for a median of 5.8 weeks (SIQR 3.2). New treatment was introduced at this stage by 28%; 12% each referred Miss E for second opinion or further treatment.

Amongst those psychiatrists introducing new treatment, analytically oriented psychotherapies, individual (brief analytically oriented psychotherapy 60%, analytically oriented psychotherapy 24%) or group (psychodynamically oriented 10%), and/or antidepressants (tricyclics 50%, MAOIs 14%) were most often considered important.

General overview of questionnaire results

In this section a general overview of results, with an emphasis on cross-case comparisons, is presented for each

of the questions. For mnemonic convenience the five cases have been identified in the text as follows: Case 1—ED (endogenous depression); Case 2—PD (psychotic depression); Case 3—ND (neurotic depression); Case 4—ED/ND; Case 5—AdjRn (adjustment reaction).

Location of treatment

The most striking differences in recommendations for location of treatment were between those for patients with clearly major depressive illnesses (ED and PD) for whom a majority of psychiatrists favoured inpatient treatment, and those patients with other depressive illnesses, for whom a majority of psychiatrists preferred outpatient treatment. Involuntary admission emerged as a significant issue only in Case 2 (PD).

Therapy plan

Therapies employed by two or more percent of respondents (in at least one case) are given for all five cases in Table I; the results are presented as the percentage of all respondents who advised a particular therapy. Virtually all psychiatrists said they would use more than one treatment, ranging from 3.0 treatments for Case 1 to 1.8 for Case 5.

Critical treatment

Practitioners were asked which element of their treatment plan they considered 'most critical'. If, for example, a tricyclic antidepressant and 12 sessions of psychotherapy

had both been advised, which, if either, was considered more important?

A great variety of treatments were labelled as 'critical'. In the interests of clarity only those labelled by 5% or more have been listed. Where respondents were not prepared to label any treatment as 'critical', data have been recorded as 'no recommendation'.

'Critical treatments' for each case follow; percentages have been rounded to integers.

Case 1 (ED): tricyclic antidepressants 79%; ECT 5%; no recommendation 8%.

Case 2 (PD): tricyclic antidepressants 41%; ECT 43%; no recommendation 8%.

Case 3 (ND): psychoanalytically oriented psychotherapy 12%; brief psychotherapy 15%; supportive psychotherapy 22%; cognitive therapy 5%; no recommendation 34%.

Case 4 (ED/ND): tricyclic antidepressants 10%; brief psychotherapy 16%; supportive psychotherapy 37%; 'other' psychotherapy 5%; no recommendation 24%.

Case 5 (AdjRn): brief psychotherapy 24%; supportive psychotherapy 27%; no recommendation 39%.

Hours of treatment

This question enquired how many hours of the psychiatrist's time, assuming that the patient made progress, would be required from initial assessment to return of the patient to his or her general practitioner's care.

TABLE I
Percentage of respondents ($n = 157$) recommending a particular treatment as initial therapy, and, in parentheses, percentage of those advising a new treatment if first management plan did not result in improvement

Treatment	Case 1 (ED)	Case 2 (PD)	Case 3 (ND)	Case 4 (ED/ND)	Case 5 (AdjRn)
Tricyclic	93 (79)	87 (82)	20 (54)	47 (79)	19 (50)
Tetracyclic	5 (11)	1 (12)	1 (4)	6 (9)	0 (1)
MAOI	2 (20)	0 (13)	10 (25)	4 (28)	3 (14)
Phenothiazine	14 (10)	27 (35)	3 (2)	3 (3)	1 (3)
Butyrophenone	0 (0)	3 (3)	0 (0)	0 (0)	1 (1)
Benzodiazepine (as anxiolytic)	11 (7)	5 (5)	11 (15)	23 (14)	17 (14)
Benzodiazepine (as sedative)	19 (14)	18 (9)	4 (4)	18 (12)	15 (17)
Lithium	6 (6)	27 (35)	2 (5)	1 (0)	0 (0)
Vitamin	4 (0)	2 (2)	1 (0)	2 (0)	0 (0)
ECT	17 (100)	54 (93)	0 (0)	1 (0)	0 (0)
Analytically oriented psychotherapy	3 (1)	5 (1)	31 (33)	6 (8)	6 (24)
Brief analytically oriented psychotherapy	5 (2)	10 (3)	33 (20)	30 (21)	48 (60)
Psychotherapy, supportive/counselling	83 (70)	73 (53)	36 (42)	59 (59)	47 (49)
Psychotherapy, other	7 (6)	6 (3)	11 (7)	8 (4)	7 (8)
Counselling for others	10 (5)	20 (5)	2 (1)	6 (2)	5 (5)
Behaviour therapy	1 (1)	2 (2)	7 (6)	1 (2)	1 (3)
Cognitive therapy	3 (3)	2 (2)	6 (10)	3 (4)	2 (5)
Hypnotherapy	0 (0)	1 (0)	3 (6)	3 (2)	1 (7)
Group psychodynamically oriented therapy	2 (1)	1 (1)	3 (6)	1 (1)	2 (10)
Group therapy, other	2 (0)	1 (1)	1 (5)	1 (2)	0 (5)
Supportive group	5 (5)	4 (2)	6 (3)	3 (3)	2 (1)
Family/marital therapy	11 (10)	13 (7)	8 (9)	5 (11)	4 (8)

TABLE II
Percentage of respondents (n = 157) taking a particular course of action for each of the five cases when the first treatment plan had not resulted in improvement

<i>Course of action</i>	<i>Case 1 (ED)</i>	<i>Case 2 (PD)</i>	<i>Case 3 (ND)</i>	<i>Case 4 (ED/ND)</i>	<i>Case 5 (AdjRn)</i>
Refer for second opinion	5	11	12	12	12
Referral on	3	2	10	5	12
Similar treatment	21	24	15	21	28
New treatment	69	62	61	61	46

A plot of the responses produced, for each of the five cases, a complex multi-modal distribution with marked positive skew, (that is, while most respondents suggested relatively low figures for hours of treatment, decreasing numbers of respondents suggested progressively higher figures.

With the exception of ND (Case 3) which had a median of 30 h, results were markedly consistent, the median psychiatrist recommending 12–15 h for the other four cases with half of the sample considering between 7 and 20 h would be required for treatment.

Weeks of treatment

This question asked how long the psychiatrist would persist with treatment if the patient failed to improve adequately, before considering an alternative plan of management. The data are reported as for hours of treatment above.

For ED and PD (Cases 1 and 2, respectively) in which physical treatments predominated, alternative treatment was considered after a median of 3.5 weeks; for ND and AdjRn (Cases 3 and 5), in which various forms of psychotherapy were the most frequently recommended treatments, a new treatment was considered after a median of 10 weeks (ND) and of 6 weeks (AdjRn). For ED/ND (Case 4), in which physical and psychotherapeutic methods were recommended about as frequently, psychiatrists were led to consider an alternative treatment at a median of 6 weeks.

A subsidiary question inquired about which of four courses of action would then be followed: referral for second opinion (psychiatrist or other specialist), referral on (to another psychiatrist/psychiatric institution), employ continuing course of essentially similar treatment, or introduce a new treatment. Results are presented in Table II as percentages of all respondents advising a particular course of action (figures are rounded to integers).

New therapy plan

The treatments advised for patients who had not improved are given in Table I (figures in brackets): the data are presented as percentages of those who proposed new methods of management, treated on a case-by-case basis. As presented, the figures thus allow cross-case comparisons of revised treatment plans for difficult cases.

Discussion

There are a number of important questions that must be considered in using a questionnaire method of survey to obtain the views of practising clinicians on optimal treatments for any group of illnesses.

In the first place, how representative are the questionnaire responses likely to be of the whole population of clinicians? Since the group for which results are analysed represents a large (14%) random sample of the whole population of psychiatrists practising in Australia, and the response rate (85%) was high, sampling error of questionnaire responses is likely to be insignificantly low, at least in the case of the major therapies described. It seems reasonable to infer that on the whole responses of this sample would differ little from those of the population.

A major assumption in using this method of survey to establish the treatment recommendations of practising clinicians, is that the recommendations reflect their actual practice. The most apparent danger inherent in the method is that the recommendations may reflect the ideal rather than the real, given the exigencies of everyday practice. In the case of the depressions, this possibility is arguably of little practical significance. For a number of reasons it seems probable that for the great majority of respondents, treatment recommendations are likely to have been based in considerable measure on personal experience. Depression is amongst the most common of reasons for referral to a psychiatrist; the patients described in the questionnaire are typical of those seen and treated in everyday practice. The therapeutic modalities proposed are likely to have been widely used: most respondents will have been trained in their use, the treatments are readily available and, except perhaps in the case of ECT, special facilities or referral to a specialist unit are not required.

A second question of major importance is just how optimal are the therapeutic practices of experienced clinicians? Two major issues bear on this question. The first is whether the practice of the clinicians reflects awareness of those treatments established by

the experimental literature as effective. This topic is discussed below in the light of the experimental data. The second issue, an assumption of very considerable importance, particularly in the case of those therapies or combinations of therapies that have little or no experimental validation, is that actual practice is based on a sensitive monitoring of what does and does not work. To what extent do clinicians alter their practice in the light of experience; to what extent do practice habits persist in spite of experience? The present paper does not, of course, provide data which bear on this question.

Therapy plan

In discussing the proposed treatment plans a number of important related issues can also be discussed. With what degree of confidence do clinicians suggest particular treatments for the various patients? How well does their confidence accord with the findings of the experimental literature?

In answering the first question, we feel that the questionnaire item, 'Critical treatment', provides pertinent information. The extent to which clinicians were prepared to label, or not label, particular treatments as 'critical' for successful therapy of a given condition, would seem to provide information about their degree of confidence in the efficacy of treatments in that condition. In those depressions presumed to have an important biological element in their determination, ED (Case 1) and PD (Case 2), treatments were proposed with a great deal of confidence, only 8% of respondents being unprepared to label any treatment as 'critical'. In sharp contrast, in depressions in which psychological factors are commonly thought to play an important role, ND (Case 3) and AdjRn (Case 5), respondents were a great deal less certain about what did and did not work, 34% being unwilling to label any treatment as critical in ND, and 39% in AdjRn. In ED/ND (Case 4), in which symptoms were somewhat equivocal, with suggestions of both biological and psychological elements, 24% of respondents would not indicate a critical treatment.

How well then do treatments advised or, more importantly, described as 'critical', accord with the findings of the experimental literature for a given condition?

In those depressions of principal biological determination physical methods of treatment predominated. In uncomplicated ED (Case 1) and in PD (Case 2), antidepressants and ECT were commonly prescribed and labelled as 'critical'. Antidepressants were advised by essentially all respondents in ED and by nearly nine out of ten in PD, and were described as

'critical' by 82% and 41%, respectively. Electroconvulsive therapy was advocated by 17% in ED and 54% in PD, being seen as 'critical' by 5% and 43%, respectively. Neuroleptics were prescribed by 14% in ED and 30% in PD, but were seen as 'critical' only in PD (by 2.5%); benzodiazepines were prescribed by almost 30% and 23%, respectively. Although some form of psychotherapy and/or family/marital therapy was advised by nearly all respondents in both cases, it was rarely seen as 'critical'. There is on the whole a high degree of concordance between such treatment prescriptions and the treatment literature of endogenous and psychotic depressions: ECT and antidepressants, with the probable exception of the MAOIs, were found effective in these disorders in our review of the literature (Quality Assurance Project, 1983).

In the non-endogenous depressions, in which psychological determinants are presumed to be of major importance, the psychotherapies were most commonly advised, combined with antidepressants by between one in two and one in five respondents. In ND (Case 3), psychoanalytically oriented, brief psychoanalytically oriented and supportive psychotherapies were each suggested by about one-third, and were seen as 'critical' by 12%, 15% and 22%, respectively. Brief psychotherapy and supportive psychotherapy were commonly advised in AdjRn (Case 5) (48% and 47%) and in ED/ND (Case 4) (30% and 59%), being considered 'critical' in AdjRn by 24% and 27%, and in ED/ND by 16% and 37%. Cognitive or behavioural therapy was suggested by 13% in ND and seen as 'critical' (cognitive therapy) by 5%. While advocated in all three cases, antidepressants were considered 'critical' only in ED/ND, by 10% (although prescribed in this case by more than 50%). Tricyclic antidepressants were prescribed by 20% in ND and AdjRn, and MAOIs by 10% in ND. The only other therapy suggested with any frequency was a benzodiazepine, advised by 15% in ND, by 32% in AdjRn and by 41% in ED/ND. In these non-endogenous depressions the degree of concordance between the literature and treatments recommended is not as high. Antidepressants are prescribed, and seen as 'critical' less frequently, and psychotherapies probably more frequently, than the literature would seem to support. It should be noted however, that with the exception of cognitive therapies, the literature is almost totally lacking in any controlled studies of verbal therapies in the depressions. Despite the apparent efficacy of cognitive therapy in neurotic depression (Quality Assurance Project, 1983), few clinicians advocated such therapy, presumably because the relatively recent development of such

therapies means that very few of the practice sample will have been trained in their use.

New therapy plan

In each of the depressive illnesses studied the major change in pattern of therapy, for those clinicians who proposed a revised treatment plan should their initial plan prove unsuccessful, was towards physical methods of treatment. In the endogenous depressions alternative antidepressants were often advised and all or almost all now used ECT. In the non-endogenous depressions antidepressants were introduced, usually by rather more than one in two of those who suggested a new therapy plan. Referral for second opinion or further treatment was recommended by up to one in four of the clinicians. No controlled studies of new treatments in short-term therapy failure are known to the authors; under such conditions of therapy failure the practice sample clearly indicated that alternative treatments of established effectiveness should be employed.

Conclusion

The Quality Assurance Project (QAP, 1982b) has published treatment outlines for agoraphobia (QAP, 1982a), the depressions (QAP, 1983) and the schizophrenias (QAP, 1984); it is now proceeding to publish outlines for the remainder of the anxiety disorders and the somatoform disorders.

In the depressive disorders in particular three points have become evident in the course of the project. The research literature is a reasonable guide to practice even though drug studies are still typified by short follow-up periods, and non-physical therapies, with the exception of cognitive and/or behavioural studies, by their rarity. The responses of the practice sample show a high level of agreement, with no evidence that Australian psychiatry in the 1980s is beset by factional rivalries. Lastly, the Quality Assurance Project by using these

sources of information, the research literature and the opinions of a practice sample, and the advice of elected experts can develop a consensus about the practice of psychiatry ca 1982. Such an achievement should benefit practitioners, hearten trainee psychiatrists, and form a springboard from which research into therapeutics can proceed.

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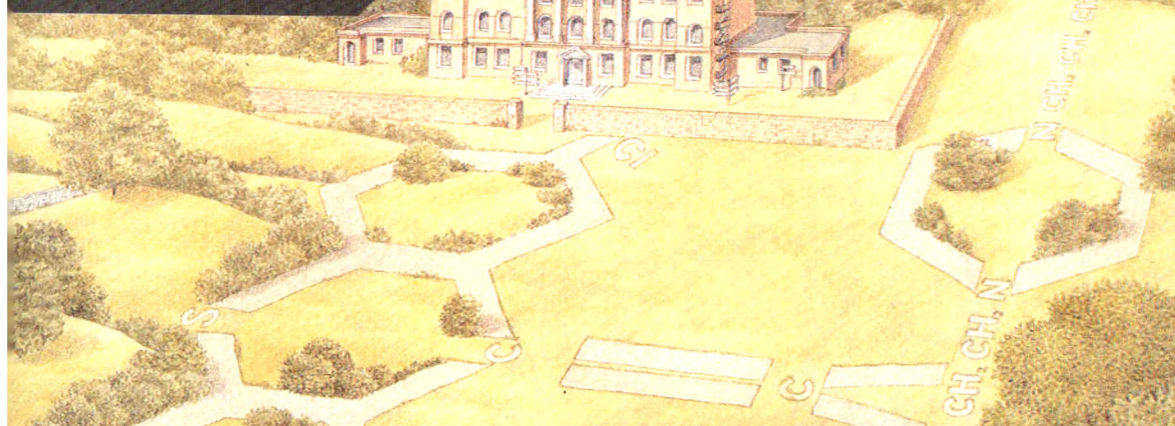
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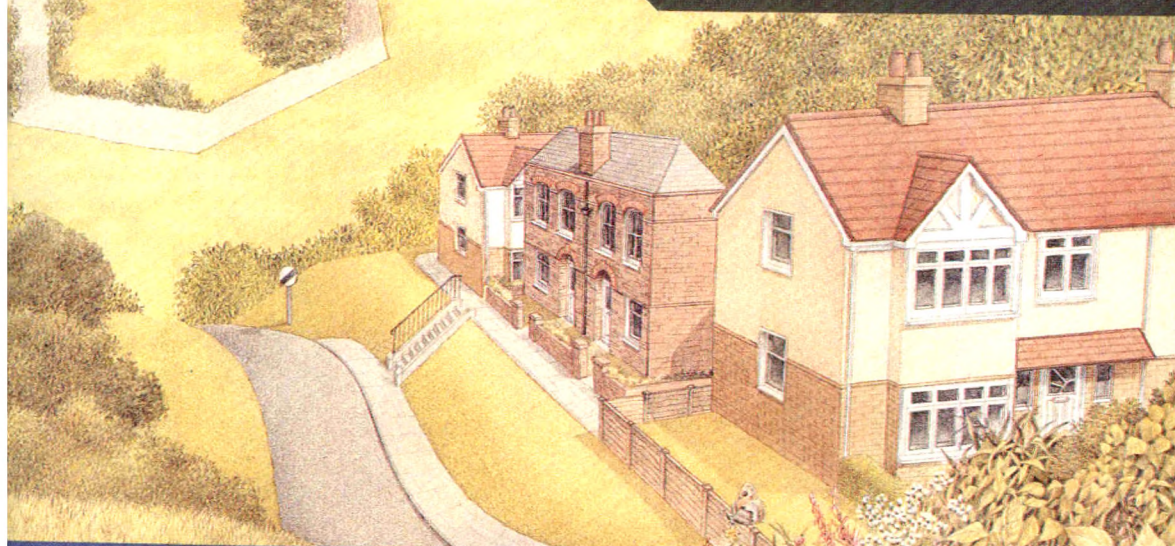
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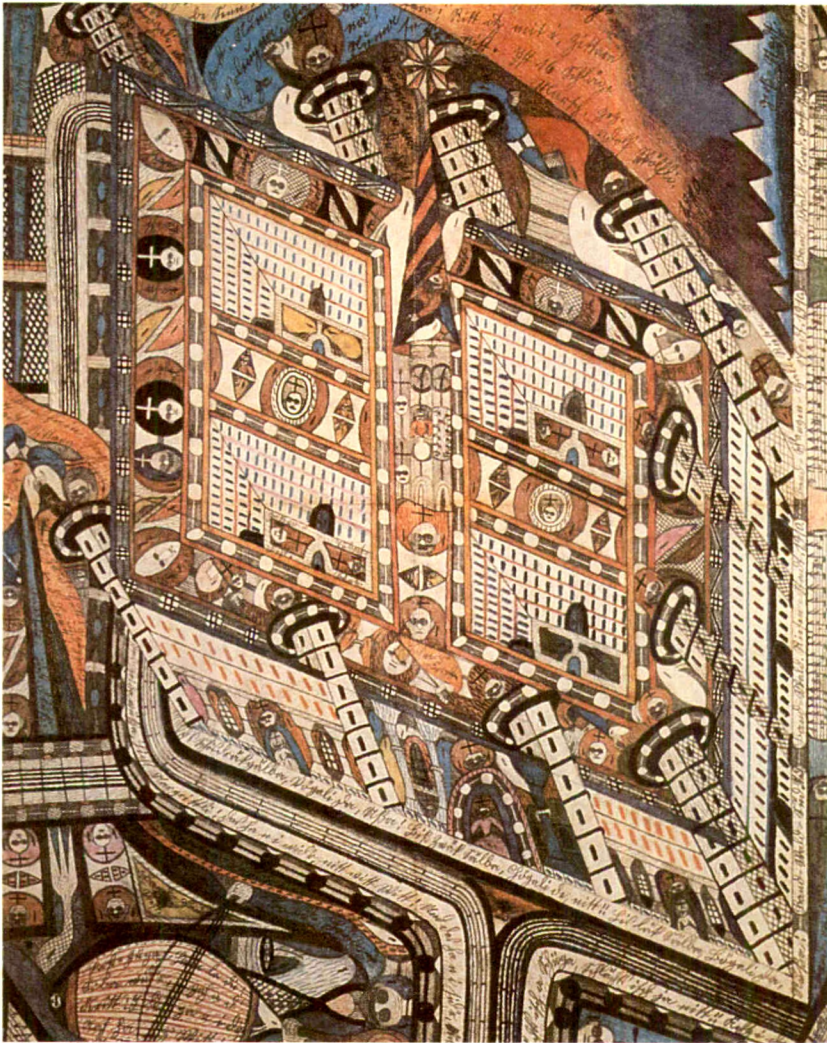
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The Effects of Weight Change on the Dexamethasone Suppression Test in Depressed and Anorexic Patients

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Prior studies on weight change and hypothalamic-pituitary-adrenal (HPA) axis functioning are reviewed. Data on 58 depressed and eight anorexic patients is presented. No significant difference in the frequency of cortisol non-suppression in the dexamethasone suppression test (DST) was found between depressed patients with a history of weight loss and those without, nor between depressed patients who lost weight during their first week in hospital and those who did not. Mean weight loss of suppressors did not significantly differ from that of non-suppressors. Of 12 patients whose DST normalised during their stay in hospital, only four gained weight. Five anorexics who were non-suppressors were < 70% of their ideal body weight (IBW), while three suppressor anorexics were $\geq 70\%$ IBW. These results indicate that mild to moderate weight change is not a significant influence on DST response in depression.

Studies of hypothalamic-pituitary-adrenal (HPA) axis functioning and its relationship to depression commenced in the 1960s (Sacher *et al*, 1970, Carroll, 1971). Early work concentrated firstly on urinary metabolites of cortisol and then on plasma cortisol levels. During the 1970s, dynamic studies of central HPA axis regulation became possible. In general, these studies have concluded that some depressed patients have altered HPA axis regulatory mechanisms similar to those found in Cushing's disease. These abnormalities include excessive cortisol secretion with increased nocturnal release, impaired responsiveness to lysine vasopressin and to hypoglycaemia and non-suppression of plasma cortisol following the administration of dexamethasone (Carroll *et al*, 1976). Due to its ease of application, the dexamethasone suppression test (DST) has been the most widely investigated test of these regulatory abnormalities and approximately 50% of depressed patients have been found to be non-suppressors of cortisol (Hirschfeld *et al*, 1983). However, the cause and mechanism of these HPA axis abnormalities remains unclear.

The relationship between weight change and alterations in HPA axis functioning has also been recognised. As weight loss is a common feature, it has been suggested that this factor alone might be responsible for non-suppression of cortisol in the DST (Edelstein *et al*, 1983). The purpose of this project was to examine this question in a systematic, prospective manner and to study several parameters

of weight change in depression in relation to the DST.

Background

The interaction of weight change and HPA axis functioning may be considered under five headings.

Fasting obese subjects

Galvao-Teles *et al* (1976), found significant increases in plasma unbound cortisol in a group of 13 fasting obese subjects. These changes were most marked at 2400 h, indicating a considerable diminution of the day-night variation of plasma unbound cortisol levels. Edelstein *et al*, (1983), studied 18 healthy, non-depressed overweight subjects before and after a protein-sparing fast. All patients received the DST and all suppressed cortisol normally before fasting. However, after 8-12 weeks of fasting, when subjects had lost an average of 14.3 kg, five (28%) of the subjects had become non-suppressors. There was no significant alteration in mood in these subjects. In both of these studies, patients were on stringent low calorie diets and lost substantial amounts of weight in a relatively brief period of time.

Fasting normal subjects

Berger *et al* (1984), studied 24 healthy volunteers who were placed on a 1000-1300 kilocalorie diet and who

lost an average of 1.5 kg/week. In nine (37.5%) of these subjects cortisol became non-suppressible to dexamethasone. Yerevanian *et al* (1984), studied 16 non-depressed volunteers and found that two (12.5%) converted from normal suppression to non-suppression after a period of strenuous dieting. Again, in both of these studies rigorous dieting was instituted resulting in sudden weight changes.

Starving subjects

Smith *et al* (1975), studied ten adult patients with protein-calorie malnutrition and found elevated levels of plasma cortisol, increased urinary excretion of cortisol, reduced metabolic clearance of cortisol, reduced plasma production rate of cortisol and non-suppression of cortisol following dexamethasone. Weight loss in all subjects was severe and several were in a state of inanition.

Anorexia nervosa patients

There are considerable data available (Weiner, 1977, Walsh, 1982) which indicate the frequent association of abnormalities of the HPA axis and anorexia nervosa. Anorexic patients have low urinary 17-OH corticosteroid levels, increased half-life of plasma cortisol, reduced metabolic clearance rate of cortisol, increased secretion rate of cortisol and elevated urinary free cortisol. Non-suppression of cortisol following dexamethasone has been found to occur in over 50% of anorexic patients (Gwirtsman & Gerner, 1981), approximating therefore the frequency in depression. These abnormalities of HPA axis functioning have been found mostly in the acute weight-loss stage of the illness when patients are less than 80% of their ideal body weight (IBW). They tend to normalise with response to treatment and gain in weight (Fichter *et al*, 1982), although this has been disputed (Gwirtsman & Gerner, 1981).

Depressed subjects with or without weight change

There have been a number of studies that have attempted to address specifically the question of the effect of weight change on the HPA axis in depressed patients. At present, results are conflicting. Carroll was the first to observe that 28 depressed subjects who had lost at least 3.2 kg in weight had significantly higher morning plasma cortisol levels than 12 subjects without such loss of weight (Carroll, 1971). Studies using the DST specifically have been of three types. One approach has been to examine the history for recorded data of recent weight change. Three studies of this type (Coppin *et al*, 1984;

Zimmerman *et al*, 1984; Yerevanian *et al*, 1984), found no relationship between a history of weight loss and DST response while two studies (Kline *et al*, 1983; Targum, 1983) both with small numbers of patients contradicted this finding. A sixth study (Feinberg & Carroll, 1984) found that weight loss was a significant variable but only when considered in isolation. When severity of the illness and the age of the patient were taken into account the history of weight loss was concluded to be an insignificant variable. These retrospective studies analysed case records, mostly by using the weight change item of the Hamilton depression rating scale or the Newcastle scale. These studies are therefore open to criticism because they are dependant on the reliability and details in documentation in the history and the care with which the weight change item had been assessed.

A second approach has been to measure weight change during the first week of a period in hospital and relate this to DST response. This is based on the assumption that such weight change will reflect and correlate with recent weight changes during the depressive episode and will therefore overcome the problem of historical inaccuracy. Two such studies (Feinberg & Carroll, 1984; Holsboer *et al*, 1984) both found no relationship between weight loss and DST response, while a third found weight loss to be a significant factor (Berger *et al*, 1983).

A third approach has been to monitor changes in DST response during the period in hospital, specifically the normalisation of the response. Only one (Targum, 1983) such study has been performed to date. This study found that three patients of ten whose DST failed to normalise during 6 weeks in hospital, gave a history of weight loss at admission.

Methods

Patients admitted to the Psychiatric Unit of The Royal Melbourne Hospital (RMH) were independently assessed by two psychiatrists. Those patients meeting DSM-III criteria for major depressive disorder (MDD) were included in the study. Carroll's exclusion criteria were applied (Carroll *et al*, 1981). All patients were assessed for severity of depression within the first few days of admission, using a Hamilton observer-rating scale and a Carroll self-rating scale. In addition to a careful history of recent weight change, all patients were weighed and their height was measured at admission. Wherever possible, family members were interviewed for further information concerning weight loss. During the first week (but not before the third day following admission) all patients received a standard DST with 1 mg of dexamethasone being administered at 2300 h and blood samples for serum cortisol being taken the same and following day at 1600 h. Plasma cortisol was analysed in the Department of Biochemistry (RMH) using the routine assay (Amerlex, Amersham, UK). Normal suppression was

defined as a post-dexamethasone serum cortisol concentration of less than 167 nmol/litre, a criterion established from a previous normal control study in the unit. All patients were re-assessed at weekly intervals during their period in hospital, with Hamilton and Carroll ratings, weighings and DSTs.

In addition, a group of anorexic patients have been studied. These patients were examined by two psychiatrists and all patients met DSM-III criteria for Anorexia Nervosa. Their assessments were similar to the depressed patients with weekly weighings, Hamilton and Carroll ratings, and standard DSTs.

As appropriate, χ^2 and *t*-tests were performed, for statistical analysis of the data. A $P > 0.05$ was used as the criterion for a non-significant (NS) result.

Results

History of recent weight loss

Of a total of 58 depressed patients studied, 29 were found to have had a recent history of significant weight loss (defined as more than 2 kg within the previous 2 months), while 29 gave no such history. Eleven of the 29 patients who had lost weight were non-suppressors of cortisol while nine of the 29 patients who had no history of weight loss were non-suppressors which represented no significant difference between the two groups (Table I). Furthermore, for the patients who had lost weight, the mean weight loss of the non-suppressors was 7 kg, while that of the suppressors was 5 kg. This difference was not significant. Also, both groups were only slightly below their mean ideal body weight (IBW), the mean for non-suppressors being 97% of their IBW and suppressors 95% of IBW.

TABLE I

Comparison of DST response, cortisol concentrations and severity of depression in the weight-loss¹ vs no weight-loss groups

	Weight-loss group (n = 29)	No weight-loss group (n = 29)	Statistics
Non-suppressors	11	9	$\chi^2 = 0.076$
Suppressors	18	20	NS
1600 h Pre-dexamethasone Cortisol (nmol/litre)	380 ± 191 ²	360 ± 122	$t = -0.47$ NS
1600 h Post-dexamethasone Cortisol (nmol/litre)	195 ± 213	138 ± 150	$t = -1.18$ NS
Δ Cortisol ³	196 ± 152	223 ± 154	$t = 0.66$ NS
Hamilton Depression Rating Score	28 ± 5	26 ± 5	$t = -1.64$ NS

1. Defined as ≥ 2 kg over the 2 months prior to admission

2. Mean ± SD.

3. Δ Cortisol = pre-dexamethasone cortisol - post-dexamethasone cortisol

TABLE II

DST response and weight loss during the first week in hospital (n = 49)

	Weight-loss ¹ group	No weight-loss group
Non-suppressors	11	9
Suppressors ²	11	18
	22	27

$\chi^2 = 0.79$

NS

1. Defined as > 0.5 kg

2. Data unavailable on nine patients — early discharge or transfer or weighings missed.

The weight-loss vs the no-weight-loss groups were compared with regard to mean plasma cortisol levels obtained from the initial DST during the first week in hospital. The mean pre-dexamethasone plasma cortisol concentration, mean post-dexamethasone plasma cortisol concentration and mean change in plasma cortisol concentrations from pre- to post-dexamethasone levels were not significantly different in these two groups (see Table I). Also, there was no significant difference in the severity of depression at admission, as measured by the Hamilton depression rating scale, between the weight-loss and no-weight-loss groups (see Table I).

Weight loss as measured during the first week of stay in hospital

Of the 49 depressed patients on whom relevant data were available, 22 were found to have lost 0.5 kg or more during their first week of stay in hospital. Twenty-seven depressed patients lost no weight or gained weight during their first week. There was no significant difference in the frequency of non-suppression in the weight-loss group vs the no-weight-loss group (see Table II). Furthermore, there was no significant difference in the mean weight loss of the non-suppressor group versus the suppressor group, approximately 2 kg in each group.

Weight changes during the course of stay in hospital

Among the 58 patients studied, there were 17 non-suppressors of cortisol who completed at least 3 weeks of inpatient treatment. Of these 17 patients, 12 converted to normal suppression during their inpatient stay (mean stay 5 weeks), while five remained non-suppressors. Of the twelve patients who converted to normal suppression only four (33%) gained weight during their stay in hospital. Seven of the remaining patients did not significantly alter their weight during their hospital stays while one patient actually lost weight.

Anorexic patients

Of eight anorexia nervosa patients who were studied, five were non-suppressors at admission, while three were normal suppressors. All five non-suppressors were 70% or less of

their IBW at admission, while the three suppressors were over 70% of their IBW. There was no apparent correlation between non-suppression and clinical assessment of depression or Hamilton depression rating score. Of the five non-suppressors, four converted to normal suppression after several weeks of inpatient treatment and did so when their weights exceeded 70% of their IBW. The one anorexic patient who remained a non-suppressor failed to respond to treatment or to gain weight.

Discussion

Studies of non-depressed groups including obese, malnourished and anorexic patients as well as normal subjects all indicate that weight loss is frequently associated with abnormalities in several tests of HPA axis functioning, including the DST. These studies suggest that either severe loss of weight or sudden substantial losses of weight will result in changes in HPA axis functioning. However, none of these groups are strictly comparable to a depressed patient population. Weight loss in depression in current clinical populations, is usually mild and gradual and only rarely severe and/or rapid. Amongst our patient group, the average weight loss reported by those patients who had lost weight prior to admission was 5.8 kg over the previous 2–3 months. There was only one patient who had lost more than 12 kg in weight and only one patient who was less than 70% of her IBW. In marked contrast in our anorexic group only those patients who were 70% or less of their IBWs were non-suppressors of the DST. Also, these patients converted to normal suppression when their weight exceeded 70% of their IBW.

The majority of the studies that have addressed the issue of weight loss in depression and the DST, conclude that weight loss is not a significant variable, whilst two studies contradict this finding. Most of these studies can be criticised from the point of view of retrospectivity and some because of low numbers and lack of definition of degree of weight loss. Although history of weight loss must by necessity be retrospective, we have attempted to overcome the problems of previous work which has largely relied on the weight change item of questionnaires. Our patients were carefully and directly questioned with regard to recent weight loss and whenever possible family members were interviewed. Also in our study, we have included a prospective and objective approach by measuring weight change weekly during the patients stay in hospital.

Our results support those prior studies which conclude that mild to moderate weight change is not a significant influence on the DST response in depression; DST response was not significantly linked to reported history of weight loss or weight

loss as measured during the first week in hospital. In addition, there were 12 patients in whom the DST response normalised with treatment during their stay in hospital and in the majority of these patients (67%) there was no weight change during the corresponding period.

The cut-off point for normal suppression, a plasma cortisol of 167 nmol/litre, had been determined from a previous study of 100 normal volunteers who had undergone a standard DST—and Carroll's recommendation of 5% of a normal population as being non-suppressors was used to calculate this value. In fact, when our data was re-examined using our former cut-off point of a plasma cortisol of 138 nmol/litre, the DST status of only one patient changed, making no significant difference to our results. Also, when our data was analysed using patients' plasma cortisol concentrations rather than the suppressor vs non-suppressor category we again found history of weight loss was not a significant variable.

Although, acute and severe losses of weight may influence HPA axis functioning and consequently the DST response, this is not usually the case in depressive illness. We conclude that weight change need only be taken into account when interpreting the DST response in depressed patients, when weight loss is unusually severe or rapid.

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Physical Illness A Risk Factor in Anorexia Nervosa

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Reports have appeared in the literature suggesting an association between physical illness and the later development of anorexia nervosa. The histories of 326 patients with anorexia nervosa were examined for physical illness. A higher rate of severe physical illness was found in this group when compared with controls suggesting that it should be regarded as a risk factor for anorexia nervosa. No statistically significant differences were found when illness was examined by time in relation to onset of anorexia, system involved, or when subjects were stratified by age. Possible explanations for the observed association are discussed.

Ryle (1936) first suggested that there was an association between a history of severe physical illness and the later development of anorexia nervosa. From his series of 49 anorexic patients, he identified an older group for whom operations and illnesses appeared to play a leading part in the predisposition to the condition. Dally (1969) found that for 38 of his series of 140 patients with anorexia nervosa, the patients' mothers considered that there had been undue physical ill-health during the daughters' life before the onset of anorexia nervosa. The disorders included asthma, tuberculosis, congenital blindness, squint and deafness following otitis media. Recently reports of cases of specific conditions coexistent with anorexia nervosa have appeared, for example, Turner's syndrome (Pitts & Guze, 1963), (Kron *et al*, 1977), urogenital abnormalities (Halmi & Rigas, 1973) diabetes mellitus (Fairbairn & Steel, 1980; Szmukler & Russell, 1983) and neurofibromatosis (Fitzpatrick *et al*, 1983).

Apart from physical illness being a cause of vulnerability to anorexia nervosa, it has been suggested (Dally 1969; Beaumont *et al*, 1978) that physical illness may precipitate the onset of anorexia nervosa. Physical illness was hypothesised to induce a physiological anorexia which subsequently developed into true anorexia nervosa. Head injury also has been cited as a precipitant (Damlouji & Ferguson, 1985) of anorexia nervosa.

In the setting of a large-scale epidemiological study it has been possible to examine the relationship between anorexia nervosa and physical illness. Two hypotheses were specifically examined:

- (1) that excessive physical illness earlier in life renders an individual vulnerable to the later

development of anorexia nervosa, i.e. should be considered among the risk factors for this condition;

- (2) that acute physical illness can act as a precipitant for anorexia nervosa in at least a subgroup of cases.

Method

Subjects

The subjects of this study were a group of patients fulfilling the criteria formulated by Russell (1970), for anorexia nervosa. This index group were patients who were referred to the Academic Department of Psychiatry at the Royal Free Hospital for the management of anorexia nervosa between 1972 and 1981.

They were compared with two control groups. The first control group was an age/sex matched population chosen at random from the lists of a large local general practice, which was chosen as the doctors routinely recorded details of past medical history when a patient was first seen. The practice served a population similar in social class to our index group. The second control group was female schizophrenic patients admitted to the psychiatric ward of the Royal Free between 1973 and 1983 and under the age of 35 years at the time of admission. This second control group was included because, unlike the first, the patients were referred to a psychiatric department and therefore would have had histories recorded in a similar manner to the index group.

Data collection

For the index group, coded data were derived from the history recorded at the patient's initial assessment interviews and for the schizophrenic control group data were similarly coded from the admission history. For the general practice

control group all relevant information until the time of case note scrutiny was recorded. The data coded included episodes of physical illness until the time of onset of anorexia nervosa or schizophrenia in the two psychiatric groups.

Illnesses were coded into two groups, 'distant' and 'recent'. Distant illnesses were those which developed more than 2 years before onset (or case note scrutiny in the general practice group), and recent illnesses were those which had occurred within 2 years preceding onset.

The criterion for inclusion as a 'distant' illness was that a definite diagnosis was made and that admission to hospital or treatment in a specialist outpatient clinic occurred. Distant illnesses were further subclassified into perinatal illness occurring in the first year of life, operative illnesses which had necessitated operation under general anaesthetic, non-operative illness and chronic illnesses, persistently present for more than 2 years before onset or case-note scrutiny.

The criterion for inclusion as a recent illness was broader, so that all illnesses which might bring about a significant physiological anorexia could be coded. All illnesses were included where it appeared from case-notes that the patient had taken more than 2 weeks off work or school. Thus, for example, glandular fever and severe influenza occurring within 2 years of onset were included even though no specialist treatment had been received. For further analysis, illnesses were classified again according to principal system involved. The fifteen subheadings are shown as part of Table III.

Results

Subjects

Data were collected for 326 patients with anorexia nervosa, 105 general practice controls and 61 schizophrenic controls. The mean age of onset of anorexia nervosa was 18.7 years (s.d. 6.5) and of schizophrenia 22.6 years (s.d. 6.2 years) (*t*-test $P < 0.01$). The mean age of the general practice control group at the time of case-note examination was 19.3 (s.d. 7.5 years), which was not significantly different from the index group.

Distribution of illnesses

Table I compares the number of physical illness episodes amongst the members of the three groups. The anorexic patients were significantly more likely to have had physical illness than both the control groups and are also more likely to have had several episodes than controls. ($\chi^2 P < 0.001$ in each case.)

Analysis of illness episodes

Analysis of episodes of illness in relation to onset (or case-note scrutiny) is shown in Table II. The index group tend to have more episodes of illness, both distant and recent, but the differences do not reach statistical significance when groups are compared on individual categories.

Analysis of episodes of illness according to system involved is shown in Table III. There were no significant differences when specific categories were compared between groups, but a trend for the anorexia group to have more episodes of illness in most categories is noticeable.

Alimentary vs non-alimentary illnesses

Subjects who had had at least one episode of illness in the anorexia nervosa group and general practice group were further examined by classifying illnesses into alimentary and non-alimentary categories. It was hoped thereby to examine whether illnesses which were more likely to have involved disturbances of eating and a physiological anorexia were more associated with the later development of anorexia nervosa. No statistically significant differences were found between the anorexia nervosa and general practice control groups in the ratio of non-alimentary to alimentary illness for either 'recent' or 'distant' illnesses.

Age of onset of anorexia and illness

Finally, an attempt was made to examine whether a history of undue physical illness might be associated with the development of anorexia nervosa in a particular age group as had been suggested by Ryle (1936). A comparison of the index group and general practice control group stratified by age of the subject showed an expected association of more 'distant' physical illness with age in both groups in the older age band. For all ages the excess of physical illness in the anorexia nervosa group reported in Table I occurred. The results did not suggest, however, that anorexia nervosa developing in an older age group was more likely to be associated with a history of undue physical illness.

Discussion

This study has attempted to examine the relationship between a history of physical illness and the later development of anorexia nervosa. Before discussing the findings it is important to examine methodological problems which may have influenced the findings obtained and conclusions which can be drawn.

TABLE I
A comparison of distribution of episodes of illnesses in index and control groups

Number of physical illnesses per individual	Anorexic patients <i>n</i> = 326	GP controls <i>n</i> = 105	Schizophrenia controls <i>n</i> = 61
	%	%	%
0	37	62	70
1	35	28	18
2	19	6	12
3	9	4	0

TABLE II
The analysis of 'distant' and 'recent' episodes of illness in index and control groups

<i>Illness episodes</i>	<i>Anorexia nervosa group n=326 %</i>	<i>GP Group n=105 %</i>	<i>Schizophrenia group n=61 %</i>
No illness	27	54	63
Perinatal	2	0	3
Non-operative >2 years ago	18	10	17
Operative >2 years ago	25	22	14
Chronic illness	6	2	0
Non-operative <2 years ago	13	4	0
Operative <2 years ago	9	8	3

TABLE III
Comparison of illness episodes classified by system affected between index and control group

<i>Illness episodes</i>	<i>Anorexia nervosa group n=326 %</i>	<i>GP group n=105 %</i>	<i>Schizophrenia group n=62 %</i>
No illness	26	56	63
Specific generalised	5	3	2
Non-specific generalised	3	1	0
Alimentary	12	6	6
Central nervous system	5	1	6
Endocrine	1	0	0
Ear, nose and throat	15	10	4
Ophthalmological	4	1	0
Dental	1	0	0
Urological	2	0	0
Cardiovascular	1	1	3
Respiratory	7	2	7
Musculoskeletal	6	5	0
O and G	5	7	7
Haematological	2	1	0
Dermatological and cosmetic	4	6	2

The subjects of this study were patients attending a tertiary referral centre for anorexia nervosa. As such they may be chronic, established or unusual cases not wholly representative of all anorexic cases. It is possible that physical illness is over-represented in those referrals. In contrast, the general practice control group was chosen to represent a normal sample of subjects matched for age, sex and similar in social class. However, subjects who had never visited their general practitioner were excluded, implying that this control group would be biased in favouring illness episodes, thus making it more difficult to observe any significant excess of physical illness in the anorexia group, if it existed.

A possible second source of error arises from the different methods of data recording between the hospital and general practice patients. The schizophrenia control group had been inpatients and therefore had histories similar in type to the index group. One problem is that physical illness as an alternative diagnosis is more likely in anorexia

nervosa and therefore may have been recorded more carefully in the anorexia nervosa subjects. This, however, is unlikely to have been an issue in relation to the general practice control group where in addition to routine recording of past medical history, a record of all attendances was kept. There is no reason to believe that clinical history-taking would have been different in the two different settings and so it seems unlikely that an illness of sufficient severity to be classified would have escaped recording in this second control group.

In chronic anorexia nervosa, difficulties may arise in determining accurately the date of onset of the eating disorder, thereby leading to difficulties in determining whether a physical illness preceded or postdated onset. As there was however no significant excess of alimentary vs non-alimentary episodes in the recent illness category, it seems unlikely that this has caused a significant bias.

The major findings of this study then is that there was an excess of physical illness episodes in the

backgrounds of subjects with anorexia nervosa, when compared with general practice and schizophrenia controls. Attempts were made to specify the characteristics of this excess of illness episodes. However, analysis of illness as classified by 'distant' or 'recent', or by system involved did not yield any statistically significant difference between index and control groups. No particular association for illnesses which cause a physiological anorexia was discerned. The attempt to evaluate the hypothesis that there is a particular relationship between physical illness and anorexia nervosa for the older age group found no evidence to substantiate this.

The first hypothesis suggested, that physical illness earlier in life renders an individual vulnerable to later anorexia nervosa, is supported by the findings of this study. While there is an excess of physical illness in the period immediately preceding the onset of anorexia nervosa when compared with controls this excess is not significantly more than that found when examining more distant illness. This suggests that a history of severe physical illness is a non-specific risk factor for anorexia nervosa rather than specifically a precipitant as suggested by the second hypothesis.

There are several possible explanations for this observed association between anorexia nervosa and past physical illness. The first possibility is that there is a direct aetiological relationship in a certain group of patients. This suggestion seems unlikely as one would expect to observe physical illnesses with specific characteristics being particularly linked to the development of anorexia nervosa. In this study no such specific characteristics were observed. A second possibility is that some other factor, possibly genetic, predisposes to the development of physical illness and to anorexia nervosa. Again this seems unlikely as one would expect to find some differentiation within the types of physical illness observed and a higher than expected prevalence of physical illness in the relatives of patients with anorexia nervosa. There is little evidence to support the latter suggestion. Halmi *et al* (1977) report that gastrointestinal disorders are not uncommon in the relatives of patients with anorexia nervosa, while Kalucy *et al* (1977) found maternal migraine to be common. These reports are of interest but do not constitute strong evidence for there being an excessive prevalence of physical illness in the families of anorexics.

A third possibility is that the observed association between physical illness and anorexia nervosa is

mediated through some other factor. Minuchin *et al* (1978) has suggested that relationships in the anorexic's family are characterised by over-protection and enmeshment. It does seem plausible that such a pattern of relationships might be initiated during an episode of serious illness. While it is possible that emotional disorders in such families may present as physical illness and be recalled as such, the explanation that illness triggers potentially pathological relationships within the family, seems the most plausible and warrants further investigation.

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Conduct Disorder and Hyperactivity: I Separation of Hyperactivity and Antisocial Conduct in British Child Psychiatric Patients

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The distinction between hyperactivity and conduct disorder was explored in a mixed group of 64 children referred to psychiatric clinics because of antisocial or disruptive behaviour. A semi-structured interview measure (the Parental Account of Children's Symptoms, PACS) proved to have adequate inter-rater reliability, internal consistency and factorial validity. The PACS scales of defiance and hyperactivity, and similar subscales from Conners' Teacher Rating Scale, were tested against laboratory and clinical measures of activity, attention, cognitive performance, psychosocial background and family relationships. The hyperactivity (but not the defiance) scales were associated with greater activity, younger age, poorer cognitive performance and abnormalities on a developmental neurological examination. The defiance (but not the hyperactivity) scales were associated with impairment of family relationships and adverse social factors. It was concluded that a dimension of inattentive, restless activity should be separated from one of antisocial, defiant conduct in children with psychiatric disorder.

Hyperactivity and conduct disorder are frequently present at the same time and in the same people. It is not even clear whether they are genuinely different problems. This paper examines the question.

North American writers on hyperactive children have made it clear that aggression and defiance are frequently the target symptoms for treatment rather than the core symptoms of poor attention and excessive movement (Safer & Allen, 1976; Barkley, 1981). Indeed, the third edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-III) states explicitly that non-compliance and bullying are part of the condition of 'attention deficit disorder with hyperactivity'—and, conversely, that many cases of 'conduct disorder' have hyperactivity symptoms (American Psychiatric Association, 1980). Stewart *et al* (1981) reported that two out of three hyperactive children had a conduct disorder as well.

A complementary situation exists in British practice. Hyperactivity is rarely diagnosed, conduct disorder frequently: but overactivity and restlessness are common among the conduct disordered. Thus, Taylor (1979) reported that Conners' Classroom Rating Scale scores of a group of children with the clinical diagnosis of conduct disorder were very similar to those of children diagnosed as hyperactive in the USA.

The extent to which the two problems overlap raises a real question about whether they are in fact distinct. Few studies have sought differences between the hyperactive and the conduct disordered; fewer still have found any; and none have found a sharp distinction between conditions (Sandberg, 1981; Taylor, 1985). Indeed, Quay's (1979) review of classification argued with authority against the use of hyperactivity as a valid construct for the description of behaviour problems. Loney *et al* (1978) found different implications of dimensional ratings of hyperactivity and of aggression; but the ratings were based upon chart records and in principle subject to bias. The issue has usually been examined in terms of categorical schemes of classification, based upon diagnostic rules for allotting children's disorders to one group or another. Such rules are bound to be somewhat arbitrary, inasmuch as most children with one behaviour show the other too. Even before establishing criteria for diagnoses, one needs to determine whether the behaviours (on which the rules are based) are functionally independent.

Accordingly, in this paper we ask whether a dimension of hyperactivity, separate from one of antisocial conduct, is valuable for describing children attending psychiatric clinics.

Method

Subjects

64 boys, aged from 6 to 10 years, were examined; all had been referred to psychiatric clinics in south London for treatment of conduct problems (including aggressive, antisocial and/or hyperactive behaviours). Three clinics co-operated in this study: two child guidance units and the children's outpatient department of a postgraduate psychiatric teaching hospital. The subjects also met the inclusion criteria of living in a family home (and not an institution), and attending normal state schools. All had IQs over 65, none had overt neurological disease or psychotic symptoms. None had had any psychotropic drugs prescribed in the preceding 6 months. They are further described in the section on 'Results'.

Measures

Parental account of childhood symptoms (PACS)

This standardised, semi-structured interview was developed as an instrument for the measurement of children's behaviour problems as seen at home. It is administered by a trained interviewer. Parents are asked, not for their ratings of problems, but for detailed descriptions of what their child has done in specified situations over the previous week. Such situations are defined either by external events—e.g. watching TV, reading a book or comic, playing alone, playing with friends, going to bed, travelling—or by behaviours shown—e.g. crying, worried talk, tempers, fighting with siblings. The interviewers then make their own ratings, on the basis of their training and written definitions of the behaviours to be rated, on a four-point scale of severity (0 to 3) and frequency in the previous week. The judgements of frequency and severity are made independently and according to written criteria. Scores on frequency and severity are then averaged to yield the score for each item.

The recall of the previous week's behaviour is then used as the basis for enquiry about the same situations in the previous year. Frequency, severity, age-inappropriateness and degree of handicap imposed are combined into a single four-point rating of problem severity for each item of behaviour.

In all, 44 items of behaviour are enquired about in this detailed way. Some, however, relate to rare symptoms (e.g. in sexual development) or to symptoms not directly relevant to this study. The remaining items are grouped into three subscales, each of which is averaged to give a metric score ranging from 0 to 3.

The subscales are:

- (1) *Hyperactivity*. This is made up of attention span (time spent on a single activity, rated separately for four different kinds of activity), restlessness (moving about during the same activities), fidgetiness (movements of parts of the body during the same activities) and activity level (rated for structured situations such as mealtimes and car journeys).

- (2) *Defiance*. This scale is composed of items concerning temper tantrums, lying, stealing, defiance, disobedience, truanting and destructiveness.
- (3) *Emotional disorder* is made up from items of misery, worrying, fears, apathy, hypochondriasis, and obsessionality. It therefore relates to overt emotional distress, not to inferences concerning the emotional basis of symptoms.

Classroom behaviour

This was measured with Conners' Teacher Rating Scale (Conners, 1969, 1973). Minor changes of wording were made to increase its acceptability to teachers in England. The modified questionnaire has acceptable reliability and stability over time; and normative values have been established in a normal school population in south London (Taylor & Sandberg, 1984). The scoring system was based on those normative values: factor scores were estimated from the standardised z-scores on those items loading on the 'hyperactivity' and 'defiance' factors, weighted by their factor score coefficients. The scale validly describes children's behaviour problems in classrooms (Sandberg *et al*, 1980; Schachar *et al*, unpublished m.s., 1984).

Psychiatric interview

The interview with the child was carried out in the standard format described by Rutter & Graham (1968). This yields a number of interviewers' ratings of psychiatric symptoms, each on a three-point scale. Factor analysis of the interview has indicated that the first factor to emerge is made up chiefly by the items of overactivity, attention span, distractibility, fidgetiness and social disinhibition (Luk, personal communication). These items were summed to give a scale of 'observed hyperactivity'.

Physical examination

This examination included a scored developmental neurological examination (Sandberg *et al*, 1978) and assessment of minor physical anomalies (Waldrop *et al*, 1968).

Psychological testing

The revised version of the Wechsler Intelligence Scale for Children (short form) was given to all children (WISC-R). In addition, a set of tests was given to measure abilities related to 'attention'. They are described in detail by Thorley *et al* (unpublished m.s., 1984). They included:

- (1) *A Continuous Performance Test (CPT)* of sustained attention (Erlenmeyer-Kimling & Cornblatt, 1978). In this vigilance paradigm, pictures representing playing cards are presented successively by a micro-computer on a TV screen; the child presses a button whenever a stimulus is identical to the one preceding it. The number of correct responses, false positives and false negatives are recorded, and non-parametric measures of observer sensitivity and observer criterion are calculated, based on signal detection theory (Pastore & Scheirer, 1974).

- (2) *A Paired Associate Learning Test* (PAL) (Swanson & Kinsbourne, 1976), in which a verbal response is learned to each of a set of visual stimuli. It is thought to reflect efficient use of strategies in short-term memory.
- (3) *The Porteus Mazes Test* (Porteus, 1947), which is sensitive to an impulsive and disorganised style of problem-solving.
- (4) *A test of Selective Listening for Digits* (SLD), in which one voice has to be listened to in the presence of another and subsequently recalled. This is intended to measure the selective 'filtering' of input on the basis of simple physical cues (Broadbent, 1971).

A composite scale of attention test performance was derived by summing the log- and z-transformed scores on the observer sensitivity measure from the CPT, the percentage success rate on the PAL, the age score from the Porteus Mazes, the selectiveness score from the SLD and the digit span from the same test. The justification for this procedure and the factor analysis of the test battery are described by Thorley *et al* (unpublished m.s., 1984).

Social background factors

These were assessed during the interview with the parents. Social class was defined by the Registrar-General's classification of occupations (OPCS 1970). Family size was defined by the number of children under 17 living in the household. The parental situation referred to the people with whom the child was currently living, and a 'broken home' was defined as any situation other than living with two natural or adoptive parents. 'Previous separations' referred to any spells of 1 month or longer when the child was living away from parents. Institutional care was defined as present when the child had spent any period of his life in a children's home, regardless of the reasons for this and the legal status of the stay.

Family relationships

Family relationships were assessed by standardised semi-structured interview methods based on those described by Quinton *et al* (1984).

Efficiency of parental coping with behaviour problems is asked about for a wide range of potential problems. The interviewer's questioning is directed to the style of coping, not to its outcome, and does not make assumptions as to which techniques are desirable. Rather, efficient responses to children's behaviour are defined as being tailored to the individual child; involving clear goals, a coherent plan and adequate follow-through; being flexible and modified in the light of events; and using pressure appropriate to the child's level of development. Ratings are made (after training) on an eight-point scale ranging from 1 (for very efficient) to 8 (for highly inappropriate responses).

Consistency between parents is separately rated, for each of the problem areas. The scale ranges from 0 (for no disagreements or countermanding) to 3 (for arguments in front of the child or mutual countermanding).

Ratings of expressed emotion are made for the mother's emotional tone when talking about the child. Warmth and criticism are separately rated on scales from 0 (for no warmth or criticism) to 5. The ratings are based on counts of positive and negative comments, content of speech and tone of voice, spontaneity of comments, sympathy for the child's position and interest in the child as a person.

Contact between parent and child is rated by the description of the previous week and all occasions when there were conversations between parent and child (regardless of content) or positive interactions, such as games, that are not merely incidental to child care. Frequency is rated from 0 (for none) to 3 (for interactions every day).

Quality of parents' marital relationship was assessed by the detailed interviewing scheme described by Brown & Rutter (1966). The scale ranges from 1 (for a marriage typified by mutual concern and affection with no long-lasting tension or important quarrels) to 6 (for one with absence of affection and negligible co-operation). The presence of open discord was separately recorded.

Procedure

When referrals were received at the clinics, subjects and parents were offered an immediate assessment by the research team, with the possibility of a trial of treatment in advance of the routine waiting list time for assessment and therapy. Only 83, out of 154 identified, accepted the offer; and a further 19 preferred not to proceed with the assessment after initial discussions. This high refusal rate should be seen as reflecting unfavourable public and media attitudes towards research and physical treatments, as well as the uncertainty of people referred to psychiatric clinics for the first time. Full informed consent was obtained from all those who accepted.

For those entering the study, parents were interviewed by a psychiatrist; the child was interviewed, and then physically examined, by a different psychiatrist; and the child was given psychometric tests by a clinical psychologist. Each assessor was blind to the other assessor's findings, and to other clinical information, until after his own ratings had been made. In 25 cases, the parents' interview was witnessed by an independent assessor, who made his own ratings as a check on the reliability of the technique. School reports and completion of rating scales were independently obtained. Medical records were requested whenever relevant.

After the assessment, the research team met for consensus decision on the five diagnostic axes of the multi-axial version of the 9th edition of the International Classification of Disease (Rutter *et al*, 1976). Diagnostic decisions were based upon all available information, except that the set of tests related to attention were not used as no normative data were available.

Results

Characteristics of sample

Since the self-selection of subjects for the study could have introduced a bias, this series is compared in Table I with

TABLE I
Comparison of subjects with previous clinical series

	<i>Present study</i> (n = 64)		<i>Previous series</i> (n = 68) (Sandberg et al 1978)	
	<i>Mean</i>	<i>(s.d.)</i>	<i>Mean</i>	<i>(s.d.)</i>
Age in years	8.6	(1.7)	8.2	(1.8)
IQ	96.4	(15.5)	96.6	(15.5)
Conners' Parent hyperactivity	10.9	(5.0)	10.7	(5.0)
Conners' Parent defiance	7.2	(5.3)	6.9	(5.0)
Conners' Teacher hyperactivity	11.2	(5.5)	10.4	(5.3)
Conners' Teacher defiance	12.5	(10.1)	12.9	(9.4)
Neurological examination	9.2	(8.1)	8.4	(7.7)
Social class: manual	<i>Percentage</i> 57		<i>Percentage</i> 73	
Broken home	41		33	
Marital discord	39		40	
> three children at home	39		43	

a previous series of consecutive cases from the outpatient department of the Maudsley Hospital (described by Sandberg *et al*, 1978). Age, IQ, neurological status and symptom levels on Conners' rating scales (using the scoring system given by Conners, 1973) were compared by *t*-tests

and showed no significant differences. Social factors, tested by χ^2 tests, did not differ between the two series. The present sample can be considered representative of boys referred for psychiatric help in this area.

The 90 boys who did not enter the study were not obviously different from those who did. Only limited information was available from initial referral sources. Their age was similar (8.4 years, s.d. 1.9); a similar number came from broken homes (35.6%); and a similar number of their referral letters mentioned overactivity or poor concentration as a specific problem (24.4%; by comparison with 26.6% of those entering the study).

Parental account of children's symptoms: reliability and factor structure

Product-moment correlations were computed for each pair of raters' scores on the scales from the PACS. These indicated an acceptably high level of agreement. For the hyperactivity scale (previous year) the correlations ranged from 0.92 to 0.95; for defiance, 0.89 to 0.95; for emotional disorder, 0.79 to 0.90. For efficiency of parental coping the range was 0.69 to 0.76; for expressed warmth, 0.78 to 0.91; for expressed criticism, 0.79 to 0.86; for father-child contact, 0.79 to 0.96; for mother-child contact, 0.81 to 0.95.

The internal consistency of the scales for behaviour in the previous year was also acceptably good. Cronbach's α was 0.89 for the hyperactivity scale, 0.87 for defiance.

Fifteen of the behavioural items were selected for a factor analysis on the basis of being relevant to the main question of the study (so that developmental and somatic symptoms were omitted), being reliably rated by two interviewers, and having a 0 score present in not less than 10% and not more than 90% of cases. They were then submitted to principal factor analysis with iteration (as maintained in the SPSS

TABLE II
Rotated factor loadings for PACS items of previous year

<i>Symptom</i>	<i>Factor 1</i>	<i>Factor 2</i>	<i>Factor 3</i>	<i>Factor 4</i>	<i>Factor 5</i>
Misery	0.25	0.14	0.09	<u>0.52</u>	-0.09
Apathy	-0.02	0.03	-0.07	<u>0.11</u>	<u>0.42</u>
Specific fears	-0.01	0.26	-0.11	<u>0.56</u>	<u>0.34</u>
Worrying	-0.01	0.01	-0.01	<u>0.63</u>	0.21
Obsessional behaviour	0.27	-0.01	-0.07	<u>0.09</u>	<u>0.76</u>
Lying	-0.10	0.15	<u>0.69</u>	0.00	-0.21
Stealing	0.05	0.14	<u>0.86</u>	-0.01	0.00
Tantrums	0.19	<u>0.65</u>	0.12	0.00	-0.10
Defiance	0.16	<u>0.69</u>	0.12	0.17	0.20
Disobedience	0.21	<u>0.68</u>	0.16	0.23	0.03
Attention span	0.59	0.21	-0.06	0.01	0.18
Restlessness	<u>0.83</u>	0.14	0.12	0.00	0.05
Fidgeting	<u>0.82</u>	0.23	-0.11	0.25	-0.20
Activity level	<u>0.63</u>	0.46	0.02	0.00	0.00
Accident-prone	<u>0.44</u>	0.06	-0.04	0.08	0.25
Percentage of variance	46.8%	20.9%	15.4%	9.2%	7.7%

Underlined figures show loadings greater than 0.40

TABLE III
Product-moment correlations of behaviour scales and overactivity measures

	PACS hyperactivity	Classroom hyperactivity	Observed hyperactivity	Observed number of gross body movements	Observed number of fine body movements
PACS hyperactivity	—	0.39*	0.56*	0.28*	0.22
PACS defiance	0.41*	0.09	0.20	-0.01	-0.02
Classroom hyperactivity	0.39*	—	0.54*	0.43*	0.48*
Classroom defiance	0.11	0.49*	0.06	-0.04	0.08

* $P < 0.05$

program package) and rotated according to varimax criteria. Table II shows the solution for the previous-year ratings.

The first factor is clearly one of restless, inattentive, impulsive behaviour ('hyperactivity'). The second is one of non-compliance and tempers ('defiance'), which emerges separately from Factor 3 ('antisocial'), made up of the behaviours of lying and stealing. Emotional symptoms load separately on to Factor 4 ('affective') and Factor 5 ('obsessional'). The item of activity level was split between hyperactivity and defiance factors. Since the orthogonality of factors could not be assumed *a priori*, oblique rotations were also taken. The same number of factors emerged, with the same pattern of variables loading on them: the only differences were in the order of emergence of the factors (and, of course, the exact values of factor loadings).

This analysis gave some support to the distinction between hyperactivity and defiance as separate dimensions of behavioural symptoms. The pattern of factors was very similar for the previous-week ratings: the same factors appeared in the same order. Fuller results on the reliability and factorial validity of the scales are available from the authors.

Home and classroom behaviour and observed activity level

Table III shows the correlations between the previous-year PACS ratings and the Conners teacher rating scales; and between these and the rating of hyperactivity made by the psychiatrist and the observations during psychological testing in the laboratory. Both the teacher and the parent hyperactivity scales significantly predicted the independent clinic measures of overactivity in interview and motor activity during testing. Neither of the defiance scales was associated with laboratory measures of activity.

Home and classroom behaviour and chronological age

There was a significant inverse correlation between the child's age and his PACS hyperactivity ($r = -0.32$; $P < 0.05$) and classroom hyperactivity rating ($r = -0.24$; $P < 0.05$). By contrast, there was no association between age and defiance as measured by the PACS ($r = -0.11$, NS) or by the Conners teacher scale ($r = 0.12$, NS).

Age has therefore been taken into account in the following analyses by the statistical method of partial correlation.

Home and classroom behaviour and developmental measures

Product-moment correlations were substantial between parent hyperactivity and attentional performance ($r = -0.54$; $P < 0.001$) and between teacher hyperactivity and attentional performance ($r = -0.47$; $P < 0.001$); but non-significant between defiance scales and attentional performance. Table IV sets out the partial correlations between behaviour ratings on the one hand; and the full-scale IQ, the attentional performance scale, and the developmental neurological examination on the other hand. In each case, age has been partialled out.

TABLE IV
Partial correlations between behaviour ratings and developmental measures, after allowing for age

	IQ (WISC-R)	Attentional performance	Neurological examination
<i>For all children</i>			
PACS hyperactivity	-0.28*	-0.27*	0.32*
PACS defiance	-0.06	-0.16	0.05
Classroom hyperactivity	-0.42*	-0.29*	0.47*
Classroom defiance	-0.06	0.00	0.05
<i>For those with IQs more than 80</i>			
PACS hyperactivity	-0.30*	-0.26*	0.31*
PACS defiance	-0.17	-0.19	0.09
Classroom hyperactivity	-0.47*	-0.31*	0.50*
Classroom defiance	-0.16	-0.03	0.12

* $P < 0.05$

Correlations, though small, remained significant between the hyperactivity scales and the three test measures, and remained insignificant for the defiance scales. When both age and IQ were partialled out, few significant correlations remained between any of the behavioural scales and the developmental measures. However, the partial correlation between the PACS measure of home hyperactivity and the attentional performance scale was just significant at the 0.05 level ($r = -0.23$) even after both age and IQ had been partialled out.

These results were not the consequence solely of including some children with dull intelligence. Table IV shows that, when the eight children with IQs of less than 80 are excluded,

TABLE V
Partial correlations between behaviour measures and family relationships

	Parental coping	Parental inconsistency	Father-child contact	Mother-child contact	Warmth by mother	Criticism by mother	Marriage quality
PACS hyperactivity	0.10	0.17	0.47*	0.14	-0.16	0.18	-0.09
PACS defiance	0.42*	0.45*	0.19	-0.03	-0.38*	0.40*	0.16
Classroom hyperactivity	0.02	-0.02	0.15	-0.19	0.05	-0.01	-0.12
Classroom defiance	0.13	0.05	0.08	-0.32*	-0.07	0.07	-0.20

* $P < 0.05$

the associations between hyperactivity and impaired cognitive performance and coordination remained positive and specific.

The distinction between dimensions of hyperactivity and defiance was paralleled by that between diagnoses of hyperkinesis and conduct disorder in the ICD 9 scheme. The mean IQ for the seven with hyperkinetic syndrome was 79.7 (s.d. 10.1); for the 42 boys with conduct disorder 95.8 (s.d. 13.1); for the 15 with other diagnoses 108.5 (s.d. 15.5); analysis of covariance with age as covariate indicated a significant difference ($F = 7.6$; $P < 0.05$). The mean scores on the attention performance scale were -4.24 (s.d. 2.16) for the hyperkinetic, -0.04 (s.d. 1.88) for the conduct-disordered, and 1.29 (s.d. 1.36) for the others ($F = 21.4$; $P < 0.01$). The attentional differences remained significant when both age and IQ were entered as covariates ($F = 5.3$; $P < 0.01$).

The children's behavioural scores were also broken down by the presence or absence of background factors relevant to developmental delay. Neither an abnormal obstetric history, nor a history of delay in the developmental milestones of language, mobility and continence, nor the presence of minor congenital anomalies proved to be associated with the level of any behaviour problem.

Behavioural measures and family relationships

The same measures of hyperactivity and defiance at home and at school are related in Table V to the scales of parental

efficiency in coping, consistency between parents, contact between parent and child in the previous week, expressed warmth and criticism by mother, and quality of parents' marital relationship. The measure of association is the partial correlation coefficient after controlling for chronological age. Defiance at home was associated with inefficient coping strategies by parents, inconsistency between parents, less expressed warmth and more expressed criticism. Defiance in the classroom was associated with less contact between mother and child at home. The measures of hyperactivity did not show this pattern of associations: the only significant finding was an *increased* contact between father and child (which may, of course, have been initiated by the child).

Behavioural measures and social factors

The frequency of some background social factors was described in Table I. Table VI presents the means of the behavioural measures for groups differing on these background social factors and also on the country of birth of parents. No significant differences were found on the home measures, nor the hyperactivity measures at school. The only differences were on the teachers' ratings of defiance, where greater severity was associated with lower socio-economic status and with having a parent born in the West Indies.

TABLE VI
Behaviour ratings and social factors

	Parental situation			No. of children			Social class				Parents birthplace				
	Family intact	Broken home		≤2	3	≥4	I & II	III	IV & V			UK	WI	Other	
PACS hyperactivity	0.9	1.0	NS	1.0	0.8	0.9	NS	1.1	0.8	1.0	NS	1.0	0.9	0.7	NS
PACS defiance	1.3	1.4	NS	1.2	1.6	1.5	NS	1.3	1.2	1.6	NS	1.4	1.1	1.4	NS
Classroom hyperactivity	1.5	1.7	NS	1.7	1.1	1.9	NS	1.9	1.4	1.7	NS	1.6	1.9	1.0	NS
Classroom defiance	1.3	1.5	NS	1.2	1.1	2.0	NS	1.3	1.0	2.3	*	1.1	2.3	1.2	*

* $P < 0.05$

NS = not significant.

Adverse events were noted in the history of a number of the children: 14 had been separated for more than a month from one parent only, 19 from both parents. The behaviour scales of the children who had undergone separations were no different from those who had not, when tested by analysis of covariance using age as covariate. Eleven children had had periods of institutional care, and their home behaviour was no different from the others. However, their hyperactivity in classroom (mean 2.3) was significantly greater than that of the others (mean 1.4). Full details are available on request.

Discussion

Is hyperactivity different from antisocial conduct?

The results above give some support to a distinction between two dimensions of disruptive behaviour problems. The classification of symptoms by factor analysis suggests that—at least in this kind of clinical series—items of restless, inattentive behaviour are associated with one another more than with non-compliant, antisocial conduct. However, for such a separation to mean anything it must be capable of predicting relationships with variables other than those which defined the dimensions in the first place. Furthermore, the factor analysis by itself is not conclusive: it is based upon a relatively small number of cases and the number of variables was correspondingly restricted. The key item of activity level was in fact split between the factor we have labelled 'hyperactivity' and that of 'defiance'.

Accordingly, construct validity needed to be tested from a network of potentially important associated variables. The most obvious of these were similar behaviours in different settings. The point is not only that hyperactivity at home showed a modest correlation with that in school, or that defiance did the same; the apparent absence of links between hyperactivity in one situation and defiance in another is as important. Similarly, the lack of a clear association between direct observation of activity and ratings of defiance is as important as the presence of an association with hyperactivity ratings at home and at school. It should, of course, be no surprise that children rated as overactive should turn out to make more movements. Nonetheless, the finding is not a tautology but an approach to validating the concept. There is a distinct pattern of associations with other variables—hyperactivity being related most strongly to neuropsychological test results, and defiance to measures of interaction between family members. The negative results, as well as the positive, suggest that these two constructs have some discriminative validity.

Some possible objections to this conclusion need to be considered. The first is that the preconceptions of the researchers could have led to halo effects and contamination of one measure by another. This certainly cannot explain all the results. Pains were taken to ensure the independence of the different sources of information. The parental measures, the teachers' ratings, the psychiatrist's ratings and the direct observations were all made in ignorance of the results from the others. However, on a few variables there is a chance of contamination: most notably, the measures of family life and relationships were not blind to the accounts of behaviour at home. We do not think that our preconceptions account even for this aspect of the distinction between hyperactivity and defiance, since our original view of hyperactivity—based on pilot studies—was as a non-specific and situation-dependent aspect of conduct disorder. The repeated checks on inter-rater reliability would also have worked against such an artefact.

A second possible objection comes from our choice of a heterogeneous group of boys as subjects. It may indeed be that the associations of hyperactivity with other variables would appear much weaker if we had confined our study to a narrow range of high scores on the hyperactivity measures. But that would not imply that the present findings are in any sense an artefact. Certainly the results cannot be attributed to the group's heterogeneity in IQ scores: they are unchanged by the exclusion of those with low IQ.

Another possible reservation comes from the series being composed exclusively of clinic attenders. Since several centres were involved, we anticipate that the results will be generalisable to other psychiatric settings. We do not assume that the distinction made on clinic attenders will necessarily apply to other groups, such as normal schoolchildren, or girls, or intellectually retarded children, or older or younger children with behaviour disorder. More research will be needed on these populations.

A previous pilot study did not show agreement between different measures of activity in different settings (Sandberg *et al*, 1978). However, the present study should be seen as extending rather than contradicting those findings. It has used more developed measures, and associations may well have been obscured by error variance in less satisfactory instruments. The few positive findings of the previous paper are replicated here: the chief of these was the association between teacher ratings of hyperactivity and a low IQ. While the associations between hyperactive behaviour in different settings are now statistically significant, they are still rather modest

in size. In general, less than a quarter of the variance of hyperactivity in any one situation can be accounted for by other hyperactivity measures. Situational factors are important too.

Is hyperactivity based on attention deficit?

'Attention deficit' can mean a set of behaviours such as frequent changes of activity and impersistence. It can also refer to an impairment of central processing manifest in psychometric tests. In the first sense, attention deficit is clearly bound closely together with overactivity in this population. In the second sense, there appears to be an association between hyperactivity and poor scores on the attentional scale. However, the association between hyperactivity and IQ is just as large; and when IQ is also allowed for, attention deficit is only weakly related to behavioural hyperactivity. The implication is that hyperactivity is indeed associated with cognitive impairment, but of a rather general kind. It may reflect a problem of reduced central processing capacity or of diminished test motivation. It may be that high activity levels impair one's ability to perform well on any kind of structured psychological test. The association might also be accounted for if both hyperactivity and cognitive impairment result from a common cause, such as immaturity of development.

Further research will be needed to determine whether any specific cognitive deficit accounts for the hyperactives' general difficulty in performance tests. This research will have to take careful account of IQ before claiming a specific deficit in the hyperactive. It will not be sufficient merely to exclude the intellectually retarded, for our results indicate that the relation with IQ holds across the whole range of abilities. For the present, one should not regard hyperactive behaviour as a result of, or synonym for, a specific deficit in central processes of attention.

Aetiology of hyperactivity and conduct disorder

It would be tempting to conclude that hyperactivity is rooted in developmental delay, conduct disorder in problems of family life. Tempting, but not yet justified. Association does not prove causal relationship. It remains possible, on this evidence, that defiant, antisocial behaviour evokes inefficient and inconsistent coping strategies from parents, and causes coldness and hostility; rather than being caused by parental styles or genetically linked to them. It is just as likely from these data that teachers'

perceptions of antisocial behaviour are conditioned by their pupils' social class and ethnic background as that those factors cause antisocial behaviour. These findings are only clues to further research. They do suggest that psychological and social factors in children's conduct problems would be obscured if hyperactive behaviour were included as evidence of conduct disorder; and that neuropsychological studies should not include defiant or disruptive behaviour as evidence for the presence of hyperactivity.

Hyperactivity in the classroom showed only one significant association with the range of background factors examined: a previous episode of institutional care. This is nonetheless noteworthy, in view of previous findings on the consequences of institutional care. Tizard & Hodges (1978) reported that there were some long-term consequences of life in a children's home, even years after leaving it; notably, the children were hyperactive and inattentive at school but not at home. Since this finding is closely paralleled by our own, it directs interest towards the question of whether some kinds of close personal interaction are a necessary condition for the development of the normal modulation of activity and attention.

Implications for classification

The associations that we have reported have related to a conceptual framework of dimensions of behaviour disturbance on which each individual can meaningfully be ranked. This has only indirect implications for a categorical system of description such as the first axis of the International Classification of Disease. Inasmuch as they appear to be separate problems, it makes sense to have separate categories for pure hyperactivity and pure conduct disorder. However, most cases show a mixture of the two problems. Their classification is doubtful, and analyses of this paper do not help. Rather one will need to know how cases cluster on these dimensions, and the predictive value of such groupings. The application of cluster analysis to this question will be reported separately.

Regardless of what scheme of classification is adopted, clinicians should recognise both kinds of problem and include them both in diagnostic formulations. Though the behaviours are often present together, they have different implications. They are likely to require different kinds of treatment. Management will be less clear if the two problems are confounded into one label, whether that be 'conduct disorder' or 'attention deficit disorder with hyperactivity'.

Conduct Disorder and Hyperactivity: II

A Cluster Analytic Approach to the Identification of a Behavioural Syndrome

E. TAYLOR, B. EVERITT, G. THORLEY, R. SCHACHAR, M. RUTTER and M. WIESELBERG

Sixty boys, aged from 6 to 10 years, were studied after their referral to psychiatric clinics for antisocial or disruptive behaviour. Their scores on reliable measures of hyperactivity, defiant behaviour, emotional disorder and attention deficit were taken for the home, school and clinic settings; and subjected to two techniques of cluster analysis. Both gave a similar set of clusters, one of which had high scores on all measures of hyperactivity and attention deficit. Membership of this cluster was associated with a lower IQ, a younger age of problem onset and referral, an abnormal neurological examination, a history of developmental delay, smaller family size, poor peer relationships and a high rate of accidental injuries; and it predicted a good response to stimulant medication in a controlled trial. Other research on the classification of hyperactivity is discussed, and proposals are made for the criteria of a rather narrow definition of 'hyperkinetic conduct disorder'.

There is dispute even about the existence of a hyperkinetic syndrome. All diagnostic schemes include such a category, but there is serious disagreement about its definition and how frequently it should be applied. Is it a rare condition, usually encountered in the intellectually handicapped or epileptic (Rutter *et al.*, 1970a); or is it among the commonest of the psychological afflictions of childhood, affecting some 5% of the normally intelligent (O'Malley & Eisenberg, 1973)?

The definitions of the major current schemes reflect the diversity of views about what kind of condition should be recognised. The third edition of the American Psychiatric Association's Diagnostic and Statistical Manual (DSM-III) includes a condition of 'attention deficit disorder with hyperactivity' (ADD-H), for which the requirements are the presence of developmentally inappropriate overactivity, inattention and impulsiveness (APA, 1980). Other problems may also be present, but in that case one diagnoses the corresponding conditions as well. The ninth edition of the World Health Organization's International Classification of Diseases (ICD-9) also includes a condition termed 'hyperkinetic syndrome'. Its essential feature is poor concentration, and 'extreme' overactivity is associated (Rutter *et al.*, 1976). However, ICD-9 requires, more than the presence of hyperactivity symptoms, the absence of any other psychiatric

condition that could give rise to this behaviour. If both hyperactive and aggressive behaviours are present, for example, one must judge which is primary and make one diagnosis only. The DSM-III condition is common, the ICD-9 disorder is rare. Neither approach is of demonstrated superiority.

The uncertainty is a part of a wider problem of classification. Every child psychiatric service encounters many children who are referred because they have serious problems of social adjustment, are disruptive at home or school and fail to make friends or to keep rules or to learn. Many, but not all, seem wretched; many, but not all, seem chaotic and restless in their behaviour. Some suffer under social adversity, some come from seriously disordered families, some have been delayed in the normal course of neurological development. Their problems are many and diverse. Where hyperactivity is a common diagnosis, it is regarded as heterogeneous; where conduct disorder is common, its diversity is no less marked. Is there order in this heterogeneity?

We have argued in Part I that hyperactivity should be seen as a different kind of problem from defiant or antisocial behaviour. It could be thought of as a dimension of psychological symptoms, present to a greater or lesser degree in many disturbed children, and associated with cognitive impairment and poor sensorimotor coordination. (This was not a contribution to typology, but to dimensional classification.)

In Part II we examine the extent to which there is a natural distinction between a category of hyperkinesis and other conduct disorders. We have applied techniques of cluster analysis (Everitt, 1980) to children's scores on reliable, independent measures of behaviour and attention. The resulting groups are then compared, to determine whether they differ in potentially aetiological factors and in short-term response to treatment.

Method

The subjects, measures and procedure of this study have been described in Part I. Here we describe only the aspects unique to this part.

Subjects

From the 64 original boys who had received full assessments after referral for antisocial or disruptive behaviour, 60 were included in this part of the study. The remaining four had been judged to show no behavioural abnormality; their referral reflected inappropriate concern. It was thought desirable to exclude them from a classification of cases that was based on the child's mental state. Thirty-eight of the subjects accepted an offer of a trial of stimulant medication, of which the full results will be reported elsewhere (see below).

Measures

The measures chosen as the raw material for the cluster analysis were intended to describe key areas of the child's current psychological state. They cover behaviour at home, at school and in the clinic. They refer to the symptom domains of *hyperactivity*, with the Parental Account of Children's Symptoms (PACS) hyperactivity scale, Conners' Teacher Rating Scale (TRS) hyperactivity factor, and psychiatric interview hyperactivity scale; *conduct disorder*, with PACS defiance and TRS defiance; *emotional disorder*, with PACS emotional disorder, TRS anxiety and psychiatric interview 'depression' rating; and *attention deficit* with the composite 'attention performance' scale from a battery of psychometric tests of different aspects of attention.

Peer relationships were assessed during the PACS interview. Two different kinds of problem were rated from the parents' account: the first, 'isolation', refers to children who are not part of a peer group, do not have friends and do not take part in group activities; the second, 'unpopularity', refers to negative interactions with peers such as bullying or being taunted. Both can, of course, be present for the same person. Accident-prone behaviour was rated from accounts of episodes of recklessness, lack of appreciation of danger, and minor injuries. Accidents were defined as non-deliberate injuries that necessitated hospital treatment (including attendance at casualty departments).

Three measures were taken to determine short-term outcome. The first was a five-point clinical global rating scale made as consensus judgement by the research team

after each treatment period. 'Clinical global response' indicates that the score was at least one point better after drug treatment than after placebo for that case. The second measure, the 'hyperactivity response', was defined as a reduction of 50% in symptoms, by drug as against placebo, on either the PACS hyperactivity scale or the Conners teacher hyperactivity factor (or both). The third measure, 'marked clinical improvement', was present when the 'clinical global response' was positive *and* the child was given the maximum change rating of 'much improved' while on drug.

Procedure

After the initial assessment had been completed, 38 of the children went on to a trial of methylphenidate against placebo, in a double-blind cross-over design with random allocation to receiving methylphenidate first or placebo first. Each treatment period lasted for 3 weeks, with a washout period of 1 week between them. Measures were repeated at the end of each treatment period. A full description of the trial and its outcome is given by Taylor *et al* (1986).

Analysis

Cluster analysis refers to a diverse set of techniques for the collection of cases into groups in such a way that cases in the same group are similar in some sense, and dissimilar from cases in other groups. Two methods were applied: Ward's method and the method of complete linkage, both of which are described in detail by Everitt (1980). These two methods were chosen from the many available because a number of simulation studies have found them to be amongst the most satisfactory of clustering techniques. Since they gave very similar solutions, the clusters found by Ward's method were then compared on a range of dependent measures from the initial assessment and the post-treatment evaluations (using analysis of variance for the metric measures and χ^2 for the categorical).

Results

Cluster analysis

The two methods of cluster analysis yielded very consistent results. As always with clustering techniques, it is difficult to answer the question of the appropriate number of groups to consider. Consequently, in the absence of any completely satisfactory statistical test for number of groups, the four-cluster solution was chosen on grounds of clinical relevance and because of the very high agreement between methods. Only six cases were classified into different groups by the two procedures: there was total agreement for the other 90%.

The separation of the clusters was clear. Figure 1 shows the profile of mean scores of the four clusters on the variables from which they were derived. The scores are given after z-transformation so as to be comparable in range. One cluster (H) was composed of children with high scores on the hyperactivity scales in all three situations, poor scores

on attention performance, and less evidence of overt emotional disorder. It was made up of 15 cases, 25% of the total. It was clearly very similar to a nuclear definition of hyperactivity.

Cluster C contained 21 cases. It was characterised by an absence of high scores on most variables, but was high on the teacher's ratings of defiance (and, to a lesser extent, hyperactivity) in the classroom. Children in this group appeared to have a situation-specific problem of conduct.

Cluster A (13 cases) was high on teacher and parent measures of emotional distress, but not on 'depression' as rated by the examining psychiatrist. Hyperactive and defiant behaviours in the classroom were both low.

Cluster D (11 cases) was notable for high scores on 'depression' during psychiatric examination.

Both analyses showed the same pattern when the number of clusters was changed. In a three-cluster solution A and D were fused into one, larger group of emotionally disordered children. In the two-cluster solutions this group was joined with C, while H remained separate. When the number of clusters extracted was increased to 5, C broke down into two groups (distinguished by high or low scores on teacher hyperactivity). The most robust feature of the classification was therefore the appearance of the group of pervasively hyperactive and inattentive children (H).

Comparisons of clusters: developmental measures

The four clusters were compared by analysis of variance, with cluster membership as the independent variable and age, IQ and developmental neurological examination as dependent variables. Results are given in Table I. Significant differences existed between the clusters: the H group (of hyperactive children) were younger when referred, had lower IQs and showed more sensorimotor uncoordination. Since the neurological examination was not age-standardised, analysis of covariance was carried out for this measure, with age as covariate. The difference between groups was still significant ($F=3.4$, $P<0.05$).

Table II shows the associations between clusters and the presence or absence of a diagnosis on Axis 2 (developmental disorders) of the multi-axial version of ICD-9; of a history of developmental delay or of pre- or peri-natal difficulties; of more than three minor congenital anomalies; and of an early onset of the psychiatric symptoms. For these comparisons, clusters A and D were combined into a single 'emotional disorder' group, in order for numbers to be large enough for testing with χ^2 tests.

The hyperactive children in cluster H were more likely to have a history of a delay in motor and language development, and current evidence of delay in motor development or language development. It is of interest that the children in group C, identified as showing behaviour problems only at school, had a high rate of a different problem—specific delay in reading ability. Children with emotional disorders (A and D) had little evidence of developmental problems. Group H also showed an earlier onset of psychiatric disorder.

Comparisons of clusters: family relationships

Table III shows the mean scores of the clusters on the interview measures of family life and atmosphere; and the results of comparisons by one-way analysis of variance. There were, in general, few differences. There was a slight tendency for the hyperactives to have more contact with their fathers than the other groups, among the 46 who had fathers or stepfathers at home. In the same 46, the parents' marital relationship showed slightly more problems where the children had emotional disorders (A and D) than in cluster C, where there were few behaviour problems at home. However, these differences are not supported by overall significant differences, and will not be interpreted.

Comparisons of clusters: psychosocial background

The children in the different clusters showed no significant differences in socio-economic status: the father or principal breadwinner was in manual occupations in 33.3% of cluster H, 52.4% of C and 45.8% of A and D combined ($\chi^2=1.3$, NS). There was no difference in whether the child's parents had been born in the UK, the Caribbean nations or elsewhere ($\chi^2=2.2$, NS); nor in the presence of a history of institutional care ($\chi^2=2.4$, NS). Periods of separation from both parents for more than a month had been present in 23.8% of cluster C, 33.3% of H and 29.2% of A and D combined ($\chi^2=3.8$, NS). Family size was indexed by the number of children under 17 at home: this was three or more for 38.1% of cluster C, 13.3% of cluster H, 54.2% of clusters A and D combined ($\chi^2=6.5$, $P<0.05$).

Relationships of clusters to clinical diagnoses

There was considerable overlap, but not complete agreement, between the typology produced by this analysis and that of conventional diagnostic schemes. The ICD-9 scheme yielded seven children with the diagnosis of 'hyperkinetic syndrome'; every one of them had been classified into cluster H (the remaining eight in this cluster being all diagnosed as 'conduct disorder'). The more frequent diagnosis of 'conduct disorder' was more heterogeneous (18 in cluster C, eight in H, eight in A, six in D).

'Attention deficit disorder with hyperactivity' was frequently diagnosed in the DSM-III scheme. It too was a heterogeneous category in the terms of our present clustering: 14 cases came into C, 13 into H, five into A, three into D. It will be noted that all but two of the cases in cluster H met the DSM-III criteria. Figure 2 displays the relationships diagrammatically.

Comparisons of clusters: response to medication

Table IV sets out the percentages in each cluster who responded to methylphenidate according to the criteria outlined in the 'Methods' section. Cluster membership was a significant predictor of the pharmacological response to methylphenidate. Non-pharmacological aspects of response

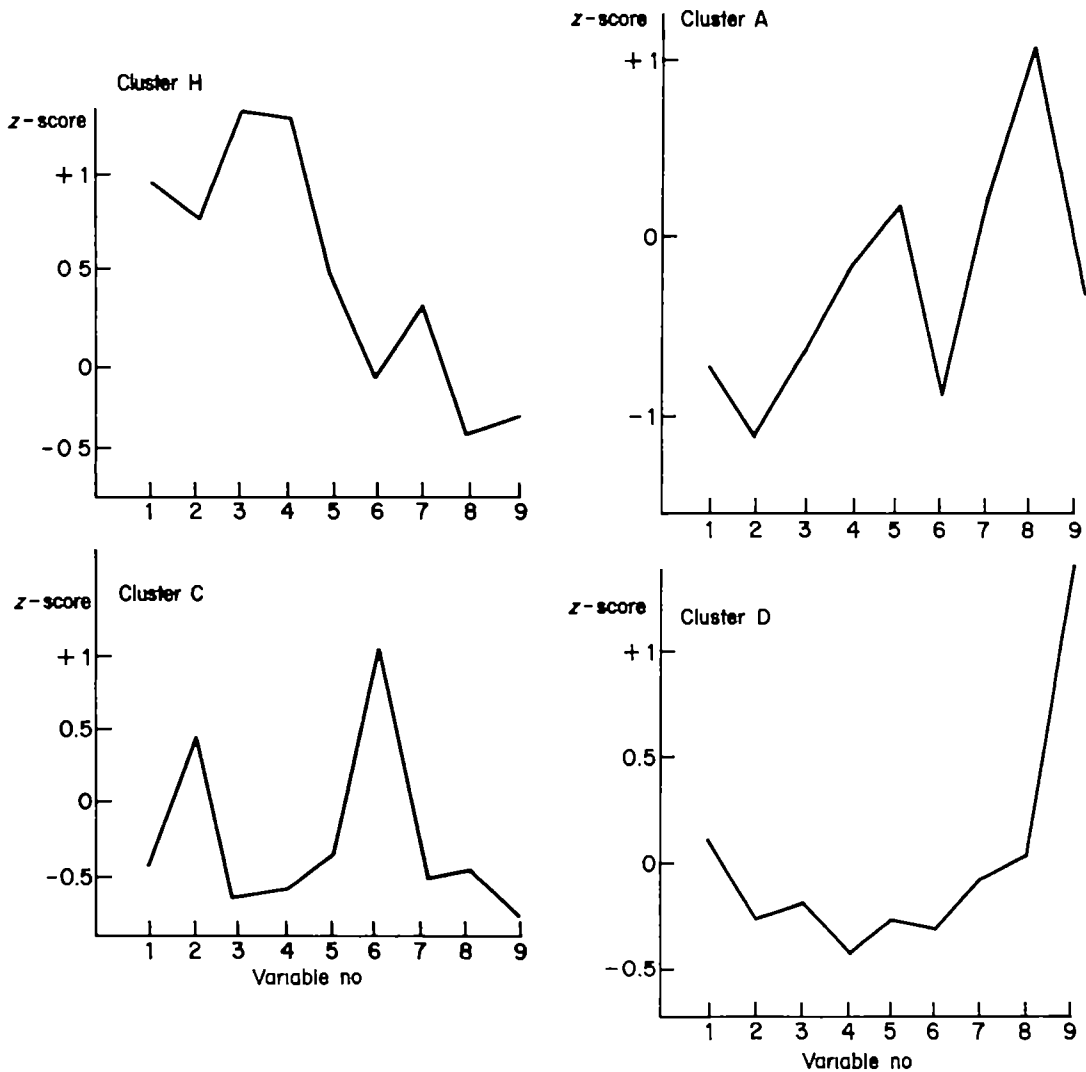


FIG. 1. Symptom scores for different clusters.

The profile of each cluster is shown on nine baseline variables, each of which has been transformed to a z-score for comparability. In each case, a positive score indicates a higher degree of pathology than the average for all cases.

Variable 1: Hyperactivity at home (from PACS interview).

Variable 2: classroom hyperactivity (from teachers' rating scale).

Variable 3: hyperactivity in psychiatric interview.

Variable 4: performance score on attention tests.

Variable 5: defiance at home.

Variable 6: classroom defiance.

Variable 7: emotional disorder at home.

Variable 8: classroom anxiety.

Variable 9: depression in psychiatric interview.

TABLE I
Clusters compared on developmental measures

Measure	Cluster				Significance	Significant pairwise comparisons
	H (n=15)	C (n=21)	A (n=13)	D (n=11)		
Age (mean, in months)	79	111	107	110	*	H<C, A, D
IQ (mean)	84.5	99.3	101.8	99.2	*	H<C, A, D
Neurological examination (mean)	20.7	6.5	4.5	7.2	*	H>C, A, D

* $P<0.05$ by one-way analysis of variance between the four clusters.

Note Pairwise comparisons made by Newman-Keuls test.

TABLE II
Comparison of clusters in the frequency of background variables

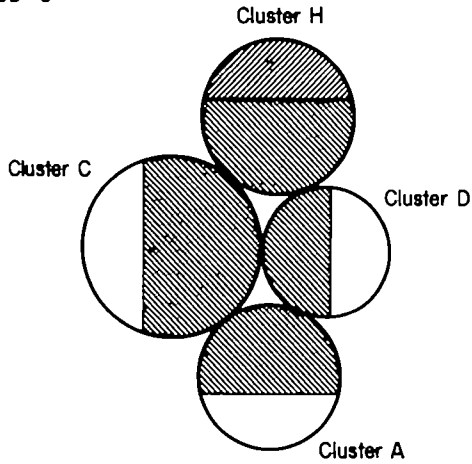
	<i>H</i> (<i>n</i> = 15)	<i>Cluster</i> <i>C</i> (<i>n</i> = 21)	<i>A + D</i> (<i>n</i> = 24)	
<i>Axis 2 of ICD-9</i>				
percentage diagnosed as				
No developmental delay	53.3%	71.4%	87.5%	
Specific reading delay	13.3%	28.6%	4.2%	
Dyspraxia or language delay	33.3%	0%	8.3%	*
<i>Developmental delay</i>				
percentage with positive history	60%	14.3%	25%	*
<i>Pre- or perinatal difficulty</i>				
percentage with positive history	33.3%	23.8%	29.2%	NS
<i>Minor congenital anomalies</i>				
percentage with ≥ 3	60%	23.8%	41.7%	NS
<i>Age of onset</i>				
(<i>n</i> = 59, one case undatable)				
percentage before 2 years	46.7%	4.8%	21.7%	
percentage 2-5 years	46.7%	52.4%	39.1%	
percentage after 5 years	6.7%	42.9%	39.1%	*

* $P<0.05$, by χ^2 test

TABLE III
Clusters compared on measures of family relationships

	Cluster				Significance	Significant pairwise comparisons
	H (n=15)	C (n=21)	A (n=13)	D (n=11)		
Contact with mother	1.87	1.62	2.09	1.91	NS	
Contact with father	2.50	1.50	1.33	1.22	NS	H>A, D
Expressed warmth	2.80	2.40	2.67	2.80	NS	
Expressed criticism	2.33	1.95	2.08	2.27	NS	
Efficiency of parental coping	5.00	4.20	5.08	5.30	NS	
Interparental consistency	1.78	1.59	1.40	1.75	NS	
Quality of marriage	3.25	2.53	3.78	3.56	NS	C<A, D

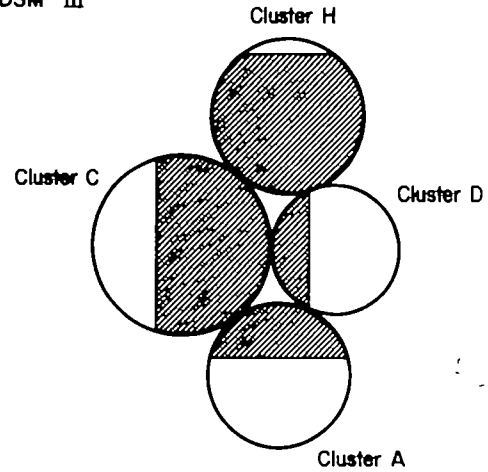
ICD-9



Key ICD-9 diagnosis

Hyperkinetic syndrome Conduct disorder Other diagnoses

DSM-III



Key: DSM-III diagnosis

ADDH present

Fig 2 Cluster membership and clinical diagnosis. The figure represents each cluster as a circle, subdivided according to the clinical diagnoses applied to cases in each cluster, for ICD-9 and DSM-III separately.

(such as the effects of suggestion, attention or regression to the mean) were controlled for, since all measures referred to the difference between drug and placebo treatments.

The group of children showing 'marked clinical improvement' obviously needs to be identified for practical purposes. Of the 38, 11 showed this marked response and of those 11, eight were in cluster H. Only three of the 27 who showed a lesser response were in cluster H. Membership of this 'hyperactive' cluster therefore predicted 73% of the marked responders, while non-membership predicted 89% of those with smaller, or no responses. This is accurate enough to be clinically useful.

TABLE IV
Response to medication in different clusters

	H (n = 11)	C (n = 14)	A + D (n = 13)
Clinical global rating			
Responders	90.9%	57.1%	30.8%
Non-responders	9.1%	42.9%	69.2% *
Hyperactivity response			
Responders	72.7%	50%	23.1%
Non-responders	27.3%	50%	76.9% *
Marked clinical improvement			
Present	72.7%	14.3%	7.7%
Absent	27.3%	85.7%	92.3% *

* $P < 0.05$, by χ^2 test.

TABLE V
Comparisons of clusters in the frequency of complicating symptoms

	Cluster		
	H (n = 15)	C (n = 21)	A + D (n = 24)
Adequate peer relationships	13.3%	38.1%	33.3%
Isolated or unpopular	20.0%	23.8%	45.8%
Isolated and unpopular	66.7%	38.1%	20.9% *
Presence of accident-prone behaviour	66.7%	9.5%	25.0% *
Accidents requiring hospital treatment:			
none	33.3%	66.7%	50.0%
one only	20.0%	23.8%	37.5%
more than one	46.7%	9.5%	12.5% *

* $P < 0.05$ by χ^2 test.

Comparisons of clusters: complicating symptoms

Certain behaviours enquired about during the assessment interviews were conceptualised as possible complications of psychological disorder. These were a propensity to accidents and an impairment of relationships with peers. Table V shows the association between these variables and cluster membership. All showed significant differences between clusters, with the 'hyperactives' of cluster H being

particularly likely to be isolated and unpopular in their relationships with other children, to be accident-prone and impulsive in behaviour, and actually to have suffered a high frequency of accidents serious enough to have required treatment at a hospital.

Discussion

Is there a syndrome of hyperactivity?

The cluster analysis identified a group of pervasively hyperactive and inattentive children among those presenting with antisocial or disruptive conduct. In itself this gives some support to the concept of a hyperactivity syndrome. For such a category to be clinically useful, however, it must be capable of predicting associations other than those which defined the group in the first place (Rutter, 1978). We have found that it does so.

Some of the associations are unsurprising, given that the cluster analysis had identified an inattentive, hyperactive group in the first place. Since they show this kind of cognitive impairment, it is perhaps to be expected that they would also show the delay in cognitive abilities that is indexed by a low IQ. The high frequency of signs of sensorimotor uncoordination, the history of developmental delay, and the presence of developmental disorders are in line with this, and contribute to the coherent pattern of findings. The importance of this pattern for development is underscored by the very high frequency of accidents (presumably as a result of impulsiveness) and of poor relationships with other children.

The younger age of the children in the hyperactivity group might reflect either a tendency for early onset and early referral, or a trend for the problems to change or lessen as children mature. It is possible, for instance, that specific reading retardation in later childhood is the heir to earlier delays in language and motor development. Longitudinal and epidemiological studies will be needed to answer these points. However, many of the findings are not attributable simply to the children being younger. Low IQ is not a result of greater youth, as of course the measure is age-standardised; abnormal neurological examination is not, since age was allowed for in the analysis; aspects of the history such as early onset and late milestones suggest that the differences between groups were present at an early age; the ability of hyperactivity to predict the response to stimulants is not mediated by age (Taylor *et al*, 1986).

Indeed, the short-term response to methylphenidate medication is the most persuasive

validation of the concept of a subgroup of hyperactive children. Membership of cluster H was a strong predictor of a good response. This must be seen in the context of previous findings that available measures predict stimulant response only weakly and doubtfully (reviewed by Barkley, 1976; Taylor, 1983). Furthermore, the ICD-9 and DSM-III diagnoses performed less well as predictors of response, showing no significant relationship with the measures of clinical global rating or of hyperactivity response (Taylor, 1983).

Relationship to other clustering studies

We are not aware of any previous use of cluster analysis to answer this particular question of the existence of a syndrome of hyperactivity. There have, of course, been previous attempts to use statistical methods to generate a taxonomy of schoolchildren's disorders (reviewed by Achenbach & Edelbrock, 1978). Results from these are somewhat conflicting — as is, perhaps, to be expected in that the methods have been applied to very differing series of heterogeneous clinical problems. Lessing *et al* (1981) reported a hierarchical cluster-analytic method, based on the product-moment correlation coefficient as the index of similarity, applied to a parents' behaviour checklist. One of the 15 clusters identified comprised over-reactive, distractible, clumsy, children with low tolerance of frustration. This was separate from 'aggressive, destructive, violent', 'academically disabled' and 'sociopathic' clusters, and is clearly made up of hyperactive children. On the other hand, Lessing *et al* (1982) applied the further strategy of choosing clusters which replicated across different samples, and reported seven groups with different profiles: one of them combined hyperactive and aggressive symptomatology into an 'aggressive, overactive' group. Furthermore (and rather surprisingly), a 'schizoid, withdrawn' group had equally high scores on hyperactive symptomatology.

Other studies seeking to establish typologies of behaviour problems have sometimes found a hyperactive group separate from other antisocial disorders (Patterson, 1964; Edelbrock & Achenbach, 1980); sometimes a common group of acting-out problems including both hyperactivity and other antisocial problems (Lorion *et al*, 1974; Langner *et al*, 1974). These contradictory findings might be seen as reflecting either the lack of a secure distinction between hyperactivity and other behaviour problems, or the limitations of the methods used. They are based on simple checklists concerning styles of behaviour, with consequent ambiguities of meaning

and problems of interpretation. By contrast, the current study is based on reliable measures from several different sources. Our numbers, however, are rather small. For this reason we do not address the more general question of the classification of psychological disorders. Our division of conduct disorders into the hyperkinetic, those confined to the classroom (often with educational failure) and those associated with emotional disorder (perhaps further divisible into anxiety- and depression-based disorders) has an obvious clinical appeal; but can only be a clue for future research.

Relationship to other studies of hyperactive children

A number of investigations have approached the question of validation by comparing those diagnosed as hyperactive with normal controls (Taylor, 1985), or children diagnosed as conduct disordered with normal and emotionally disordered controls (Rutter *et al.*, 1970b). Few have made comparisons between different types of disturbance of conduct, yet such studies are crucial for the definition and understanding of syndromes.

A previous clinic study from this Institute found the pervasively hyperactive to differ from matched psychiatric controls in ways that, like the present findings, pointed towards a developmental origin—namely early onset, inaccurate psychometric test performance, and sensorimotor uncoordination (Sandberg *et al.*, 1978). The present study has produced comparable results through a rather different route.

Stewart *et al.* (1981) and August & Stewart (1982) have presented two analyses of a series of children referred for psychiatric help. The first of these found that children with hyperactivity, but no other conduct disorder showed evidence of cognitive impairment by comparison both with the aggressive and with those with mixed aggression and hyperactivity. The criterion for identification was parental report solely. That study is therefore in line with our finding, in a companion paper, that the parental account of hyperactivity (but not of defiance) was associated with cognitive and neurological immaturity. The second of the papers from the Iowa group synthesised several sources of information into a definition of pervasive hyperactivity (August & Stewart 1982). Those who showed pure hyperactivity in this sense were generally similar to those who also showed an aggressive conduct disorder—except that those few with subnormal IQ scores showed a history of developmental disturbances. This is no contradiction to our present findings. In the first place, the dependent measures for the comparison

were those of putative aetiological factors and social background. We too have found that these measures do not distinguish clinical groups: the causes remain unknown. In the second place, the distinction between 'pure' and 'mixed' hyperactivity does not emerge from our analysis. Our group of pervasively hyperactive, inattentive children also score highly on measures of defiance and antisocial behaviour. 'Pure' hyperactivity seems a rare phenomenon at our clinics, and we have not yet identified it as a natural group.

Firestone & Martin (1979) also used the strategy of comparing a pervasively hyperactive group with clinical controls. The 'behaviour problem' controls in this case may in fact represent rather similar children: they were those for whom observers disagreed about whether or not they were hyperactive. Objective testing, however, did not show them to be any less active or more impulsive. The only difference between these groups—but an important one—was in the poor attention displayed by the hyperactive during psychological testing. The finding adds credibility to our identification of a group of pervasively hyperactive children who were also inattentive. The lack of other differences between their clinical groups is not in opposition to the present study, for they used a very different set of measures that did not include developmental or outcome variables.

Campbell *et al.* (1977) followed up a group of children, identified in a day nursery, until they had entered elementary school. The children with adverse outcomes at follow-up tended to be those who had previously displayed a lack of situation specificity and had been hyperactive in home and nursery alike. A study by Schleifer *et al.* (1975) has also suggested a worse outcome in later childhood for preschoolers with pervasive hyperactivity than with other behaviour problems.

Schachar *et al.* (1981) identified pervasively hyperactive children on the basis of questionnaire ratings from an epidemiological survey of 9-year-old children. They found a pattern (by contrast with a group of children with different problems) of poor cognitive test performance, lower socio-economic status and adverse prognosis. Their finding that hyperactivity predicted the outcome of a conduct-disordered group is supported by Thorley's recent follow-up of grossly overactive child patients with matched psychiatric controls (Thorley, 1984). It can be seen as the complement to the finding by Loney *et al.* (1978) that aggressive conduct disorder predicted the outcome of a hyperactive group. The combination of problems is likely to be particularly important. Indeed, McGee *et al.* (1984) have recently reported

results from an epidemiological survey that give direct support to this thesis, albeit on the basis of small numbers that may have been further biased by missing data. Children identified by teachers and parents as having both the problems of hyperactivity and conduct disorder were more prone to later behaviour disturbances, reading retardation and psychiatric referral than were those with only one of the problems.

In short, recent studies on pervasively and severely hyperkinetic children suggest that they are validly to be distinguished from other psychiatric categories in their prognosis and in their link with cognitive and motor immaturity. By contrast, studies of widely or loosely defined hyperactivity have found little discriminative validity in the idea (Sandberg, 1981; Ferguson and Rapoport, 1983; Taylor, 1985).

Towards diagnostic criteria

We suggest that the heterogeneous diagnoses of 'conduct disorder' (in ICD-9) and 'attention deficit disorder with hyperactivity' (in DSM-III) can be further refined. A substantial minority within both needs to be separately identified. Inclusion criteria for this 'hyperkinetic conduct disorder' group could be:

- (1) pervasive overactivity and restlessness, that is evident across two or more of the situations of home, classroom (or nursery) and clinic (or laboratory)
- (2) impaired attention, detected either by psychometric tests or by directly observed inattention and distractibility in behaviour
- (3) absence of overt symptoms of severe anxiety or other emotional disorder
- (4) presence of severe degrees of persistent non-compliance, aggression or destructiveness

A group defined in this way is likely to be composed of children with other evidence of developmental delay, at risk for accidental injury and with unsatisfactory peer relationships. Many, but by no means all, will have fitted the description of 'hyperkinetic syndrome' in ICD-9. They are likely to respond very well to stimulant drugs, at least in the short term. Longer term outcome, efficacy of other treatments and aetiology will need further research.

The first two criteria will have the effect of excluding many children at present regarded as 'hyperactive' in countries following North American diagnostic usage. It is unclear whether children, who meet the first three inclusion criteria but show no antisocial behaviour, should be given the same diagnosis. In present knowledge, outcome data

suggest that they be categorised in a separate group of pure 'hyperkinetic disorder'.

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Severe Self-Mutilation Among Kenyan Psychotics

Four case reports are presented of psychotic patients seen in clinical practice in Nairobi over a period of one year, who inflicted serious bodily harm to themselves during that period. Possible psychopathological basis for this self-damage is briefly considered. A review of the literature reveals paucity of similar reports from Africa and the third world. Reasons for this paucity are hard to come by.

Deliberate self-harm, as seen in psychiatric practice, tends to fall into two broad categories—a mild form usually associated with attention-seeking manipulative behaviour, and a severe form usually occurring in psychotic states. The former tends to occur in young individuals with neurotic or personality disorder. In the latter category the patients may be acting under some delusional belief or they may be obeying auditory hallucinations commanding them to do something which may be injurious to them. Rarely are modalities other than auditory involved. The delusions often involve beliefs that certain parts of the body are either deformed or causing the subject some trouble and should therefore be removed (Standage *et al*, 1974). A rather rare cause of self-mutilation is masochistic sexual behaviour. Schott (1973) has described self castration due to sadistic impulsive perversion and Thompson & Abraham (1983) have reported two rare cases of male genital self-mutilation following paternal death.

Severe self-destructive behaviour is rarely reported from the African continent and the third world in general. This raises certain basic questions such as (a) Does self-mutilation occur at all in these countries? (b) If so, is it in any way different from that seen in the Western countries and finally, (c) Are there any cultural beliefs which either protect or aggravate such behaviour?

Below, we report four cases of severe self-mutilation seen by us during the year 1984. The first three were seen at the Government Mental Hospital (Mathari) and the last at our National Teaching Hospital (Kenyatta National Hospital).

Case reports

Case 1

A 35-year-old inpatient former police constable who had been admitted several times to Mathari Hospital with a diagnosis of schizophrenia presented as an emergency with severe bleeding half an hour after he had extracted his four

lower incisors. Twenty days later he extracted a further six teeth from his lower jaw and continued to self-mutilate his gums with any hard object he would come across. A week later he attempted to hang himself with a blanket but was rescued by a nurse.

On examination he had extensive lacerations of gums and adjacent soft tissues, leaving ridges of lingual and buccal plates bare. Curettage to remove necrotic tissues, alveolectomy and stitching of buccal/labial and lingual flaps was done.

The patient continued removing stitches and developed osteomyelitis. He voluntarily stopped mutilating himself and made a good recovery but remained thought-disordered and aggressive to other patients. He would give no explanation for his actions.

(NB While this paper was being prepared the patient, by means of a sharp pointed piece of metal, dug a hole into his right flank up to the kidney and bled to death).

Case 2

A 16-year-old housemaid was admitted at midnight with a 2 week history of aggressive behaviour, insomnia and refusal to feed.

One hour after admission the patient was found to have bitten off one-third of her tongue which was found on the floor. The remaining stump was sutured, haemostasis achieved and she was put on antibiotics. The patient removed the stitches from her tongue and left the wound gaping. She denied being depressed or having been mistreated by her employer. She did not give any motive for her actions. There was no history of epilepsy in the patient or her family nor was she ever observed to have fits. After 3 weeks' observation, a diagnosis of schizophrenia was made and she was put on depot phenothiazines. She made a satisfactory recovery and was discharged through the out-patient psychiatric follow-up clinic.

Case 3

A 30-year-old childless housewife was admitted to the maximum security section after allegedly having murdered her mother-in-law. After 2 years as an inpatient, she developed suicidal tendencies although she denied being depressed. She threw herself from the roof of the ward and

sustained multiple fractures of both lower limbs. Her right foot became gangrenous and had to be amputated. On recovery, she began mutilating her breasts with any sharp object she could get; later, she went on to self-mutilate her genitalia and sustained a third-degree tear. She talked of being worthless since she could not give birth, hence she had no use for the organs she kept mutilating. There was no history of psychiatric illness in the family. A consultant psychiatrist had diagnosed paranoid schizophrenia and put her on appropriate treatment but she has continued to mutilate her external genitalia.

Case 4

A 24-year-old man presented as a surgical emergency with a deep cut into the shaft of the penis which he could not explain. He was a lonely and withdrawn single and unemployed young man with minimal education. He had no girl friend and lived with his elderly parents in the slums of Nairobi. At interview, he revealed that he had attempted to amputate the penis because some voices kept telling him to do so. He believed that the penis was useless to him. Surgical repair of the penis was done and he made satisfactory recovery on oral phenothiazines.

Discussion

Self-mutilation in psychotic patients is not rare in the West (Greilshamer & Groves, 1979), though we could not find any case report from the African continent. Reports of oral self-mutilation are predominantly male (Mendez *et al*, 1972; MacLean & Robertson, 1976; Greilshamer & Groves, 1979; Pattison & Kahan, 1983; Rada & James, 1982). This is surprising in view of the reported excess of self-damaging behaviour in women. Our cases show no specific pattern either by age, sex, or site, except that two mutilated their mouths and two their genitalia. In either category, one was male and other female. Genital self-mutilation has been associated with unresolved sexual conflicts (MacLean & Robertson, 1976). Symbolically, oral self-mutilation might, in theory, be considered to represent a desire to avoid saying something which is stress-provoking. This would be particularly so in case number 2. In case number 3 there may be severe social pressures. The social and cultural stigma associated with infertility in Africa in general and Kenya in particular does sometimes lead to suicidal attempts but not to self-mutilation, in our experience.

Most of the published reports stress the surgical management and there is very little said about the psychiatric management (Mendez *et al*, 1972; Engelman *et al*, 1974; Standage *et al*, 1974). This is a pattern which should be corrected. Once the emergency surgical management has been carried out, urgent psychiatric referral is essential (Standage

et al, 1974). The majority of such cases will probably turn out to be psychotic, and should therefore benefit from neuroleptics. There will, however, be a small minority of personality and neurotic disorders (Thomson & Abraham, 1983) who will benefit from psychotherapy, as do the majority of psychotics once insight has been restored.

With regard to the questions raised earlier in the second paragraph of this paper, although we have not found published reports on this subject, the occurrence of four cases within a period of 1 year in Nairobi alone (population approx. 1 million) suggests that severe self-mutilation may be occurring as commonly in our setting as in the more developed Western countries. From our series the pattern, mode and severity of such self-destructive behaviour does not differ much from reported cases in the West. We were unable to find any cultural beliefs which have an effect on such behaviour, and overall one is impressed more by the similarities across the cultures than any differences.

It is the belief of the present authors that 'detrification' has less influence on manifestations of psychotic illness but in neurotic disorders the situation may be quite different. Earlier psychiatrists in Kenya (Gordon, 1936; Carothers, 1947) had forecast increased psychiatric morbidity with the erosion of traditional coping mechanisms, such as the extended family, resulting from urbanisation and western style of living. All our cases, however, had lived a substantial part of their lives in rural Kenya where traditions are still closely adhered to. In this regard it would have been interesting to find out about any visits to traditional healers as recent work in Kenya (Dhadphale, 1984, unpublished thesis) has shown that a significant number of psychiatric patients visit traditional healers as compared with those with physical disorders.

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Anorexia Nervosa and Dysmorphobia

A Case Study

Anorexia nervosa is frequently associated with neurotic traits and symptoms. The symptomatology and history of a housebound 20-year-old woman with anorexia nervosa and dysmorphobia are described. The role of the family in maintaining the problems, use of external cues to control behaviour, overvalued somatic ideas and the definition of dysmorphobia are discussed.

Anorexia nervosa frequently occurs alongside a variety of neurotic symptoms and traits. These include neuroticism, introversion (Smart *et al*, 1976; Hsu & Crisp, 1980), depression (Morgan & Russell, 1975; Eckert *et al*, 1982) and obsessiveness (Morgan & Russell, 1975; Smart *et al*, 1976; Gomez & Daly, 1980). Following recovery of weight and menstruation these neurotic symptoms and social difficulties frequently remain. (Stonehill & Crisp, 1977; Hsu & Crisp, 1980). On average, anorexics also overestimate their bodysize compared to both normal and neurotic controls (Slade, 1985). Preoccupation with body image and food may become an overvalued idea (Thomas, 1984) and has been considered delusional by some (Bruch, 1974).

This paper gives details of the symptomatology and history an unusual case of anorexia nervosa and dysmorphobia.

Case study

A 21-year-old woman was re-referred to a university-based clinical psychology department following an overdose of

50 paracetamol tablets at 2 a.m., 1 week previously. Assessment was carried out during three domiciliary visits. The patient's pattern of eating, as assessed by 1 week of self-recording was highly abnormal. She claimed to eat an average of 122 calories (range 79-148) per day. Food was restricted to grapes, melon, apple, tea with skimmed milk and unsweetened orange juice. Most eating occurred between midday and 7 pm. She reported that she did not induce vomiting. She did not use laxatives as they were not available to her, although in the past she had used them. Her height was 5'7" and she weighed approximately 8 stone (51.3 kg).

She reported being highly pre-occupied with her physical appearance. Her aim was not to reach any particular target weight, but rather to attain a slim appearance. She considered herself to be generally too "broad". Using a Body Satisfaction Scale (Slade, 1985b) she rated 16 body parts as follows: *Very Satisfied*—none; *Moderately Satisfied*—eyes, ears; *Slightly Satisfied*—none; *Undecided*—head, nose, mouth, chest, hands; *Slightly Unsatisfied*—face, jaw, shoulder arms; *Moderately Unsatisfied*—teeth, legs, feet; *Very Satisfied*—tummy. In order to gain a better body shape, she said that she carried

out exercises for up to 1½ hours per day to 'tone-up' her muscles. She typically dressed in a skimpy nightie, underwear and housecoat and would not wear daytime clothes because she felt she looked 'broad' in them. She reported that when talking to anyone she was constantly worried about what they thought of her appearance. When people reassured her she believed that they were first trying to be kind.

She reported that she did not leave the house because of the way she looked, extreme embarrassment, and fear of comments from other people on her appearance. She had remained almost completely house-bound for over 2½ years and had left the house less than 10 times during this period. These were almost all hospital visits. Consequently she had developed a daily pattern of life within the home doing housework and reading and making notes extensively about anorexia and bulimia from books and television programmes. Secondary gains were apparent from remaining at home. These included friends (and professionals) visiting her at home, avoidance of work and anxiety-provoking social situations, and a lot of attention from family, neighbours and professionals concerning her condition. In particular, her mother was the major vehicle in her transactions with the outside world.

She also reported being highly socially anxious due to her preoccupations with what other people thought of her appearance. In addition, she was socially unskilled. Her eye contact was brief and infrequent; she often blushed, and laughed nervously and inappropriately; her posture was orientated away from the speaker, and she frequently placed her hand over her mouth and talked excessively about her appearance. Her dress was inappropriate and paradoxically drew attention to those bodily features which most embarrassed her.

There was no evidence of formal thought disorder, systematised delusions, hallucinations or a depressive disorder. Diagnosis of both anorexia nervosa and dysmorphophobia could be substantiated.

History

The patient was born by Caesarian section, weight 7 lb (3.2 kg). She had been referred to a psychiatrist during childhood for sleeping difficulties but her mother had been told that she was simply a bright, active child and there was no cause for concern. During attendance at infant school she was reportedly a fussy eater. At junior school the family reported that she did well and they were proud of her. However, she reported being self-conscious at times and recalled having been teased by her father and two brothers for 'having legs like tree trunks'. Menarche occurred unremarkably at 12 years of age.

All the family explicitly dated the onset of the problem to a school nurse remarking that she was overweight (10½ stone/66.8 kgs) following a routine health check at the age of 12 and advising that she should lose weight. She lost a few pounds but was told by her GP that this was not enough (10 stone 2 lbs/64.5 kgs). She then starved herself for 6 weeks getting down to 9 stone (57.3 kg) eating only occasionally rye bread and apple. When she was 14 years old she starved herself during the summer holiday and lost

weight dramatically. At around this time her paternal grandmother died and she was upset for quite a few days afterwards. When she was 16 years old her GP had noted possible early anorexia nervosa. At this time her elder brother suffered a single schizophrenic attack. This followed his being teased at school about his curly hair whereupon he became preoccupied with this. (He was admitted to hospital, made a good recovery and has had no relapses in 5 years since.) During the same year she sat her mock and CSE/GCE exams (6 CSEs, 1 'O' level) and under-achieved. She recalled that she was unable to concentrate and revise because of continual thoughts of food and her appearance.

After school she attended a college to study office work but only stayed for 10 weeks. She left because she thought people were laughing at her. Following this she did not leave the home once in 8 months. By this time she weighed 8 stone (53.6 lbs) and the GP referred to a psychiatrist following her mothers concern at laxative abuse. However, she refused to attend a day hospital and take medication. She was referred to a clinical psychologist but attended only one appointment. Over the next 2 years she was referred three times and again refused family therapy, psychotherapy and behaviour therapy within two or three appointments of each re-referral.

Discussion

Anorexics have been shown to depend on external cues more than control subjects in relation to food regulation (Crisp & Kalvcy, 1974; Russell *et al*, 1975; Garfinkel *et al*, 1978). Despite this finding in several independent studies involving different paradigms, anorexics do not have a more external locus of control than normal or thin patient controls (Garner *et al*, 1976). This suggests that dependence on external cues to regulate behaviour is restricted to food regulation and perception of body image rather than a general cognitive style. In the present case the patient frequently sought reassurance from others concerning her appearance and was highly preoccupied, especially in social situations, with what others might think of her appearance. Her dysmorphophobia can be seen as developing from her extraordinary reliance on external feedback concerning her physical appearance. The term 'dysmorphophobia' has been used in several distinct ways. It can refer to a non-specific personality disorder in patients seeking cosmetic surgery without physical cause (Crisp, 1981) and quite distinctly as a social phobia (Marks, 1980). In our case the term refers to a social phobia as evidence of high anxiety in social situations, avoidance of these situations, fear of criticism and adverse comments about physical appearance. Such social anxiety and commonly associated traits of neuroticism and introversion are relatively common in anorexia, both during periods

of low weight and following restoration of normal weight and menstruation (Hsu & Crisp, 1980; Smart *et al*, 1976) although a true phobia leaving the patient housebound is relatively rare.

The role of family members in the maintenance of these problems is not clear, although some speculations can be made. Firstly, a number of secondary gains were apparent both from her mother who acted as a median with the world on her behalf; her younger brother also arranged friends to visit her. The importance of secondary gains is highlighted by the obvious untruth in at least one of the patient's three claims of extremely low food intake, stable weight and long periods of exercise. Her GP confirmed that her weight had been stable for many months. Both the patient and the family appeared to subscribe to a 'romantic' view of anorexia nervosa: references to well-known anorexics were often made. Loss of high academic potential was made both by the patient and the mother and anorexia was the explicit reason given for academic underachievement. She appeared to be of average academic ability. Her brother's single schizophrenic attack may be of relevance; this was precipitated by bullying at school and was preceded by pre-occupation with physical appearance. He may have acted as a model, both specifically in terms of the content of her overvalued somatic ideas, but also

generally by legitimising, within the family, avoidance of school and academic situations through bodily preoccupation.

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Organic Factors in Catatonia

A retrospective case-control study was performed using the records of 59 catatonics, 59 non-catatonic schizophrenics, 59 manics, 59 depressives and 59 surgical controls. The findings suggest that prior brain injury and physical illness at onset of psychosis are much more common in subjects with catatonia. While these findings do not account for all cases of catatonia, they may indicate an aetiology for some phenotypic cases of catatonia.

Catatonia is a clinical syndrome characterised by symptoms of mutism, psychosis, bizarre motor activity and either agitated excitement or stupor. This syndrome is generally associated with the diagnosis of schizophrenia; however, it has been noted in non-schizophrenic individuals and has been said to be a non-specific syndrome (Abrams & Taylor, 1976). Catatonia is an interesting condition because the prevalence of this disorder is felt to have decreased over time (APA, 1980).

It is possible that the reduction in the occurrence of catatonia reflects the fact that more effective treatments are now available in psychiatry and that these treatments are arresting the progression of common psychiatric disorders before they develop into a catatonic state. There are problems with this assumption. Firstly, it assumes that a common psychiatric illness must necessarily be a precursor to catatonia; this has never been proven by scientific studies and has never been strongly indicated in the clinical literature. Secondly, no intervention studies have been performed which, while evaluating the efficacy of psychiatric treatment for the larger diagnostic categories, have ever specifically evaluated the association of these treatments with catatonia as an outcome.

Since the clinical literature suggests that the frequency of catatonia appear to be decreasing with time, it is possible that the syndrome of catatonia may arise from some type of environmental exposure which has changed over time. The relative decrease in the frequency of catatonia may then reflect some type of cohort effect. Similar events have happened in the past; for example, the encephalitis epidemics of the early twentieth century resulted in a cohort of individuals with Parkinson's disease. Similarly, an exposure to a now uncommon illness may possibly have predisposed people to catatonia. Both brain injury (Gelenberg, 1976) and infection (Raskin & Frank, 1974) have been associated with the occurrence of catatonia in the past, suggesting that such exposures may be risk factors for this syndrome.

This study was conducted in order to evaluate the association of specific environmental exposures to the outcome of catatonia later in life. The specific exposures which were selected as possible precursors to catatonia were possible childhood brain injury and history of encephalitis-like symptoms shortly before the onset of psychiatric symptoms.

Method

A case-control design was used to examine prior noxious exposures among catatonic subjects and controls. Since catatonia is no longer a frequent occurrence, it was decided

that a retrospective examination of catatonic individuals would be performed using the records of our Psychiatric Hospital from patients who were admitted during the decades when catatonia was more common (1930-1945). In this way a reasonable number of subjects could be obtained. We had access to a large number of records of individuals whose psychiatric histories were very clear. Mental state examinations in these records were detailed and often 15 pages long, containing transcribed discussion with patients and their families. These patients were re-diagnosed using operational criteria (Feighner *et al*, 1972). Medical histories were also very clear and extensive. Patients with the syndrome of catatonia were randomly selected from these records. Since catatonia is so severe a condition that a period in hospital is required, we did not feel that a large selection bias was introduced by using a university hospital population for their selection. Our criteria for inclusion into the group of catatonic cases were: (1) stupor or mutism; (2) negativistic or oppositional behaviour; (3) rigid motor activity; (4) uncoordinated purposeless excitement; (5) bizarre posturing. Cases had to meet at least two of these criteria to be considered catatonic. These criteria are generally accepted as characterising the syndrome of catatonia and conform to the description of the catatonic subgroup of schizophrenia as described by the *Diagnostic and Statistical Manual of Mental Disorders (Third Edition)* of the American Psychiatric Association (APA, 1980). Using these criteria, 59 catatonic subjects were obtained of whom 57 were stuporous.

Since catatonia is generally a disorder of the young (Gjessing, 1938) and since exposure variables may differ between sexes, the catatonic subjects were matched on the variables of sex and age to control for confounding. Fifty-nine schizophrenics, 59 manics, and 59 depressed patients were selected for comparison with our group of catatonics. These psychiatric control patients were also admitted between 1930 and 1945. These patients were re-diagnosed using operational criteria (Feighner *et al*, 1972). An additional 59 sex- and age-matched surgical inpatients were also selected from the years 1931 to 1945 for comparison to the catatonic population. The two categories of exposure which were examined were: (1) possible brain injury; (2) symptoms of severe physical distress at onset of psychosis.

The criteria for admission to the first category required that a patient have a previous history of head injury so severe that it required medical attention or a history of encephalitis or a history of seizures or was a forceps delivery. The criteria for admission to the second required that patients have a history of vomiting, diarrhoea, or severe headache with severe neck stiffness or high fever immediately prior to the onset of psychiatric symptoms.

It was assumed that if there was an association between these exposures and catatonia then the catatonic group would have a higher frequency of these problems than would their counterparts in the other samples. Statistical analysis of the data was carried out by using the χ^2 test.

Results

For the first category (possible brain injury), significant differences were found in the following categories:

(1) catatonics vs manics, $P < 0.05$ (23 compared to 11); (2) catatonics vs depressed, $P < 0.01$ (23 compared to 7); (3) catatonics versus surgical controls, $P < 0.001$ (23 compared to 2). No significant difference was found in the frequency of possible brain injury as defined by our criteria between catatonics and schizophrenics (23 compared to 18).

Concerning the outcome of category 2 (physical illness at onset), the significant differences were as follows: (1) catatonics vs manics, $P < 0.05$ (18 compared to 7); (2) catatonics vs depressed, $P < 0.001$ (18 compared to 2); (3) catatonics versus schizophrenics, $P < 0.001$ (18 compared to 3). Comparison with surgical controls was not applicable in this category since they did not have an onset of psychiatric symptoms with which to compare.

Discussion

The analysis of these data was consistent with previous anecdotal findings that the onset of catatonia is frequently associated with concurrent physical distress (Gjessing, 1938). The higher frequency of possible brain injury among catatonics as compared with other (non-schizophrenic) psychiatric illnesses and controls suggests an association between psychosis (specifically catatonia) and brain dysfunction.

Although an association may have been demonstrated between the two described categories and the occurrence of catatonia, more definitive statements would not be appropriate at this time. We felt that the generally high quality of these records minimised the possibility of information bias. We attempted to use objective statements from the records in order to minimise problems with this category. It was felt

that encephalitis and seizures would generally indicate brain insult and that any head trauma severe enough to require medical attention would be very likely to also indicate this type of injury. Similarly, in the second category, stress was put on objective signs of illness in an attempt to exclude somatic delusions. By matching for age, we controlled for the possibility that variation in use of forceps delivery, over the years, would systematically isolate a diagnostic group in an artificial way.

The findings of this study suggest that there is an association between childhood brain injury and the syndrome of catatonia. This may mean that the changing prevalence of catatonia is reflective of the general improvement in health status of children over the last 50 years rather than the introduction of specific psychiatric therapies. We encourage further research in this area and hope that attempts will be made to replicate this type of study.

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Suicide Among Schizophrenics:

A Comparison of Attempters and Completed Suicides

Schizophrenics who completed suicide were compared with those who made suicide attempts, on the basis of blind ratings of previous hospital records and follow-up

interviews with treating clinicians. Results indicated that the two groups were relatively distinct. Suicides tended to live alone and to feel depressed, hopeless, worthless, and suicidal. Attempters, on the other hand, lived with their families or others and were less likely to manifest several features of depression during a period in hospital. In evaluating suicide potential among schizophrenics, living situation and mental state changes indicating depression, suicidal intent, worthlessness, and hopelessness are more important than a history of suicidal behaviour.

Suicide is a frequent complication of schizophrenia (Miles, 1977), yet clinicians evaluating these patients for suicide potential face a difficult task. The empirical literature emphasises the importance of previous suicidal behaviour (Warnes, 1968; Breier & Astrachan, 1984), particularly multiple attempts (Cohen *et al.*, 1964; Shaffer *et al.*, 1974). Suicide attempts are, however, so frequent among young schizophrenic patients (Niskanen *et al.*, 1973; Planansky & Johnston, 1973) that other risk factors may be more discriminating. As is the case with other diagnostic groups, suicides and attempters probably represent different but overlapping populations (Stengel, 1962). The purpose of this study is to compare schizophrenics who complete suicide with those who make attempts.

In previous work, we identified the characteristics of schizophrenics who complete suicide through an extensive literature review (Drake *et al.*, 1985) and two empirical studies (Drake *et al.*, 1984; Drake & Cotton, 1986). In this study, we compare attempters with completed suicides on the basis of a blind examination of data from prior periods in hospital and follow-up interviews with treating clinicians.

Method

Fifteen schizophrenic patients who had undergone periods in hospital, in an urban, public mental health system between 1976 and 1980 subsequently committed suicide. A comparison group of 89 other schizophrenics, who had undergone periods in hospital, in the same treatment system during the same time period was followed up 3–7 years after index admission. Of these patients, 19 had made suicide attempts, defined as any intentionally self-inflicted injury, during the follow-up period. All 19 attempts were medically non-serious. Using a semistructured interview, treating clinicians were questioned regarding the circumstances of all suicides and attempts. They were asked to identify any changes in behaviour, mental state, family, or other relationships that preceded the suicide or attempt. All patients met DSM-III criteria for schizophrenia according to blind records review by two psychiatrists.

Patients' hospital records were abstracted to exclude any reference to subsequent suicidal behaviour and were rated independently by two research assistants according to risk factors from the literature and depressive symptoms from DSM-III. As described previously (Drake *et al.*, 1984; Drake & Cotton, 1986), these ratings were made blindly and

reliably. The results presented here are based entirely on the blind ratings of previous hospital records but in the discussion section, we use information from the non-blind follow-up interviews to generate possible explanatory hypotheses.

Results

A partial list of the 50 variables is presented in Table 1. Complete lists appear elsewhere (Drake *et al.*, 1984; Drake & Cotton, 1986). Because of the small sample size, only large differences between suicides and attempters are statistically significant. Therefore, the large number of significant variables (over 20%) suggests that there are important differences between schizophrenics who kill themselves and those who make attempts. Current follow-up of the attempters, now 5–9 years after index admission, has revealed no further suicides and supports the view that attempters are a relatively distinct group.

The two groups were largely similar in terms of socio-demographic characteristics, history of illness, and background variables. Although the suicides were, on average, 7 years older than attempters, this difference was not

TABLE I
A comparison of schizophrenic patients who completed suicide with schizophrenic patients who attempted suicide

Characteristics	Suicides (n = 15)	Attempters (n = 19)	Sig. level ¹
Age (years)	31.7	24.8	NS
Sex (male)	60%	47%	NS
Previous suicide attempt	73%	47%	NS
Three or more attempts	40%	21%	NS
Explicit suicide threat	67%	16%	<0.01
Lives with family of origin	27%	63%	<0.05
Lives alone	60%	11%	<0.01
Depressed mood	80%	52%	<0.05
Feels inadequate	80%	42%	<0.05
Hopelessness	67%	26%	<0.05
Worthlessness	53%	21%	<0.05
Suicidal ideation	87%	37%	<0.01
Fears of mental disintegration	33%	5%	0.05
High self-expectations	47%	16%	0.06
Improved at discharge	67%	32%	<0.05

1. Significance levels refer to *t* tests or χ^2 tests

statistically significant. In terms of suicide history, only a history of explicit suicide threats differentiated the two groups statistically. Other possible predisposing factors, such as a history of violence or substance abuse, failed to separate the two groups. Behaviour during stays in hospital, such as being impulsive, demanding and agitated, also failed to discriminate, perhaps because of the time-lag between the period in hospital and suicidal behaviour for most patients.

Living situation and mental state examination revealed several important differences between attempters and suicides. Attempters were more likely to be living with their families of origin. Suicides, on the other hand, tended to live alone. On mental state examination, the suicides reported feeling depressed, inadequate, hopeless, worthless, and suicidal. They also expressed higher performance expectations and fears of further mental disintegration. In addition, suicides were more likely to be 'improved' at discharge.

Discussion

Our results indicate that schizophrenics who kill themselves are more depressed, suicidal, and isolated than those who make attempts. Consistent with findings by Roy *et al* (1984), many of the attempters were depressed during their period in hospital. Those who completed suicide were, however, even more likely to be depressed and were especially prone to manifest the psychological features of depression, such as hopelessness. They were frequently 'improved' at discharge but returned to more isolated living situations.

To explain these findings, we offer several hypotheses, some of which are based on our non-blind follow-up interviews. Schizophrenics who kill themselves may be predisposed to depression and isolation by a prevailing sense of aloneness (Buie & Maltzberger, 1983). Because they are more hopeless about their futures with chronic mental illness and fear continued mental disintegration, they may kill themselves specifically to avoid this fate (Baechler, 1979). Perhaps they are predisposed to despair and self-destruction because of high premorbid achievement and persistent self-expectations that are no longer realistic (Drake *et al*, 1984). Although improved at discharge, follow-up interviews indicated that these patients lapsed into further hopelessness and depression following discharge. Suicide usually occurred in the context of interpersonal isolation. Similar to findings by Breier & Astrachan (1984), our follow-up interviews revealed that many of the suicides had experienced some loss of familial support within weeks of killing themselves (Cotton *et al*, 1985). Perhaps unwanted separation from family was a crucial stress for some of these patients. Alternatively, as suggested by

Virkkunen (1976), they may have withdrawn from family as well as others as a manifestation of their extreme hopelessness.

While completed suicides present a picture of overwhelming depression, withdrawal, and despair, suicide attempters appear to be a more heterogeneous group. Consistent with findings by Planansky & Johnston (1973), our follow-up interviews revealed a variety of situations, clinical states, and apparent motives among attempters. According to treating clinicians, these patients were often agitated or psychotic rather than depressed immediately prior to the attempt. Psychosis, lack of awareness, or lower self-expectations may have protected them from developing despair over their illness and future. The follow-up interviews also indicated that attempts frequently occurred in the context of interpersonal conflict, such as a fight with family or housemates, rather than isolation. It appeared to clinicians that some patients were trying to manipulate others, consolidate support, or gain entrance to the hospital. In addition, some of the more impulsive attempts were associated with dysphoric side-effects of medication such as akathisia (Drake & Ehrlich, 1985).

Our findings have several implications for clinicians evaluating suicide potential in schizophrenic patients. Some young schizophrenic patients, particularly when they have separated from their families of origin, will develop depression, worthlessness, hopelessness, fears of further disintegration, suicidal ideation, and social isolation. This profile is a more dangerous indicator of suicide risk than a history of suicidal behaviour or a current suicide attempt. We suggest that these high-risk patients be monitored carefully and considered for more intensive interventions, such as a period in hospital, day treatment, family therapy, and structured, group-living situations.

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Trazodone-induced Mania

Two bipolar depressed patients and one unipolar depressed patient developed manic symptoms after receiving trazodone. The symptoms resolved when the treatment was discontinued or reduced. We believe the two bipolar patients are the first cases of this reaction reported in the literature.

Trazodone, a triazolopyridine derivative, is a novel antidepressant which has been studied thoroughly in the last years. More than 400 clinical studies, including 20 000 patients, have been performed on trazodone (Agnoli *et al*, 1984). Clinical trials have shown that trazodone is as effective as the tricyclic antidepressants in the treatment of depressed patients (Fabre *et al*, 1979; Kellams *et al*, 1979). Trazodone does not bind to muscarinic cholinergic receptors *in vitro* (Taylor, 1981) and has minimal anticholinergic and cardiovascular side-effects (Gershon & Newton, 1980; Cassano *et al*, 1982). Therefore, it is an attractive choice in depressed geriatric patients and in patients suffering from angina, prostatism, constipation and glaucoma (Gershon, 1984).

Roy-Byrne *et al* (1984) in their article on approaches to the evaluation and treatment of rapid-cycling affective illness, mentioned that trazodone has not yet been reported to induce mania.

In our review of the literature we found only two case reports of trazodone-induced mania (Warren & Bick, 1984) and one report of psychosis following trazodone administration (Kraft, 1983). All three patients had unipolar depression.

In this report we describe three cases of trazodone-induced mania. Two of the patients are bipolar affective patients. To the best of our knowledge this is the first report of such reaction in bipolar patients.

Case 1

Mr P, an 82-year-old widowed male, met DSM-III criteria for mixed bipolar affective disorder: for the last 22 years he has suffered from manic and depressive episodes, each lasting 3–4 months. Since the death of his wife, 15 years ago, he was admitted several times to our hospital for short periods. In between these periods in hospital he enjoyed full remissions lasting up to 3 years. Mr P was readmitted due to a relapse into a depressed state. The examination

on his admission revealed depressed mood and affect, psychomotor retardation, weight loss, and lack of interest in life: "I had better been dead".

We started treatment with maprotiline, a drug which he had received intermittently for the last 5 years, but we discontinued it after 10 days since it caused confusion. We started treatment with trazodone, 50 mg/day, 7 days later and increased the dose to 100 mg/day the following week. After 2 weeks of treatment with trazodone Mr P regained his appetite, showed interest in everyday life of the hospital, and was given permission to visit his apartment. On the following day his mood became elevated, he worked day and night, cleaning his apartment, spent money without limits, and did not want to return to the hospital. When he finally returned, he showed pressure of speech, fanciful ideas, restlessness, and reported a marked reduction of sleep. Trazodone was discontinued and treatment with haloperidol was started. One week later Mr P's condition improved, and his mood and behaviour stabilised.

Case 2

Mrs N, a 35-year-old single woman, met DSM-III criteria for mixed bipolar affective disorder. For the last 15 years she had suffered from nine episodes of either depressed or manic states, each lasting approximately 5 months; in between she enjoyed full remissions. She was readmitted in a depressive episode with marked psychomotor retardation. Mental state examination on admission revealed depressed mood, slow speech, and rumination of inadequacy feelings. We started treatment with chlorimipramine, up to 250 mg/day, for 6 weeks, followed by imipramine, up to 300 mg/day, for another 6 weeks, with no improvement. We started treatment with trazodone 5 days after imipramine discontinuation and increased it in 4 days up to 300 mg/day. She demonstrated psychomotor agitation 5 days after the beginning of this treatment, hypereuphoria accompanied by irritability, and insomnia, resembling her behaviour during previous manic episodes. She even became violent, attacked one of the nurses and tore her uniform. Trazodone was discontinued and treatment with haloperidol was started. Within 4 weeks Mrs N returned to her premorbid state and was discharged.

Case 3

Mrs S an 84-year-old married woman, met DSM-III criteria for recurrent major depression: during the last 20 years she had several depressive episodes, each lasting 6-8 months, and did not have manic episodes. She was readmitted after a full remission of 3 years, 1 month after she underwent a nephrectomy for a malignant renal tumor. At that time her mood became dysphoric and she demonstrated loss of energy, reduced concentration, insomnia, and poor appetite. On admission her mood and affect were both depressed, and she revealed suicidal intentions. Her laboratory examinations including renal function tests and a whole blood count were normal. We started treatment with trazodone, and increased the dose up to 300 mg/day. After 8 weeks of treatment Mrs S's state improved: she slept

well, her appetite was better and she was more active. However, 2 weeks later she became very restless, talkative and easily distractible, showed inflated self-importance and tended to react angrily towards the staff and her visitors. She was running around the ward cursing the nurses and her family, and slept only a few hours (3-4) each night. This change lasted for a week. The dose of trazodone was reduced to 150 mg/day with a marked improvement, and follow up during the next 4 months found the patient in a good stable condition.

Discussion

The association between trazodone administration and the development of a manic state is supported by the following observations:

1. The symptoms appeared a short time after the beginning of the treatment in patients 1 and 2 (the bipolar patients). Patient 3, in whom the symptoms appeared after 10 weeks, had suffered only unipolar depression for 20 years.
2. The symptoms disappeared with the discontinuation (patients 1 and 2) or the reduction (patient 3) of trazodone.
3. The manic episode in the bipolar patients was different from the previous manic states: it lasted only weeks and resolved quickly following drug discontinuation and treatment with haloperidol.
4. The patients received trazodone without any other antidepressant drug. There was a 'wash-out' period of 5-7 days in the bipolar patients, and patient 3 received trazodone as the first antidepressant drug in this admission.

We assume that trazodone induced the manic reaction in the three patients described.

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A Case of Resistant Schizophrenia

In an era when it is generally believed that the acute symptoms of schizophrenia can be controlled pharmacologically, the case of a young man who has remained almost continuously floridly psychotic for 13 years, despite treatment, is disquieting. Conventional psychiatric treatment appears to be rendered impotent. It is in this context that it may be of interest to report a summary of the proceedings of a Special Problems Conference held at the Institute of Psychiatry on 18 February 1985 to discuss such a case.

Case report

Mr A is a 31-year-old single Caucasian first seen in 1971 with the diagnosis of schizoid personality disorder. In 1974, he was admitted to hospital for the first time, diagnosed as a paranoid schizophrenic. He had florid symptoms of schizophrenia including thought disorder with frequent neologisms, thought echo and broadcasting, delusional mood and persecutory delusions. Initially he showed some minimal response to phenothiazines but he remained an inpatient for nearly all of the next 3 years with only two short periods—of 21 and 10 days—when free of psychosis. Although virtually continually and floridly psychotic, there was a marked fluctuation from week to week in the degree to which the symptoms were evident. In addition, a consistent exacerbation of psychosis was noted with social stress. Treatment included a wide variety of antipsychotics as well as high-dose propranolol. He was finally transferred to the hostel ward in the Maudsley Hospital in 1977 and later to a local hostel in 1982. Over this period he became a little more sociable and less overtly psychotic, although some paranoid features remained. Drug treatment at this stage had been considerably reduced to haloperidol 15 mg daily. He voluntarily stopped all medication in December

1981 and 1 month later was noted to be increasingly psychotic and to show evidence of tardive dyskinesia. He was out of contact with hospital care until readmitted in an actively psychotic state in January 1983. He has remained an inpatient since, with no remission at all in his florid schizophrenia despite a range of antipsychotic medication, at times in high doses. He has become much more distressed on occasions and attempted to climb from a high level window. He was treated with a course of six ECT for a possible affective component to his disorder but with little benefit. Current medication is fluphenazine decanoate 100 mg weekly, chlorpromazine 200 mg four times daily and procyclidine 5-10 mg infrequently.

Background history

Mr A comes from a working-class Irish family and is the younger of two siblings. His father disliked social contact and left home when the patient was 18 months old, with no subsequent contact. Mr A's mother remarried a man considerably younger than herself when the patient was aged 14 and 1 year later she had the first of several myocardial infarctions. A maternal aunt died of Parkinson's disease, but no family history of psychiatric illness is known. The

family were very close-knit, living with the maternal grandmother until she died. He refuses any contact with his family now, asserting that "they are all replicas".

The patient was born by a normal delivery after maternal pre-eclampsia and induced at 36 weeks. His birth weight was only 2.04 kg (4.5 lb) and there were severe respiratory difficulties, requiring artificial ventilation. Information on the neonatal course is limited but mother was told that there was "no hope". Developmental milestones, nevertheless, were normal. He was an average scholar, but noticeably quiet and withdrawn, with few friends. Other than a few, short-term casual jobs after leaving school, he has been unemployed. He has no history of alcohol or drug abuse.

Present condition

Mental state

Mr A's mental state has not essentially changed over the last few years and shows a full complement of schizophrenic symptoms. He is often seen, a little dishevelled, in heavy army clothes, lying on the floor. He interacts little with other patients but on occasions he may participate surprisingly well in groups, showing considerable warmth. His expression is usually blank but he may be worried or suspicious; he may roll his eyes up voluntarily when particularly distressed. At these times his speech is often slurred. Usually, he will answer promptly, but rapidly goes off the subject to reveal marked thought disorder, one delusion following the other with no logical sequence and with frequent neologisms. He is apprehensive and worried, often about his physical health, but not thought to be currently depressed. On occasions he is much more distressed and seems tormented by his persecutory or hypochondriacal thoughts. Some ideas of reference and passivity experiences are present as well as thought echo, insertion and broadcasting. Both third person auditory hallucinations and somatic hallucinations feature. There is no cognitive impairment. Insight is generally minimal.

Physical examination

This did not reveal any neurological abnormalities.

Investigations

Investigations have all been normal including repeated CT scans, EEG and urinary drug screens. Serial psychometric testing shows no evidence of deterioration in the last 11 years: full-scale IQ has been consistently in the range of 85-95. Prolactin level is 939 mu/litre and chlorpromazine levels are: pre-dose 0.65 mg/litre, post-dose 0.75 mg/litre.

Conference

The conference was asked to consider two broad questions. What relevance do current biochemical theories of schizophrenia have in the light of the evidence of dopamine blockade in our patient? Secondly, how best should he be managed both in terms of situation and medication? Is his present environment too stressful for him? Is there any

justification for continuing medication at all now in a man who appears to be truly neuroleptic resistant? Alternatively, could any other drug be useful for this man?

Professor Griffith Edwards, chairing, introduced the discussion in an optimistic tone by commenting that currently we were making a case conference occasion of a schizophrenic who did not get better whereas even 30 years ago it would have been more probable that we should have been meeting to discuss with surprise a schizophrenic who had actually recovered.

Professor Malcolm Lader agreed but observed that this patient was different from most cases of refractory schizophrenia. Chronic schizophrenia is usually of the socially withdrawn type with a defect state and our patient was unusual in this his symptoms were so unrelentingly acute.

Author's note: Mr A is clearly an unusual case and poses considerable management difficulties. The literature, however, is not very helpful. Even the number of patients with persistent positive symptoms of schizophrenia is uncertain since many long-term follow-up studies (Sherman *et al*, 1964; Bleuler, 1972; Huber *et al*, 1975; Gelder & Kolakowska, 1979; Ciompi, 1980) do not specifically mention this group, presumably subsuming such patients into the category of 'chronic schizophrenic'. Earlier follow-up studies distinguished only between hospitalised and discharged patients. More recently, combined estimations using clinical, social and occupational measures have been introduced. Even so, as Bland & Orn (1978) make clear, rates of recovery in the literature are not always comparable. There is some agreement, however, that about 5-15% of schizophrenics remain continuously in hospital. The group who remain actively psychotic is probably smaller: in Huber's (1975) sample, 4.2% showed 'chronic pure psychosis'. A recent study of 77 schizophrenics by Kolakowska *et al* (1985) in Oxford suggested that at least 11 of those 20 patients with a poor outcome had been continuously ill since their first episode.

Professor Lader commented that even though the high plasma level of chlorpromazine may have been beyond the possible therapeutic range, there seemed little evidence to support the possibility of a toxic psychosis in our patient.

Dr John Crammer was particularly interested to pursue the possibility of depression at times of increased distress in this man. He emphasised that schizophrenics may well suffer from secondary affective illnesses, noting that the discovery of imipramine as an antidepressant had been made when treating schizophrenia and a mood change had been observed.

Author's note: Having excluded these possibilities, even then not all those schizophrenics with a poor outcome are necessarily neuroleptic resistant: both the nature of the schizophrenic illness and drug-related factors are important determinants. Gelder & Kolakowska (1979) have proposed a model in which they classify schizophrenic patients into four groups: remitters, responders, inadequately treated and neuroleptic resistant. The groups are distinguished on the basis of both clinical and pharmacological criteria. Thus, clinical improvements may occur because of either spontaneous remission or the effect of medication. Poor outcome, on the other hand, may result from suboptimal pharmacological effects of medication or because of true neuroleptic resistance. Clarification of this sort aids both clinical management and research.

Patients may be inadequately treated for a variety of reasons including non-compliance and individual differences in drug metabolism which alter dose requirements. Over 100-fold variations in chlorpromazine blood levels between different patients on the same dose have been reported (Curry *et al.*, 1970). There may also be extensive metabolism of an oral dose of chlorpromazine in the gut wall, accounting for the improved response that may be seen with a parenteral antipsychotic. The usefulness of phenothiazine levels has been debated at length: chlorpromazine, for example, has at least 168 metabolites, of varying potency. In addition, whilst there seems to be a relationship between plasma chlorpromazine level and plasma prolactin level, the association between chlorpromazine level and clinical response is less certain. Whether or not a 'therapeutic window' exists for neuroleptic treatment is similarly not yet established.

Professor Lader nevertheless suggested that medication could be withdrawn to see if any response occurred perhaps corresponding to such a window.

Dr John Cutting pointed out that not only had neuroleptics been disappointing in this man, but furthermore, it was unlikely that phenothiazines would induce remission in the future in the presence of thought disorder and possible antecedent brain damage.

Author's note: Most investigators have concentrated on this question: whether those patients with an unfavourable outcome can be predicted at an early stage. It is generally accepted that the following factors are all associated with a poor prognosis: low IQ, childhood difficulties, poor premorbid adjustment, presentation before 15 years, low social class, single status, insidious onset and absence of affective features. Huber (1975) found that those with somatic and auditory hallucinations and with a predominance of hebephrenic symptoms had an unfavourable prognosis. According to Murray (1984), however, positive symptoms are not of much importance prognostically. Negative symptoms such as emotional blunting and withdrawal are bad signs. Although it has been suggested that response to neuroleptic medication may depend on the stage of illness (Left and Wing, 1971), in Kolakawska's study (1985), both clinical response and outcome were related to the type of illness rather than its duration.

Much interest has centred on the possible association between 'organicity' and response to neuroleptics and outcome. Weinberger (1980) originally reported that clinical response was significantly worse in patients with cerebral ventricular enlargement. This finding was confirmed by Luchins *et al.* (1984). The Oxford study did not replicate this association (Williams *et al.*, 1985) nor did it find evidence for an association between the presence of organic brain disorder (both neurological signs and cognitive impairment) and poor outcome. Cognitive impairment alone, however, was related to the severity of the residual disorder and resistance to treatment (Kolakawska *et al.*, 1985). It is of interest to note that there was no evidence of such deterioration in Mr A.

How could organic brain disorder arise? Speculation has centred mainly for a putative role for perinatal trauma.

Dr Robin Murray was of the firm opinion that this case of schizophrenia was one in which the aetiology was obvious, pointing out the reported perinatal problems and

lack of a family history of psychiatric illness. Perinatal difficulties associated with later schizophrenia are most frequently in those without a family history of psychosis. Indeed, it has been suggested that there are two predominant aetiologies in schizophrenia: a genetic predisposition and environmental trauma to the brain. Perinatal trauma, Dr Murray maintained, was then clearly responsible for the illness in this man and it was only because of the insensitivity of current brain imaging techniques that the minimal brain dysfunction had not been demonstrated. He also felt that, although no studies had considered this question adequately, familial cases responded more readily to antipsychotics, perhaps implying a less fundamental role for dopaminergic abnormality than in the sporadic cases.

Author's note: That there are patients who are truly neuroleptic-resistant raises doubts about the way in which neuroleptic drugs are thought to have a therapeutic effect in more responsive patients. Indeed, it raises uncomfortable questions for the hypothesis that schizophrenia results uniquely from dopaminergic over-activity. In our patient, for example, there is convincing evidence of dopamine blockade. It seems that the assumption that schizophrenics are a homogenous group is becoming increasingly less tenable. Are neuroleptic-resistant patients dopamine independent? Have irreversible changes occurred in the central nervous system? These questions are evidently so uncomfortable that the conference was reluctant to pursue them.

Professor Lader did comment, however, on the history of Parkinson's disease in an aunt of the patient since it used to be thought that this was incompatible with schizophrenia in the same family.

Dr Stuart Checkley continued, however, that this was no longer thought to be the case. Different parts of the brain were predominantly affected; the corpus striatum in Parkinson's and probably the frontal regions in schizophrenia.

Author's note: Is there any more effective treatment for the unfortunate group of neuroleptic-resistant patients? A wide variety of treatments, including diet, acupuncture and ECT have been investigated in the past. That haemodialysis had no place in the treatment of schizophrenia was only convincingly shown as late as 1983 (Carpenter *et al.*, 1983).

The role of conventional neuroleptic in megadoses was reviewed by Aubree & Lader (1980). They conclude that a minority of psychotic patients may benefit from increased doses. This was, however, for a group of presumed fast metabolisers who were not, therefore, strictly neuroleptic-resistant, as our patient. Other antipsychotics have been relatively recently studied such as sulpiride, molindone, loxapine and clonidine. *Sulpiride* is a substituted benzamide which blocks dopamine (D_2) receptors which are independent of the enzyme adenylate cyclase. It may induce relatively few extrapyramidal side-effects and it has been claimed that the drug has both an anti-autistic and antidepressant effect. As an antipsychotic, it seems to be at least as effective as more conventional neuroleptics, but by virtue of its prolactin increasing effect, may induce galactorrhoea and amenorrhoea (Gerlach *et al.*, 1985). Both *molindone*, an indole, and *loxapine*, a dibenzoxapine derivative, have been investigated in the USA and are thought to be dopamine receptor blockers (Davies, 1976).

There is little evidence, however, that they are any more effective than standard antipsychotics although Lehmann *et al* (1981) reported three chronic, treatment-resistant patients who markedly improved on very high doses of loxapine. Transient numbness was reported in two of the three patients. *Clonidine* has received more interest in the UK. Although it may be an effective alternative to neuroleptics, its usefulness may largely be for those patients who have already responded to conventional antipsychotics but have troublesome side-effects (Freedman *et al*, 1982). Our patient would, therefore, be unlikely to benefit. *Propranolol* has been widely investigated as a treatment in schizophrenia, both on its own and as an adjunct to neuroleptic medication. Results from uncontrolled investigations are contradictory but a double-blind, placebo-controlled trial by Pugh *et al* (1983) showed propranolol used as an adjunct to have a more beneficial effect than placebo alone, although not as markedly as previously reported. The improvement was greatest after 3 months of treatment which may indicate the need for a prolonged trial in chronic patients. Our patient, however, was treated for many months with propranolol but with little effect. Propranolol is a β -adrenergic blocker but its mode of action in schizophrenia is not clear. It may possibly interact with 5HT receptors or, as more recently suggested, interact pharmacologically with neuroleptics. *Ceruletide*, an analogue of cholecystokinin, which is now known to be a central nervous system transmitter, has also been reported to be beneficial in schizophrenia in several open trials. A double-blind study involving chronic schizophrenic patients whose previous response to neuroleptics had been suboptimal found that there was no beneficial effects (Mattes *et al*, 1985).

In spite of the drawbacks of more recently introduced antipsychotics, could further medication be useful in our patient?

Professor Lader felt that it should first be ascertained whether cessation of medication had been followed by relapse or relapse was followed by cessation of medication.

Dr Checkley made the point that as attempts to block dopaminergic neurotransmission had failed to produce a therapeutic response, another line should be tried and suggested an attempt to block noradrenergic as well as dopaminergic neurotransmission. Neuroleptic drugs block α -adrenoceptors as well as dopamine receptors and amphetamines release adrenaline as well as dopamine. Hence, he went on, it was as logical to posit an over-activity of noradrenergic and dopaminergic mechanisms as it was to suggest an over-activity of dopaminergic mechanisms alone. Clonidine reduces noradrenaline activity by stimulating auto-receptors, thus reducing both noradrenaline release and firing. He felt that a trial period of clonidine was warranted in this man but acknowledged that those who had previously had a good response to neuroleptics seemed to respond better to clonidine.

Professor Lader wondered whether an amine depletor such as tetrabenazine or oxyperline could be useful if symptoms persisted in Mr A.

Dr Cutting, pessimistic of a response to antipsychotics in this patient, suggested instead that medication should be directed against specific abnormalities of the mental state such as anxiety or suicidal behaviour.

Dr Crammer remarked that in the past a schizophrenic with this degree of disturbance, unhappiness and unresponsiveness might well have had a leucotomy. One such patient under his care was continuing to do well after his operation in 1952.

Author's note: The problem, then, of the drug-treatment resistant patient remains unsolved pharmacologically. A more fruitful line of management may lie in a change of social environment. Our patient may well find his active ward environment too stressful.

Professor Lader agreed: there was, he thought, a strong argument for asylum in this man. It was, therefore, important to assess the relationship between his symptom severity and type of social pressures on him.

Dr Murray concurred, acknowledging the undoubted improvement in the quality of the lives of those schizophrenics who respond well to antipsychotic medication. He was, however, sceptical of the ability of the more resistant cases such as this to cope outside a non-hospital environment. This man had never done so and, in Dr Murray's view, it was cruel to keep attempting to place him in environments with levels of social stress that were harmful to him. He clearly required long-term care in an unthreatening asylum environment, such as he would have received 30 years ago. A combination, however, of therapeutic zeal on the part of psychiatrists and Government economies has resulted in the running down of asylums. He was encouraged that the effects of such a policy were at last becoming apparent with, for example, the publication of the recently highly critical report of a Select Committee of the House of Commons (1985).

Author's note: Manfred Bleuler was not an advocate of heavy and prolonged use of powerful antipsychotic drugs; he emphasised the importance of patients' environments and in particular the need for therapists to relate to the healthy aspects of psychotics. This conference, however, paid scant regard to a more psychotherapeutic approach. Is there any role for psychotherapy in such patients? A few studies have looked at this question but they are fraught with methodological problems (Feinsilver & Yates, 1984; Tuma & May, 1979). Whilst it would be a brave psychotherapist indeed who would take on our patient, that should not preclude striving for some empathic understanding with the psychotic schizophrenic. This was clearly illustrated by Van Putten *et al* (1976) who found that grandiosity was the most powerful discriminant between drug defaulters and compliers. The former may well prefer their delusions of grandiosity to drug-induced normality.

Professor Edwards acknowledged that the conference would have gained from hearing more of a psychotherapeutic view. He emphasised that we should be aware that the framework of the conference discussion—largely biochemical, neuropsychiatric and genetic—was a function of current thinking and within a given context. It could, therefore, change. Indeed, there were hints of such a change in the parameters of the debate with the importance attached in the conference to the milieu of the hospital and more broadly in the work of Dr Julian Leff.

Before bringing the discussion to a close, Professor Edwards was keen to consider the prospects for future research in this country. Should we concentrate our efforts

in investigating antipsychotic medication, for example, into a biological approach using animal models or use a more clinical approach?

Dr Checkley clearly indicated the advisability of large-scale empirical testing.

Professor Edwards doubted whether this country had a sufficient clinical research organisation for such a programme to take place.

Author's note: The parameters of the debate can be seen quite clearly to include more political issues also. Further research is urgently needed for neuroleptic-resistant patients such as ours. Current treatment offers little and he remains floridly psychotic and very distressed.

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Panic Attacks

SIR: Gelder's review of panic attacks (*Journal*, September 1986, 149, 346-352) provides opportunity for a further comment on this topic. Gelder does not conclude that hyperventilation is a cause of panic attacks, but he does lean to the view that lowering of arterial CO₂ may be a contributory factor. That hyperventilation is the principle cause is fast becoming a fixed belief, and some arguments in support of this are set forth by Hibbert (1984). Furthermore, a therapeutic technique based on the induction of the symptoms of tetany (listed in the second column of Gelder's table), and cognitive restructuring of the consequent apprehension, supposedly supports the argument that lowering of arterial CO₂ is an essential component in the development of a panic attack. Before this view becomes dogma and is taught as fact to generations of clinicians in training, the contrary evidence should be considered.

Firstly, there is the fact, recognised by Gelder, that not all patients who develop panic attacks hyperventilate. Secondly, Hibbert (1984) states that people who hyperventilate become anxious—but this is contradicted by observation and research (Bass & Gardner, 1985).

The third misleading assumption is that the tetanic symptoms of hyperventilation are the same as those experienced during a panic attack; some of the somatic symptoms, e.g., paraesthesia, may be common to the two states but the 'apprehensive feeling' of hyperventilation is of a different order to the overwhelming anxiety accompanied by fear of dying or becoming insane which is the pathognomonic feature of a panic attack. Fourthly, the relationship of CO₂ to the neuronal systems mediating the experience of anxiety is complex, but other evidence points to the conclusion that *raising*, not lowering, arterial CO₂ induces anxiety (Gorman *et al.*, 1984; Woods *et al.*, 1986). Whereas the proponents of the hyperventilation hypothesis induce anxiety by deep breathing in their therapeutic technique, others (e.g., Griez & van den Hout, 1983) use inhalations of CO₂ to induce anxiety.

The categorisation of panic disorder in DSM-III followed the observation that this form of anxiety may respond to antidepressant drugs and this implies a 'biogenic' explanation of the disorder. I have the impression that the hyperventilation theory has

gained ground among those who wish to establish an alternative 'psychogenic' explanation. Much of the problem, as is always the case in psychopathology, rests on the definition of states; the reading of many research reports and some books on the topic leads me to the conclusion that the flexible word 'panic' is used in different senses and to support different views. No doubt debate is in progress as to whether ICD-10 should follow the example of DSM-III and categorise 'panic disorder'. If so, we must all be sure what we mean. It seems to me that the debate is irrelevant: West & Dally (1959) described another form of anxiety disorder responding to antidepressants before the observations of Klein (1964) which were the impetus to the DSM-III category. Of greater importance than categorisation is the establishment of clearer clinical guidelines as to which forms of anxiety disorder may respond to pharmacotherapy and for which forms psychological therapy is the procedure of first choice.

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Childhood Encopresis Extended into Adult Life

SIR: Fraser & Taylor (*Journal*, September 1986, 149, 370-371) wonder how many people have encopresis

extending into adult life. We report a small follow-up study which involved 22 teenage soilers (4 girls and 18 boys) admitted to an in-patient psychiatric adolescent unit who were followed up for 2–11 years (mean = 5 years; s.d. = 2 years) after discharge. They had been treated at High Lands Adolescent Unit, Scalebor Park Hospital, Burley-in-Wharfedale, between 1973 and 1983. Mean age on review was 19 years (s.d. = 4 years, range = 15–25 years); mean age at the time of admission was 13.5 years (s.d. = 3.7 years). All were of normal intelligence, and social class was mostly III (Registrar General's Classification). There was associated nocturnal enuresis in eight of them. Average duration of stay in hospital was 5 months (s.d. = 2 months); two were re-admitted. Twelve of them were considered to be conduct disordered, one had obsessional neurosis, one had a severe anxiety state, and one was depressed. Treatment consisted of toilet training, often with supplementary laxatives, therapy and family counselling.

Family doctors provided information on follow-up in all instances. Five former patients agreed to be interviewed, and another eight agreed to complete a review questionnaire. These 13 completed the 60-item GHQ and the Leeds Scale for Anxiety and Depression. It was found that there were three definite soilers between the ages of 17 and 23.

Case reports: (i) A 23-year-old man had been treated at the age of 16 with severe soiling and conduct disorder. Corroborative information came from both questionnaire and family doctor.

(ii) A 19 year-old man who was employed on a high rise building-site had been treated at the age of 14 years, and had subsequently been re-admitted for a recurrence of soiling. He was interviewed at follow-up and said he had a tendency to soil when out of reach of toilets.

(iii) An 18 year-old man who had been admitted at the age of 14 had been considered as conduct disordered and had nocturnal enuresis. Information in his case came from the family doctor. He was still soiling.

None of these three showed any evidence of psychiatric disorder on the GHQ or Leeds Scales.

The family doctors provided a control group selected randomly from their lists. None showed any evidence of soiling, although one 18 year-old had been treated for faecal impaction without soiling. For the 13 who completed the GHQ the mean score was 3.6 (s.d. = 1.9). One individual had a score of 11, indicating possible psychiatric disturbance. He had been treated by a psychiatrist. The mean anxiety score on the Leeds Scale for the 13 was 4 (s.d. = 2) and for depression was 2.3 (s.d. = 1.5).

We would like to thank Mrs B. Morris, who greatly assisted in the carrying out of this small project

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Clopenithoxol in Aggressive Mentally Handicapped Patients

SIR: We read with interest the report by Mlele & Wiley (*Journal*, September 1986, 149, 373–376). They conclude that "clopenithoxol decanoate may be useful in reducing aggressive and disruptive behaviour in mentally handicapped adults". But has this finding any reliability? We do not know whether the effect was specific to depot clopenithoxol because "none (of the patients) had been given depot phenothiazines" (presumably meaning depot neuroleptics, because only one depot phenothiazine is available). We cannot be certain that there were beneficial effects, because the ratings of behavioural changes were crude, as the authors observe, and even more importantly, they were not carried out blind to the treatment. A control group having placebo injections would be essential.

The paper is further weakened by the absence of diagnostic criteria for the use of the medication. It is disturbing that potent medication given by injection is being advocated for non-specific behaviour disturbances, which invites accusations of the use of 'chemical strait-jackets'.

We appreciate the diagnostic problems in people with mental handicap and the difficulties in their clinical management, but we think that the administration of drugs for behavioural control, rather than for the treatment of specific diagnosable illness, needs justification. With contemporary multiprofessional interest in behaviour modification techniques and non-institutional care, doctors need to be clearer about the indications for intervention with medication, rather than advocating its use on non-specific grounds.

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Cost of Community Psychiatry

SIR: Hoult (*Journal*, August 1986, 149, 137–144) is to be commended for the enthusiasm and enterprise he

has shown in developing an alternative to hospital-based psychiatry in Victoria. However, we are concerned that the figures quoted in his article may be uncritically repeated as showing that his alternative form of psychiatric care, based on heavy community involvement and boarding house care, is cheaper than conventional hospital-based psychiatry and should make hospital care redundant. The standard method of calculating such figures involves the cost of each in-patient day taken as a fraction of the total expenditure of that institution on clinical work. This figure is usually fairly large, and is therefore unlikely to be exceeded by other costs, even when these are relatively complicated such as setting up the boarding house supervision described in Hoult's paper.

Provision of community-based psychiatry, even when it is as effective as that described by Hoult, does not necessarily reduce the cost of hospital psychiatry. A full range of services is necessary to treat the most disturbed patients, a service that was necessary for even some of Hoult's intensively treated experimental group, and if such a service is to be maintained the total cost per in-patient will rise as the number of beds is reduced. Recently, following expansion of our services into the community, we were able to reduce the acute in-patient services from our inner-city sector in Nottingham (population 70 000) from 30 to 18 beds. This involved transfer to a different ward, and was expected to be accompanied by a reduction in nursing staff. However, because of the need to maintain adequate numbers of well-trained staff to deal with the smaller numbers of often disturbed patients, the number of nurses on the ward was reduced by only one. If we had used the standard cost-effectiveness calculations as described by Hoult our community-based service would appear a great deal cheaper, as there have been fewer admissions staying for a shorter time. Significant savings can only be made by running down the hospital end of the service. This would be quite inappropriate, and would lead to a two-tier system whereby all the best care was given in the community and those who failed to remain in community care would receive an inferior form of custodial care in hospital. For this reason we advocate that all community psychiatry should be closely linked with a hospital base so that such a two-tier system cannot develop. The hive system of care (Tyrer, 1985) emphasises the need for staff to work from a hospital base but to have extensive community involvement and, in the longer term, this can only be achieved by rotating staff between hospital and community activities. This has been tried in Sweden (Perris *et al*, 1985) and preliminary results are encouraging.

Despite the enthusiasm and effort of Hoult's experimental team, the fact that some patients needed to be admitted to hospital demonstrates that hospital and community psychiatry cannot exist in isolation. The future lies in integrating the essential components of both, not in abandoning one at the expense of the other.

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Witchcraft and Psychotherapy

SIR: The article by Neki *et al* (*Journal*, August 1986, **149**, 145-155) is not only of relevance to mental health professionals in Africa and other areas of the Third World, but also to those working with immigrant populations in Western countries. The review confirms that beliefs in magic causation are not abandoned under 'Westernising' influences in Africa; in fact, magic beliefs and practices are often intensified under conditions of rapid socio-cultural change, with its attendant psychopathology. This was apparent twenty years ago (Jilek, 1967).

The article conveys an overall negative view of African traditional healers by using the label witch-doctor in a pejorative sense. In colonial times witch-doctor was the blanket label for all 'native' diviners, healers, medicine men, magical ritualists and herbalists. The demise of colonial rule brought a re-evaluation of non-Western therapeutic systems and a more positive assessment of traditional therapists (Jilek, 1971). Neki *et al* convey the impression that witch-doctor is more or less synonymous with sorcerer or witch. There are, however, quite distinct categories of traditional diagnosticians and therapists, with different functions and often specialisations, everywhere in Africa, including Tanzania and even in smaller tribes, as we found out in field work among the Wapogoro (Jilek & Jilek-Aall, 1967). It is regrettable that Neki *et al* fail to find any usefulness in the collaboration between Western-trained health staff and traditional practitioners, pioneered with considerable success by Lambo and his colleagues in

Nigeria (Lambo, 1964). They hint that such collaboration is "undesirable on moral grounds". This authoritative censure is made in connection with a reference to our co-operative relationship with traditional healers in a different culture area, that of the Northwest Coast Indians of North America. The therapeutic ceremonials described (Jilek & Todd, 1974) have nothing to do with witches, witchcraft or sorcery. The witch-doctors referred to in our article endeavour to assist, by means of a culture-congenial traditional psychotherapy and sociotherapy, those North American Indians who under the impact of rapid socio-cultural change are showing symptoms of anomic depression and anxiety.

While working with tribal societies in three continents, I have had the experience that friendly contacts and monitoring collaboration with traditional practitioners who have an established role in their community is more beneficial to the patient than ignoring or condemning them. Such collaboration becomes a necessity in the planning of comprehensive primary health and mental health care in a developing country with limited professional manpower resources (Jilek, 1985).

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Depression in Schizophrenia

SIR: The paper by Elk *et al* (*Journal*, August 1986, 149, 228–229), looking at rates of depressive symptoms in schizophrenic patients from three "racial" groups, raises a number of issues.

One might question results based on small numbers (groups of 19, 15 and 22 patients) but, more importantly, is it methodologically valid to compare three such disparate groups without considering other variables? No information is given in the paper about patients' social circumstances or how typical they are of patient populations in the three "racial" groups. Referees and readers of the *Journal* may not know that black, coloured and white patients in South Africa by law have to be nursed in separate wards, usually separate hospitals, and that black and coloured people have extremely limited access to medical care.

A further point is that the reasons for comparing these three groups are not explained; is it because they were thought to be biologically different or were from different social backgrounds? Referees should ensure that the assumptions underlying a research project are clarified for the readership in either the introduction or the discussion.

Finally, there are many who would suggest that at the present time, papers from South Africa should not be published by British journals. It would be helpful to have some editorial comment on this issue.

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Macrocytosis and Cognitive Decline in Down's Syndrome

SIR: Welfare & Hewitt (*Journal*, April 1986, 148, 482–483) suggest that in Down's syndrome cognitive decline and macrocytosis may be related. The causal relationship they suggest is unlikely. While red blood cell (RBC) membranes may age more rapidly in patients with Down's syndrome, this would lead to these RBC becoming smaller rather than larger. The macrocytosis seen in patients with accelerated ageing of RBCs due to thalassaemia or haemolysis is caused by the large size of the reticulocytes produced in response to the RBC loss (Beard, 1978).

Welfare & Hewitt state that in their Down's syndrome patients "there was no evidence of vitamin B12 or folate deficiency to account for this further increase" in RBC size. Eastham & Jancar (1983) state that Down's syndrome patients with macrocytosis were "not being treated with anticonvulsants and were not anaemic". It is well recognised that functionally significant folate deficiency in association with macrocytosis may be present in patients without megaloblastic anaemia being apparent (Botez &

Reynolds, 1979). I would suggest that macrocytosis and cognitive decline in Down's syndrome is likely to be related to undetected folate vitamin deficiency consequent on institutional nutrition, complicated by the gastrointestinal malabsorption that some Down's syndrome patients have.

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Dependence on Pseudoephedrine

SIR: Sympathomimetic amines are a major ingredient in proprietary medications for the treatment of upper respiratory tract infections. We describe a patient dependent on pseudoephedrine.

Case report: The patient first presented at the age of 21, with symptoms of depression. This responded poorly to treatment, and continued for 12 years. In the eleventh year she was convicted of the theft of a medical prescription pad and for making a prescription for herself for Actifed. She subsequently admitted taking between 50 and 300 ml/day of this preparation — the recommended dosage is 30 ml/day. She said that she took it because it "gave her a lift". A year later she began to describe psychotic symptoms, which have lasted for four years. She had auditory and visual hallucinations and passivity feelings. These symptoms fluctuated and were variable in content; she showed none of the negative symptoms of schizophrenia. Although it was not possible to make a diagnosis of schizophrenia, she clearly suffered from depression, had an unstable personality, and had abused Actifed. Treatment included both oral and depot phenothiazines in addition to supportive psychotherapy, but her compliance was poor.

The present report is important because it shows that dependence and possibly psychosis can occur with over-the-counter preparations. Each 5 ml of Actifed contains 30 mg of pseudoephedrine hydrochloride and 1.25 mg of triprolidine hydrochloride. Dependence on amphetamines and other sympathomimetic drugs is well known. There is, however, only one report of dependence on pseudoephedrine (Diaz *et al*, 1979). Visual hallucinations on attempted withdrawal are described in that report. Paranoid psy-

chosis after abuse of Actifed has also been described (Leighton, 1982). The patient's fluctuating psychosis is thus in accordance with previous findings.

We thank the Wellcome Foundation and Mr P. Stone for their help

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Admission Rates and Lithium Therapy

SIR: Dickson & Kendell (1986) recorded admissions for mania and psychotic depression in Edinburgh over the years 1970–1981 and found a rise which they could not explain. They had expected a fall, because the use of long-term lithium therapy had increased ten-fold during the same period, and they felt that their findings cast doubt on the efficacy of lithium prophylaxis in ordinary clinical practice. Such a conclusion might have far-reaching consequences for patients with recurrent manic-depressive illness, and Dickson & Kendell's analyses merit close scrutiny.

It seems a dubious procedure to draw conclusions about the efficacy of a treatment given to a limited number of patients from admission rates for a much larger number. Not all manic-depressive patients receive prophylactic lithium treatment: it is given only to those with frequent recurrences and is started only after the patients have had several episodes. So even if lithium treatment were 100% effective, it could be expected to prevent only a fraction of the admissions for mania and depression.

Even so, a fall in the admission rate, albeit a small one, would be expected if lithium treatment was the only factor influencing admissions. It obviously was not; powerful forces with an opposite effect must have been at work. Dickson & Kendell examined some factors, such as change in diagnostic fashions or admission thresholds, but were unable to account for the rise. One could think of several others.

Be that as it may, the fact remains that the admission rate for mania and depression showed a pronounced rise, and the rise must have been caused by something. The moderate effect of lithium may

easily have been overshadowed by the marked effect of the unknown factor or factors, and Dickson & Kendell's inference about the poor effect of lithium therapy under ordinary clinical conditions is not vindicated by their data. Examination of rates of readmission for manic-depressive patients actually in long-term lithium treatment might have provided information more suited to assess the efficacy of lithium prophylaxis.

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Community Care of the Acutely Mentally Ill

SIR: Whether Hoult's study (*Journal*, August 1986, 149, 137-144) shows that "it is feasible to treat most psychiatric patients in the community as an alternative to mental hospital care" is open to doubt.

For example, 40% of his experimental group required hospital admission at some time. Certainly this was much less, in terms of numbers and duration, than the control sample, but the latter's rates for hospital admission seem inordinately high. It appears that admission was almost automatic for any control group patient turning up at the hospital. Furthermore, there is for some reason a significant increase in neurotic syndrome scores for this control group, and over one fifth are lost to follow up, despite being local residents. It is also worrying that the most diffi-

cult cases in psychiatric practice (those over 65, mentally retarded, brain-damaged and drug/alcohol dependent) are excluded. It is certainly reasonable to have a refined sample for research into aetiology, but assessment of care must surely include all the complicated problems that so often provide the greatest management difficulties. Finally, I am uncertain as to the basis of his costing of the alternative care systems. Does he include the on-call availability of psychiatrists "covering the rest of the catchment area service", and the fixed costs of running a hospital anyway — (regardless of patient usage) — since his patients certainly had to use this resource?

It seems that the problem in this work is the "degree of artificiality" created by a research project. The "assertiveness", 24 hour availability, enthusiasm and job satisfaction of the staff involved are crucial factors, and a false dichotomy between hospital and community care is set up. Yet it is not the "where" that matters, it is the "what" and "who" that are essential. Hoult and his workers have clearly shown that the quality of care, at the personal level, is central to a good outcome in psychiatric illness. They have fallen into the non-scientific trap of obtaining findings that "confirmed our aims" — (where is the null hypothesis in this?) — and have not pursued the obvious control situation, namely putting their motivated research team, and its methods, into the hospital. While it is gratifying to see their success — and the value of having a psychiatrist on-call continually — it may be more realistic to see the hospital as a community resource and not to create either/or contrasts.

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A HUNDRED YEARS AGO

Asylum Reports

YORK. DR. HITCHCOCK REPORTS: "During the winter months I have been giving a course of lectures on elementary anatomy, physiology, and the immediate treatment of injuries and accidents, etc to the nurses and attendants of the asylum. I was much gratified by their regular attendance and the interest manifested in the subject; the more so that several, not specially engaged in attendance on the patients, were amongst my class; even some of the patients asked if they

could attend, but I drew the line there. I shall not mind if the knowledge they gained is not tested by practice; but at all events the subject was right and necessary for them to be instructed in, and if all's well I shall give a similar course next year."

Reference

Journal of Mental Science (April, 1886) 32, 146

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.

Book Reviews

Multiple Personality, Allied Disorders and Hypnosis.

By EUGENE L. BLISS. Oxford: Oxford University Press. 1986. Pp 271. £28.00.

I very much enjoyed reading this book. It combines a scientific review of multiple personality and of hypnosis in which the author puts forward an argument for the conceptualisation of multiple personality as a form of self-hypnosis together with a description of his extensive therapeutic experience with multiple personality using this theoretical framework.

His thesis states that people vary in their capacity for self-hypnosis and some of those who are more gifted have used this ability usually since childhood to cope with traumatic events, by the assumption of a different persona with amnesia for their normal persona. In the less extreme forms of the syndrome they use this facility to share difficulties with one or many alter egos. These subjects almost always show a high capacity for hypnosis as judged both clinically and on the Stanford Hypnotic Susceptibility Scale. The phenomena of hypnosis, e.g. hallucinatory experience, regression, etc. fit readily into the symptomatology of multiple personality.

Professor Bliss argues that far from being an exotic syndrome, the alerted psychiatrist will commonly find cases misdiagnosed under other labels. A substantial number of patients diagnosed as schizophrenic prove to have multiple personality, although he does point out that the patients he investigated had been diagnosed on looser American criteria and not those of the International Study. His therapeutic approach is to gain access to the patient's other personalities through hypnosis and to help them gradually to accept their experience and feelings into normal consciousness and either to integrate all the personalities into his/her own or more usually to arrange a reconciliation between them.

Professor Bliss puts great emphasis on eliciting earlier traumatic experience which has often been sexual and freely acknowledges his debt to Janet and Breuer. His approach has much in common with psychoanalysis in the pursuit of unconscious material, at times by free association. However, he is often directive and uses a variety of other strategies in the therapeutic process.

It seems likely that whatever the arguments about aetiology, his patients find it easier to discuss their problems at arms length by concretising

them into alter egos and the therapist must then use his skills to help the patient to accept the data and its associated feelings into consciousness with subsequent resolution.

It may well be heuristically useful to think more often of the diagnosis of multiple personality in our patients, and challenging to follow the therapeutic path that Professor Bliss has mapped out for us. He emerges from these pages as a very gifted, intuitive and persistent therapist whose example it will not be easy to follow.

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Aspects of Epilepsy and Psychiatry. Edited by MICHAEL R. TRIMBLE and TOM G. BOLWIG. Chichester: John Wiley. 1986. Pp 256. £24.50.

In recent years there has been a great increase in the number of books published on epilepsy. One of the editors of this volume, Michael Trimble, has contributed to this change. Is there really more to write that is new, informative and helpful in the management of epilepsy for the psychiatrist? The answer in relation to the present volume must be a qualified 'yes'. Both chapters by Trimble are of value, the observations on hypographia of interest, while those on PET scanning potentially of much greater importance. The hypo-metabolism found in the inter-seizure records in temporal lobe epilepsy, particularly the dominant hemisphere of psychotic patients, is a notable feature. The changes themselves extend beyond the temporal lobe and the limbic system to frontal cortex and the basal ganglia. There is some evidence of a relationship between these reported findings and those in non-epileptic psychotic patients. The electrophysiology of psychosis is dealt with and the puzzling phenomenon of 'forced normalisation' is well reviewed. Emphasis is placed not only on the electrophysiological aspects, previously the main plank of this concept, but giving it a more clinical connotation. There has always been conflict about the possible specificity of personality disorder found in epilepsy as well as whether the temporal lobe seizure disorders have a greater likelihood of psychosis than patients with a primary generalised fit pattern. Here the great knowledge and experience

of Janice Stevens, with information from pathological studies on six cases of epilepsy and psychosis, furnish more evidence to her stance on non-specificity. The difficult topic of aggression in relation to epilepsy is further evaluated but problems still remain. The fact that hypo-sexuality occurs in epilepsy has long been recognised, but the possible underlying hormonal basis is fresher and merits the space afforded by the editors. Clinical aspects such as the impact of chronic handicap and the effects of anticonvulsants are also discussed as well as the treatment of depression, the commonest psychiatric disorder requiring active management with psychotropic drugs. The use of anti-convulsants in non-epileptic conditions is reviewed at length. This remains one of the most controversial areas perhaps requiring less emphasis for the general reader who is otherwise well served by this volume.

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Depression in Young People—Developmental and Clinical Perspectives. By MICHAEL RUTTER and CARROLL E. IZARD. New York: Guilford Press. 1986. Pp 550. £34.50.

The steady growth of interest in depressive disorders in childhood and adolescence is exemplified by this volume, in which developmental and clinical approaches are brought together to present a range of current views and a number of research findings. The 18 chapters have their origins in a 1982 symposium held in the United States, and emphasise development and clinical perspectives. Much of the original material has been revised and brought up to date.

A vast landscape is surveyed here, and the reader will be wise to begin at the beginning. In the prologue, Rutter discusses the developmental psychopathology of depression with customary thoroughness, marshalling the evidence to show that depressive disorders change in form and frequency as children develop into adolescence, and pointing up the clinical distinction between childhood distress of an essentially developmentally normal type, and recognisable depressive disorder which involves biological and cognitive changes, as well as alterations of mood. Contributors to Part 2 examine theoretical approaches to the development of depression, in social, cognitive and biological terms. The risk factor of parental depression is discussed in Part 3, as a genetic influence and as an environmental, day to day presence in the lives of significant numbers of

children. The way in which such parental depression may alter patterns of family interaction receives attention, as does the important question of how affective disorder in a parent may have effects on a child's competence and vulnerability. Risk indices and mechanisms are discussed in Part 4, which also touches on parental loss, stressful life events, and biological factors. Part 5 is concerned with measurement and includes examination of developmental issues in the classification of depression. The work concludes with a forward-looking research postscript.

A number of the contributions particularly attracted your reviewer's attention. Emde, Harmon and Good present a valuable consideration of how depressive feelings may alter during the course of development. Seligman and Peterson demonstrate ways in which the learned helplessness model of depression in adults may also serve as an aetiological model for affective disorder in childhood. There is a comprehensive review of psychobiological research into childhood depression by Puig-Antich, the findings being discussed in relation to what is known of biological markers in adult affective illness. Carlson and Garber's consideration of developmental issues in the classification of depression in children, and Kovacs's examination of the potential of the clinical interview as a tool for the diagnosis of depression, are two especially enjoyable sections.

The editors have largely been successful in steering a safe course between dull uniformity of presentation, and the unevenness which at times can impair large scale multi-author works. Occasionally there is some feeling of repetition across different chapters, but this is balanced by one's satisfaction at the cross-fertilisation which is often apparent, as for example when Dunn, writing on stress, development, and family interaction, amplifies and extends some of the remarks made by Shaffer in his chapter on developmental factors in child and adolescent suicide.

Clinicians are unlikely to find this an easy book, despite the clinical perspective of the title. The practising child and adolescent psychiatrist wondering about how to improve treatment of children and adolescents with affective disorder will not find answers here, for little is said on management. What will be immediately apparent to the reader, however, is the ample stimulation provided for a reconsideration of exactly how the affect of a young person can become disordered, and of the influences which may determine the progress of such disorder. The volume will be important as a work of reference, which should certainly be in every psychiatric library. But it is as a source of ideas, and a guide to some current thinking, that it should ultimately be

considered and on these criteria alone, it deserves the closest attention.

HUGH MORTON, *Consultant Child & Adolescent Psychiatrist, Ninewells Hospital, Dundee*

Alcoholism and Drug Addiction. By D. RAISTRICK and R. DAVIDSON. Edinburgh: Churchill Livingstone. 1985. Pp 198. £18.00.

This is the second volume in the new series of *Current Reviews in Psychiatry*, the editors of which are E. S. Paykel and H. G. Morgan, intended to provide succinct, balanced and topical reviews of the many advances now taking place in psychiatry, for the benefit of postgraduate and practicing clinicians alike. The book consists of 6 chapters, each of which ends with concluding remarks and a list of relevant references, and a 6-page index.

The first is an introductory chapter giving some historical background, a useful account of various terminologies and an interesting model of drug-seeking behaviour. The authors emphasise the importance of a multi-disciplinary approach to the problem of drug dependence as a whole, rather than that of distinction between drugs. This is a philosophy now widely regarded in the biomedical world of drug dependence as the best likely path for advancing our knowledge in this area. In Chapter 2 'Drug Effects and Physical Harm', the authors provide short and useful accounts of factors influencing drug effects as well as descriptions of such effects at the pharmacological and clinical levels. The authors, however, take a somewhat narrow view of the possible mechanism(s) of alcohol dependence and make the erroneous statement that MEOS (the microsomal ethanol-oxidising system) is responsible for the metabolism of certain drugs other than alcohol; they probably mean the microsomal cytochrome P-450-dependent mixed-function oxidase system.

The third chapter 'Psychiatric Disorder' considers psychiatric symptomatology in alcoholism and drug abuse neatly and concisely in relation to the 4 major groups of disorder (affective states, delirium, paranoid psychosis and dysmnestic states) and emphasises the importance of both good knowledge of psychiatry and deeper insight into psychiatric symptoms in alcoholism and drug dependence patients. Chapter 4 'Social Aspects' provides a short historical account and considers aspects such as the integration model, parental attitudes, peer pressure, personal characteristics, environment and the deviance theories. The authors also describe the consequence of misuse of alcohol and drugs and emphasise that a major problem in social research in

this context is that of cause, effect and coincidental findings.

The fifth chapter 'Treatment' considers this aspect in its broad sense, from initial interview to rehabilitation, again emphasising commonalities in the management of both alcohol and drug dependence, and addresses issues such as longitudinal views and studies, current issues in treatment provision, methodological aspects, treatment intensity and setting, patient and therapist characteristics, pharmacological treatments, therapeutic communities and the various psychological approaches to therapy. In Chapter 6 'Prevention', the authors, after a brief account of alcohol and drug consumption, discuss control strategies, alcohol availability and fiscal measures, then move on to prevention strategies (education, media campaigns and advertising), emphasising the need to consider a number of important research and policy issues in this context and to develop a comprehensive and informed prevention policy.

This book is well written and nicely produced, with very few typographical errors, and its content will provide an easy and concise introduction to the psychiatric student of alcoholism and drug dependence.

A. A.-B. BADAWY, *Top-Grade Biochemist, Whitchurch Hospital, Cardiff*

Psychology for Social Workers. Revised 2nd edition.

By MARTIN HERBERT. Basingstoke: Macmillan. 1986. Pp 322. £18.00 hb, £7.95 pb.

Psychology for Social Workers is one of a series of books published by the British Psychological Society. The stated aim of the series is to "... illustrate how psychology can be applied in particular professional contexts, [and] how it can increase the skills of practitioners..." A worthwhile aim which bears somewhat patchy fruit on this occasion.

The current volume is arranged into two parts. The first, written by the author Martin Herbert, sets about describing the clinical psychologist's basic set of concepts. Thereby, we are introduced to the terminology of the psychologist; and perhaps more importantly, to the basic assumption on which this discipline rests. That is, the psychologist begins with a person's behaviour. Whilst there is interest shown in the motives which arise from inner world factors, such as feelings, fantasies, needs and so on, the emphasis is on how current behaviours are reinforced and can be used to predict how a person may behave in the future. The implication is that the practitioner can thus devise strategies to change particular behaviour patterns.

The second and most extensive part, includes contributions from a range of authors who attempt to link psychological theory to specific areas of practice. These include a number of contexts where social workers would expect to find themselves; for example, in institutions, or in casework with the aged, those people who are dying and bereaved, and with families. This seems to be an attempt to describe how theories of psychology can be used to orient social workers in their *thinking* about their practice. Useful illustrations of this will be found in the chapters on The Behavioural Casework Approach (Ch.11) and Counselling and Helping (Ch.12).

From a practicing social worker's point of view, the major limitations of this book lie in the case illustrations. It appears that the authors are mostly based in educational or training settings, and this may account for some of the textbook flavour to be found. With this in mind, I would have little hesitation in saying that students who are training as social workers, and professionals in the medical world, will find this a useful companion to their lectures and seminars on the theory of psychology.

PETER FULLERTON, *Psychiatric Social Worker, The Emanuel Muller Clinic for Families and Children, London*

Schizophrenia: An Integrative View. Edited by C. L. CAZULLO and G. INVERNIZZI. London: John Libbey. 1985. Pp 402. £22.00.

This volume, which forms part of a series entitled *Biological Psychiatry—New Prospects* is a collection of papers from a conference held in Milan in June 1984. 42 papers by 101 authors, 66 of them Italian, are included.

The papers are arranged in four parts. The first, 'Cultural and Social Aspects' opens with a paper by the senior editor that attempts to live up to the book's title by incorporating discussion of symptom pattern, lithium, social intervention and movement related brain macropotentials to conclude that "a mental disorder like schizophrenia can be understood by an interpersonal approach and consequently has to be always treated within an interpersonal relationship". This section is devoted mainly to brief essays on epistemology and there is little presentation or analysis of scientific data. Hamilton's brief but sparkling discussion of the influence on the historical development of ideas of the world of schizophrenic thought disorder is, however, a delight.

The second section 'The Psychic Milieu' contains a good review by Andreasen of recent progress in

increasing the reliability of measures of schizophrenic symptoms, and of the persistent problems in the diagnosis and subclassification of the disease. Also interesting is Invernizzi on psychodynamic aspects of psychosis following kidney transplantation.

The third and meatiest section is on 'Biological Aspects'; the best of these papers, by Shagass *et al* on brain electrical potentials, and by Nurnberger *et al* on genetic markers, combine scholarly review with presentations of original research data. The final part, 'Integrative Strategies for Treatment' is thinnest on the ground, though there is a clear review by Maj and Kemali of the developing concept of schizoaffective disorders and an extraordinary essay by Reda on the history of 'shock' treatment which includes fuzzy illustrations of a 'rotating chair' and of 'synapsis: a microscopic world of the cerebral galaxy which fascinates and worries us'.

The book has no editorial introduction and, indeed, little evidence of editorial intervention. Though some of the papers are worth reading, many are of only local interest and the translation from the Italian is often poor. The typewritten text is unattractive, misprints abound, and the binding is flimsy. My copy had several pages uncut, a section omitted, and another duplicated. Though its price is, by today's standards, quite low, I cannot wholeheartedly recommend this volume either to individuals or to libraries.

C. L. E. KATONA, *Senior Lecturer in Psychiatry, Middlesex Hospital, London*

The Substance Abuse Problems. Vol 2. New Issues for the 1980's. By SIDNEY COHEN. The Haworth Press: New York. 1985. Pp 323. \$34.95 h/b \$19.95 p/b.

The essays are in many cases revisions of contributions to a drug abuse and alcoholism newsletter and none the worse for that. However, they are of somewhat variable quality and Dr Cohen would not I am sure claim that this is intended as a textbook. As a reader, based on the very wide and considerable experience of a psychiatrist, well known both for his academic contributions and his concern for patients, it is well worth reading. Prominence is given to the drug, cocaine. This currently presents new problems on both sides of the atlantic, by reason of increasing availability, social acceptability and by the development of more potent forms, in particular the treatment of cocaine hydrochloride with alkali and then extracting it with solvents to produce a volatile preparation "cocaine free base" which can be consumed by heating and then inhaling, producing a very rapid effect. Dr Cohen draws attention to the

importance of the method of consumption in facilitating compulsive use which he promulgates as a general law whereby techniques or drugs that produce more intense and immediate effect tend to displace those that provide slower or more moderate effects. A useful chapter on management of compulsive cocaine use stresses the value of working with the family and assisting relatives in setting clear limits and neither directly nor indirectly financing a continued drug habit. He raises the possibility of a management technique not common in this country, described as contingency contracting whereby the patient/client writes a confession regarding their behaviour, with instructions that this should be mailed to some important other agency such as law enforcement or employer should they break their contract of continued treatment and abstinence. While this may seem excessively authoritarian it should be noted that the most successful alcohol problem treatment services are operated by employers, and particularly by the Armed Forces, where the consequence of relapse is clear, but the advantages of success are assured by the employer. Throughout the book Dr Cohen is eminently balanced in his comparison of illegal drugs with alcohol and tobacco, and there are extensive contributions on alcohol issues. A series of articles examine the evidence for controlled drinking and Dr Cohen provides his own personal criticism regarding the controversy surrounding the work of Sobels, whereby he stresses that the subsequent progress of the patients is the important issue rather than arguments regarding the detailed proprieties of the research. In general some of the most enjoyable aspects of the book are his pithy and refreshing personal comments, thus when considering the evidence for genetic factors in the aetiology of alcoholism he comments that this certainly has one advantage "one can always blame it on one's chromosomes" and "people with problems of loss of control must either: abstain completely; learn stratagems of maintaining control; or accept inevitable alcoholism". An essay of great value concerns training non-psychiatric workers in the importance and techniques of early identification of alcohol problems.

MARTIN MITCHESON, *Consultant Psychiatrist, County Hospital, Hereford*

The Psychoanalytic Study of the Child. Edited by ALBERT J. SOLNIT, RUTH S. EISSLER and PETER B. NEUBAUER. London: Yale University Press. 1985. Pp 568. £45.00.

This is the latest volume of an annual publication founded by Anna Freud, Heinz Hartmann and Ernst

Kris, originally centred around child psychoanalysis and the ways in which psychoanalytic concepts furthered the understanding of the development of children from the first weeks into adulthood. Previous volumes have contained many important contributions to psychoanalysis and analytical psychotherapy, as evidenced by the publication's survival for over forty years.

The volume is for those interested in analytically-oriented psychotherapy. In spite of its title the book is as relevant to those in adult psychotherapy as it is to those working with children. There are papers comparing adult analysis to child analysis, therapy of homosexual men, two papers on working with parent and child, two papers on the effects of siblings on development (one concerning the effect of having a handicapped sibling), a study of children raised by their fathers as primary care giver, the problems adoption poses to families (including suggestions about "when to tell"), a disturbing paper about psychic trauma (e.g. surviving the Tenerife Air Disaster) and its relation to ghosts and feeling haunted, and several other equally worthy of mention. All of these contain good clinical material, often spanning several years of therapy.

Three or four of the contributions are primarily theoretical. In line with the mostly USA based authorship, these papers are concerned with developing Freudian metapsychology and ego-psychology, a framework which is not so commonly adhered to in Britain.

The Applied section of the volume, in addition to some of the papers already referred to, contains five papers for the general reader interested in the understanding (and limitations) which psychoanalytic concepts can bring to art and the nature of creativity.

Overall, I found the volume enjoyable, readable and informative. It is probably not for the pre-membership psychiatrist. Those who do not normally work in an analytically-oriented way in psychotherapy should skip some of the papers, but not be put off the volume as a whole.

TOM MURPHY, *Consultant Psychotherapist, Royal Dundee Liff Hospital, Angus*

Testing Psychological Tests. By DOUGLAS SHELLEY and DAVID COHEN. Kent: Croom Helm. 1986. Pp. 203. £17.50 (hb), £8.95 (pb).

Although written in an easy to read, journalistic style the authors seek to address themselves to some serious questions concerning psychological testing. These include the possibility of generalising from laboratory to real-life settings, 'consistency' both of people and of tests; different types of validity, sampling and reliability. The book is laid

out clearly, each chapter containing a series of 'case histories' to illustrate points, quotations from papers and concluding remarks.

The authors' aim is not to write a 'which' guide to testing but to examine features and limitations of both well known and unusual psychological tests. A chapter on newspaper and magazine 'tests' is also included, showing how badly these are often constructed and how sweeping their claims usually are. The book is amusing and provocative, written with the general rather than specialist reader in mind. The chapters vary in interest and quality, the first and last being the best in my opinion.

The authors comment on the 'middle class' bias of many existing tests and the inappropriateness of U.S. student norms. They offer interesting and practical suggestions for designing new assessment tools and improving current ones to make them more relevant to present day needs. The book should succeed in making the reader question statements about psychological testing.

SUSANNE REIMAN, *Senior Clinical Psychologist,
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Culture and Depression: Studies in the Anthropology and Cross-Cultural Psychiatry of Affect and Disorder. Edited by ARTHUR KLEINMAN and BYRON GOOD. London: University of California Press. 1985. Pp. 535. £38.25

Depression as an illness and depression as a mood or symptom is explored in this volume by psychiatrists and anthropologists

Religious and social expectations in various cultures and their influence on depressive affect and disorder are investigated and categorised under historical, cognitive and epidemiological headings with a useful, final, integrating category.

Historically Christians seemed to have viewed depression as an example of 'bad' behaviour caused in the devout by the conflict between a desire for spiritual perfection and man's fleshly inclinations. Manual labour, social rejection or occasionally a 'gentle soothing approach' is recommended. Buddhism on the other hand takes the view that depressive mood characterised by hopelessness, distress and shame is a valued state since it leads to salvation achieved through a greater understanding of the nature of the world.

Attempts are made throughout the book to construct a universal paradigm for depressive emotion and disorder through the interaction of psychocultural and biosocial factors. This leads to the appreciation of the phenomenologies of depression as soul loss, existential despair, somatic distress and possession states.

Affect is also considered as rhetoric and as a stylistic characteristic of speech with metacommunicational markers which adds to the understanding of depression through the analysis of discourse. But the sociolinguistic paradigm poses questions for which disappointingly as yet no answers are available. Culture rather than biology encourages the reader to consider a final common ethno-behavioural pathway rather than the usually accepted biological one.

The editors are to be congratulated on bringing together many stimulating papers on the social and psychological causes of depression. This is an impressive work of scholarship and recommended for all those who seek to extend the boundaries of their understanding of this subject.

DENNIS FRIEDMAN, *Visiting Consultant,
The Priory Hospital, London*

Adolescent Relations with Mothers, Fathers and Friends. By JAMES YOUNISS and JACQUELINE SMOLLAR. London: The University of Chicago Press. 1985. Pp. 201. £21.25.

This American book describes the accumulated findings of eight different surveys carried out in recent years on the peer and family relationships of 1049 normal adolescents aged 14 to 19 years living in the United States.

It begins with a very useful and up-to-date introduction to the relevant literature, but major British work (eg Coleman) receives scant attention, and comparison with the findings of Offer's studies proves difficult. The importance of understanding adolescent relationships within the context of a family system is recognised in the writing up of the studies, but played no part in their original design nor in the interpretation of results. The book's primary contribution is therefore to the sociology of normal adolescence, and few of the findings have implications for clinical psychiatry.

It is not difficult to read and the structure of the book helps. Detailed descriptions of the research designs are mercifully largely confined to a single chapter, and the following eight chapters consist of five, describing results, interspersed by three summarising/discussion chapters. Taken with earlier work, the findings establish beyond reasonable doubt that for most adolescents the process of emancipation and establishing an adult identity is associated with a transformation of, rather than the detachment from, parental respect and relationship ties. Agreeing with Piaget's concept of autonomy, "autonomy . . . appears only with reciprocity", they found it to be one of the most crucial aspects of adolescent friendships, permitting joint exploration and understanding of reality.

Piaget and Sullivan are obviously profound influences, and it is possibly unfair to criticise the absence of any consideration of object-relations theory or modern ego psychology, or even the influence on adolescent family relationships of parental morbidity; however, the absence of any examination of the effects of unemployment, or membership of ethnic minorities, on the pattern of adolescent relationships seemed surprising omissions.

A book to be recommended for the library rather than for personal purchase.

R. M. WRATE, *Consultant in Adolescent Psychiatry,
Royal Edinburgh Hospital, Edinburgh*

Art Therapy for Groups: The Handbook of Themes, Games and Exercises. By MARIAN LIEBMAN. Beckenham Croom Helm. 1986. Pp. 226. £9.95.

This is an excellent, stimulating account by an experienced art therapist of how and with what result this approach may

be used in groups. The author has drawn on the experience of other therapists in the field, especially those in the Bristol area. Sensible advice is given in part one on how groups differ, may be composed and art integrated into sessions or workshops. The second part comprises brief descriptions of nearly 300 themes, games and exercises that the leader may suggest as ways of exploring group interaction, family relationships, and particularly well done, self-perceptions; the approaches may also be used to improve concentration, dexterity and memory. There are good suggestions for further reading.

This book is aimed at a wide range of practitioners whose caring profession may be as teachers, social workers, youth and community group workers and the new, separate profession within the N.H.S. of art therapists. Art is used as a means of personal expression to communicate feelings. The leader does not interpret the content, but helps the individual to let the picture 'speak back', and reveal aspects of its personal meaning. However, it is clear that personal issues of great complexity may surface and powerful group dynamics may be engaged. While the leader is strongly advised to seek supervision, especially if working in a clinical setting, consideration of the literature on individual and group dynamics only occupies 3 pages out of 225. This imbalance is worrying and reflects a deficiency in the formal training of art therapists. As the author states, the suggested activities are like tools which may be used constructively or destructively, clumsily or skillfully. Experience is one guide to their use. For the reviewer, supervision and knowledge of the established literature on human interaction are the others.

MARK AVELINE, *Consultant Psychotherapist,
Nottingham Psychotherapy Unit*

Negative Outcome in Psychotherapy and What to do About It. Edited by DANIEL T. MAYS and CYRIL M. FRANKS. New York: Springer Publishing, 1985. Pp. 365. \$34.95.

This book is a wide-ranging study of deterioration occurring in patients during the course of psychotherapy. It is specifically interested in negative outcome as opposed to treatment failure which of course would be 'zero outcome'. A major inference of proven negative outcome is that the treatment approach in question is a powerful one, which might have both positive and/or negative effects under certain conditions. A debate is still going on as to whether patients undergoing one of the psychotherapies not only improve more but also deteriorate more than those patients in a control group. The editors of this book contribute to this debate and come down on the side of no increased deterioration proven for treatment groups versus control groups in a large number of studies.

The book is tidily organised under the following headings, Introduction; Negative Outcome and Therapeutic Modality; Negative Outcome and Patient Population; Negative Outcome: Parameters and Special Considerations; Conclusions and Implications. The sum total of the book involves a wide study of therapist, patient and other variables which may lead to a deterioration effect. It ends with consideration being given to ways in which the risk of

patients becoming worse may be reduced. Overall, I found the first half of the book useful and illuminating with a marked deterioration occurring in the middle section. The editors themselves returned to save the day in the 'Conclusion' section. The research based chapters one and two, 'Negative Outcome: Historical Context and Definitional Issues' by Mays and Franks, and 'Negative Effects and Their Determinants' by Strupp and Hadley, I found to be closely argued, well researched, and thought-provoking.

The chapters by Dies and Teleska on Group Psychotherapy, Horne on Anorexia Nervosa, Vaughan and Beech on Obsessionals and Hollon and Emerson on Depressive Disorders, I found to be of poorer quality. These were more anecdotal and ruminative, providing far less research evidence and very often indicated more interest in the zero response (no change) than the negative response (deterioration). A chapter by Thoits on the 'Influence of Factors Outside Therapy', I found to be a useful reminder of the fact that one must always be on the alert for the interaction between our clients' inner and outer worlds.

There is also an anachronistic little article by Eysenck attempting to develop a theoretical framework for negative outcome using Pavlovian psychology. It seemed strangely out of place in these pages.

Overall, the good aspects of the book outweigh the bad and I would like to recommend it to those interested in the clinical and research aspects of those patients who deteriorate in the course of their psychotherapy.

J. P. ROBERTS, *Consultant Psychotherapist,
The London Hospital*

A Comprehensive Approach to the Treatment of Normal Weight Bulimia. Edited by WALTER H. KAYE and HARRY E. GWIRTSMAN. Washington: American Psychiatric Press, 1985. Pp. 131. \$12.00.

This is one of the first books devoted exclusively to the treatment of bulimia nervosa. It consists of seven review chapters each written by recognised authorities in the field. The book opens with two introductory chapters. The first outlines the clinical features of bulimia nervosa and the little that is known about its prevalence. This is followed by a description of some novel observation work on the over-eating of these patients. In the third chapter, which is concerned with the medical implications of bulimia nervosa, it is gratifying to see the eponym 'Russell's sign' being proposed for the characteristic abrasions and scars on the hands of those who regularly use their fingers to induce vomiting. There are few physical signs in psychiatry and it is entirely appropriate that this one is named after Professor Russell who has contributed so much to our understanding of the disorder. The remaining four chapters are devoted to treatment. The first by Yager and Edelman is an excellent description of the various approaches to the management of bulimia nervosa. It considers both assessment and treatment and examines the respective roles of psychological and pharmacological approaches. This chapter can be strongly recommended to anyone wanting an overview on the management of the disorder. It is followed by chapters on group therapy and in-patient treatment. The latter is especially

interesting since it provides a clear description of an in-patient management programme. Hospitalisation is rarely appropriate for patients with bulimia nervosa, but in some cases it is unavoidable. Almost invariably in-patient treatment proves difficult. This chapter describes how some of these difficulties may be overcome. The final chapter is concerned with the use of anti-depressant drugs. Unfortunately its authors are somewhat uncritical when describing the studies which support the use of these drugs and it is particularly regrettable that they omit to point out that there has been no systematic research into their long-term use.

This brief and relatively inexpensive book is a welcome addition to the literature on bulimia nervosa since it contains a number of useful reviews on several different forms of treatment. It would perhaps have been improved by the inclusion of two additional chapters: first, one on the diagnosis of bulimia nervosa since several different diagnostic schemes are currently in use; and second, a chapter on cognitive behavioural treatments since a mounting body of evidence suggests that they are one of the most effective ways of helping many of these patients.

CHRISTOPHER G. FAIRBURN, *Wellcome Trust Senior Lecturer, Department of Psychiatry, University of Oxford*

Psychological Disorders in Obstetrics and Gynaecology. Edited by R. G. PRIEST. Sevenoaks: The Butterworth Group. 1985. Pp. 236. £32.00.

A review of the many psychological problems that are found within obstetrics and gynaecology has been needed for some time. This book covers this subject very fully indeed and Professor Priest must be congratulated for assembling a list of authors whose contributions are comprehensive, detailed and immensely readable. Written entirely by psychiatrists this publication covers not only the psychological problems caused by pregnancy and obstetric and gynaecological problems in general, but it also discusses the psychological disorders that may present with gynaecological symptoms. The book begins with an excellent survey of sexual dysfunction and the psychological causes of infertility, and is followed by a clear and factual discussion of the emotional and psychological problems that may result from gynaecological surgery, in particular hysterectomy. One is pleased to see, at long last, an objective and well balanced review of these emotive subjects. The difficult problems of pelvic pain and premenstrual syndrome are discussed at some length but the largest chapter in this book is concerned with the psychological disorders that may be associated with pregnancy. This very comprehensive contribution is clear, well referenced and immensely informative. Many of the sections in this book illustrate their text with case reports and such reports were particularly helpful in relation to pregnancy. Further equally excellent sections in this book cover the puerperium and termination of pregnancy. The last chapter concerns itself with the menopause, where the psychiatric problems, said to be associated with the menopause, are objectively evaluated.

This splendid contribution to the literature must surely be of interest to all those psychiatrists involved with obstetric and gynaecological patients. It certainly should be mandatory reading for all obstetricians and gynaecologists, and I hope that this book will come to hold an important and respected position on the shelves of all medical libraries.

ANNE M. JEQUIER, *Senior Lecturer, Queen's Medical Centre, Nottingham*

Susto, A Folk Illness. By J. ARTHUR, CARL W. O'NEIL and ROLANDO COLLANDO-ARDON. Berkeley: University of California Press. Pp. 186. \$22.95.

This book is an interesting attempt by two anthropologists and a physician to carry out a study of the folk-illness 'Susto' in three adjacent communities in Mexico.

The authors in a useful introductory chapter, make the point clearly that this folk illness should not be encapsulated within pre-arranged Western diagnostic categories. They have started, therefore, with the local belief that Susto is caused by separation of soul and body which is precipitated by the intervention of a supernatural force. They have then compared patients who complained of having Susto with a local matched control group. Furthermore they have acknowledged that a proportion of patients with Susto might be suffering from a physical illness or indeed, have a clear cut psychiatric disorder. In a novel way, therefore, the authors have juxtaposed anthropological, physical and psychiatric clinical approaches and so highlighted the contrast between the medical and the ethnographic paradigms.

Inevitably, difficulties with this combined approach were encountered. There was a problem translating the Langner 22 item scale into the local language and the limitations of this scale became readily apparent. Thus no differences were found in scores on the Langner scale between the Susto patient and control although, using crude clinical categories of psychiatric disorder, Susto patients were more likely to be emotionally disturbed.

It is a pity that the authors did not include a research psychiatrist on their team.

Nevertheless, the book is worth reading by anyone who is about to embark on a study which will involve close collaboration between social anthropologists, psychiatrists and physicians. The authors have courageously attempted to combine social anthropological research methodology with behavioural science measures. In this way they have pioneered a collaboration, from which others will learn.

This collaboration is indeed vitally important, but, as the authors have found, is very difficult to carry out. The account of the attempt made to overcome the difficulties and the full details of the questionnaires used that are included in the appendix makes the book worth buying, or certainly borrowing from a local library.

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Autumn Quarterly Meeting, 1985

Friends House, Euston Road, London NW1 (14-15 November 1985)

Winter Quarterly Meeting, 1986

*The Royal Society, 6 Carlton House Terrace, London SW1
(21-22 January 1986)*

Spring Quarterly Meeting, 1986

*University of Manchester Institute of Science and Technology,
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Abstracts of papers presented at the Quarterly and Annual Meetings of the Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PG

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Autumn Quarterly Meeting, 1985

Friends House, Euston Road, London NW1 (14-15 November 1985)

Classification of affective disorders

The influence of periodicity on the classification of affective disorders

Dr C. Thompson, Charing Cross Hospital, London

Periodicity, the regularity of occurrence of affective episodes, has been seen as an important property of affective disorders since they were first distinguished from schizophrenia by Kraepelin (1921). The value of periodicity in the classification of affective disorders is discussed, and recent attempts to use periodicity in a different way is described.

The Newcastle scale and measurement in depression

Dr M. W. P. Carney, Northwick Park Hospital, Middlesex

Fresh evidence in the long-running debate between the monist and dualist views of the classification of depression is presented. The Newcastle Scale (for the differentiation of endogenous from neurotic depression) scores of a group of 64 and sub-sample of 52 severely depressed in-patients were found to be not normally distributed, the scores of the sub-sample of 52 assuming a typical bimodal distribution. Though the shape of the distribution curve is not accepted as conclusive, evidence for discontinuity in these distributions was adduced from the contrast in outcome between the endogenous (with Newcastle scores greater than 5) and neurotic (with Newcastle scores of 5 or less) patients thus defined, the endogenous patients faring consistently better than the neurotic group. On the other hand, the unipolar/bipolar system of classification failed to predict a differential outcome in a sub-group of patients, unless the unipolar was divided by Newcastle score into endogenous unipolar and neurotic unipolar. The DSMIII criteria for major depression and melancholia failed to identify sub-groups of differing prognosis. It is concluded that the distinction between endogenous and neurotic depressions as judged by Newcastle score has validity in terms of clinical outcome and is not to be confused with severity of depression.

The WHO study on the dexamethasone suppression test

Dr A. Coppen, MRC Neuropsychiatry Research Laboratory, Surrey

A large study was carried out by the World Health Organization in order to examine the incidence of an abnormal response to the dexamethasone suppression test among depressive patients. Thirteen research centres, representing 12 different countries, took part in the study. Plasma samples from 543 patients and 246 healthy controls were assayed at the MRC Neuropsychiatry Laboratory in Epsom. The results showed variation between centres, but in most of them the percentage of depressive patients with an abnormal DST response was much higher than in the controls.

French diagnostic practices in the field of affective disorders

Professor C. Pull, Centre Hospitalier de Luxembourg

In the French classification of mental diseases, elaborated by the Institut National de la Santé et de la Recherche

Médicale (INSERM), depressive disorder is listed in the three following categories: manic and depressive psychoses (INSERM O1) with basically only two sub-categories, i.e. the melancholic episodes of manic-depressive psychosis and involuntional melancholia; non-psychotic depressions (INSERM 13) subdivided in neurotic and reactive depressions; and chronic schizophrenia (INSERM O2) which presents a sub-category termed chronic schizophrenia with depression, or atypical depression.

The INSERM classification is a simple nomenclature which provides neither glossary like ICD-9 nor diagnostic criteria like DSM III. As a consequence, it would make little sense to proceed to direct comparisons between the French and any other classification system. In the present report, comparisons are based on a poly-diagnostic approach, in which clinical diagnosis is documented by means of a standardised list (LICET-D 100) which integrates the diagnostic criteria proposed in a number of recent classification systems.

In 1984, a survey was carried out in a sample group representative of French psychiatrists. The group consisted of 50 qualified psychiatrists who had previously collaborated in similar studies. The participants were invited to provide several cases of depressive patients diagnosed according to the INSERM classification. No criteria were imposed to make a diagnosis. The only requests were that the clinicians should be reasonably positive about their diagnoses and that they should document their diagnoses using a set of internationally understandable criteria.

This report presents the first results of this investigation.

Mental handicap

A study of the socio demographic, familial and clinical factors associated with the admission of the mentally handicapped to hospital

Dr J. M. Rao, University of Wales College of Medicine, Cardiff

The case records of 293 mentally handicapped persons admitted to Ely Hospital over a 10 year period were examined and four categories of information were recorded comprising demographic data, sources of referral, reasons for referral and family factors. A preliminary analysis indicated that the majority of those admitted came from socio-economically disadvantaged circumstances. More than half the total sample had severe mental handicap and a large proportion had physical handicap as well. More than half had behavioural problems and a quarter had medical problems leading to admissions. A large proportion of families suffered stress from a concatenation of causes. A considerable number of families lacked intellectual, emotional or material resources. It was thus evident that the need for admission to hospital in a majority of cases arose as a result of not only severe handicap and behavioural, medical problems but also due to adverse influences in the family environment.

Lessons to be learned from closing a hospital for the mentally handicapped*Dr Lorna Wing, Institute of Psychiatry, London*

The effects on the residents of the run down of Darenth Park adult mental handicap hospital are being evaluated. The behaviour, skills and life styles of those who move, and of individually matched controls temporarily remaining in the hospital, are being recorded shortly before moving and one year later. Data are collected by interviewing residents, staff and relatives and observation and psychological testing of residents. Since 1980, before the opening of planned, locally based DHA facilities, over 250 have been placed in various NHS, social service, voluntary and private establishments throughout S.E. England. These *ad hoc* placements have led to improvements in life style for some, deterioration for others and no change for the rest.

The lessons learned from such placements include the following. It is difficult to move out of hospital those residents with severe impairments of social interaction and communication, poor skills and difficult behaviour. Future plans for hospital closures should ensure that, from the start, movers should include difficult as well as amenable people, to avoid creating a residue of those with severe problems. A slow run down causes great anxiety to many who understand what is happening, especially older, long-stay residents. Careful preparation and, where possible, explanation are necessary to help residents settle in new placements. Sudden removals to new units cause much distress. It is easy for those organising new services to be unaware of long and deep friendships between residents, and separation of such friends of the same or opposite sex can lead to much unhappiness and depression. Those living outside hospital have fewer amenities provided free of charge, which leads to personal money problems. Placement in old people's homes of mentally handicapped people who are elderly but not demented often proves to be inappropriate.

For all levels of handicaps, small living units with adequate staffing levels, staff autonomy and a flexible organisation are necessary for, but do not, of themselves, guarantee a lifestyle with more opportunities for occupation, education and leisure. However such units do not appear to lead to increase in skills nor much change in behaviour. The effects on people with different types of handicaps of living in ordinary houses in groups of two, three or four people have still to be evaluated.

Developments in parasuicide**The problems of assessing the epidemiology of suicide and parasuicide***Dr R. D. T. Farmer, Westminster Medical School, London*

Epidemiology is concerned with investigating the causes and natural history of diseases by studying their behaviour in populations. This involves exploring the relationship between the disease under investigation and temporal, social and personal variables. The fundamental problem encountered with the study of suicide and parasuicide is that neither comply with generally accepted concepts of disease. Suicide is the termination of a person's life as a result of actions initiated by the individual with the deliberate and conscious intent of achieving death. Parasuicide is

a non-fatal self-injury usually initiated without the intent of achieving death. Both definitions depend on making assumptions about the motive and outcome of an act.

In order to establish a suicide it has first to be recognised as due to other than natural causes. In England and Wales investigation of unnatural deaths is the responsibility of the Coroner who is bound by rules of evidence; in Scotland the Procurator Fiscal has this responsibility; he has a different set of rules. In much of Europe medical certification of cause is separated from the processes of criminal investigation. Consequently, international comparative studies present major problems.

There is no widely recognised prodromal symptom complex that can be said to be the 'early stages' of suicide. The suicide only becomes apparent after death has occurred; the parasuicide becomes apparent after the event (provided that the injury justified hospital attendance) and the patient is, by definition, alive, because the dead cannot be interviewed. Clearly there must be a relationship between suicide and parasuicide but the exploration of this relationship is hampered by the inescapable fact that the two phenomena cannot be investigated using the same methodology. From the point of view of the clinician this fact has only marginal relevance but from the standpoint of an epidemiologist it is of critical importance. Ways of overcoming some of these problems are discussed.

Long-term outcome of attempted suicide*D. K. Hawton, Warneford Hospital, Oxford*

People who make suicide attempts are a very heterogeneous population—the common factor being their having all engaged in this form of behaviour. Except in comparative research (as in treatment studies), the most meaningful way of examining outcome is in terms of subsequent suicidal behaviour. This paper is largely concerned with repetition of attempts and with eventual completed suicide.

Between 12% and 25% of attempters make repeat attempts within a year of a previous attempt, repeats most commonly occurring during the first three months. A particularly problematical group are chronic repeaters, who have a high risk of eventual suicide. Although risk factors for repetition have been identified, special treatment services have had no effect on repetition rates.

A recent long-term follow-up study of a large group of suicide attempters has demonstrated that death rates are exceptionally high, because of both eventual suicide and death from other causes. During the first year the suicide rate was one hundred times that of the general population, but remained high throughout the nine year follow-up. Suicides were distinguished from survivors in terms of being older, poor physical health, psychiatric disorder, especially chronic psychiatric illness, long-term use of hypnotics, and less often having made their attempts following a major row or the break-up of a relationship with a partner. Death from circulatory, respiratory and endocrine disorders were also far higher than expected. This may be explained partly in terms of physical illness and contributing to suicidal behaviour and partly in terms of the stressful life-styles of those individuals.

Outcome of attempted suicide*Dr Jouko Lonnqvist, Associate Professor of Psychiatry, Helsinki University, Tukholmankatu 8 C, 00290 Helsinki, Finland*

Preliminary results of a study which comprises 3,265 consecutive suicide attempts at the emergency departments of Helsinki University Central Hospital in 1973-79, and for whom a psychiatric consultation was arranged, are given. In addition to demographic data, lethality and intent to die and the intensity of the further psychiatric treatment was assessed. By the end of the follow-up period (1973-80) 305 patients had died, 217 of them male and 88 female. In 49.8% the cause of death was suicide. The suicide rate among males (7.8%) was much higher than among females (2.3%). The risk of suicide by age among males is highest (8.7%) for those under 20. Lethality and intent clearly distinguish between attempts in terms of the risk of suicide. The amount of suicides was 2.9% for mild, 4.7% for moderate and 8.4% for severe lethality (somatic severity) and 3.0% for mild, 4.3% for moderate and 7.8% for severe intent to die. In cases of severe intent the risk of suicide during the first year of follow-up is much higher (5.0%) than for moderate (2.1%) or mild (1.2%) intent.

The intensity of further psychiatric treatment correlates with the suicide rate during the follow-up period. Proportion of suicides was 7.7% for compulsory hospital treatment and 6.6% for voluntary hospital treatment, from 3.6% to 4.6% in different outpatient facilities and 2.0% for those who had not received any psychiatric treatment.

Recent clinical and epidemiological trends in parasuicide: A tale of two cities

Drs Stephen Platt, MRC Unit for Epidemiological Studies in Psychiatry, Edinburgh and Keith Hawton, Warneford Hospital, Oxford

This paper presents a comparative analysis of trends in parasuicide among hospital treated patients in Edinburgh and Oxford, 1976-84. Over the period the overall parasuicide rate declined in both cities, but more markedly among women than men. While the female rate was consistently higher than the male rate, there was some suggestion that the female:male ratio was becoming more equal in Edinburgh. Among men, the rate in Edinburgh was significantly higher than the rate in Oxford, while the rates among women in both cities were similar over the years and significantly correlated. An analysis of rates by age group in 1983-84 shows the familiar inverse relationship with rates tending to decline with increasing age. However, this pattern was more marked among females in both cities (rates peaking in the 15-19 age group), while the highest rate among males was in the 20-24 age group in Edinburgh and in the 25-34 age group in Oxford. Over the study period there was a marked fall in the rate in Oxford among males aged 45+ years and women aged 55+ years. Among men in both cities rates were lowest among the married and highest among the divorced, while the pattern for women was somewhat different: lowest among the widowed and highest among the divorced (Edinburgh) or among the single (Oxford). There was a strong inverse relationship with social class, and a marked increase in parasuicide among the unemployed compared to the employed, for women as well as for men.

Major clinical features included: a higher short-term repetition rate for Edinburgh men compared to Oxford men; a dramatic decline over time in the use of barbiturates and a marked increase in the use of non-opiate analgesics, particularly paracetamol; a greater proportion of the Oxford sample reporting previous psychiatric treatment

and alcohol problems, but use of alcohol at the time of parasuicide being more prevalent in Edinburgh; and a significant variation in the pattern of aftercare between Edinburgh and Oxford, with more use of outpatient facilities in Oxford and greater reliance on the general practitioner in Edinburgh.

Mental illness and the temporal lobe

The neuropathology of temporal lobectomy

Dr J. C. Bruton, Department of Neuropathology, Runwell Hospital, Essex

The study presented is a clinico-pathological survey of 249 temporal lobectomy cases operated upon by Mr Murray Falconer, during the 25 years beginning 1 January 1950.

The first aim was to identify the appearances, nature and behaviour of the various abnormalities found in the resected lobes, and in order to do this it was considered beneficial to modify to some extent the current system of neuropathological nomenclature of the temporal lobe lesions.

The second object was to identify, if possible, those patients most likely to benefit from temporal lobe surgery, using follow-up information which had been collected in some patients for almost 25 years.

The results of the survey have been encouraging in that the revised system of classification appears not only to highlight certain differences between lesions, showing that some behave as neoplasms, whilst others are maldevelopmental, but it also enables the formation of a 'league table of benefit from surgery' which may be of use to clinicians who deal with these cases.

The results confirm that patients who have the commonest lesion (Ammon's horn sclerosis) are likely to benefit greatly from surgery. Other less common lesions, such as the mixed glial and the ganglioglioma malformations, also do well. However, three groups were significantly worse after operation; these were the patients with post-traumatic damage, those with indefinite pathology and those with no demonstrable pathology at all.

During the survey, details of psychiatric illness and of post-operative social adjustment to life were recorded and certain significant changes were seen. The presentation concentrates, to a certain extent, on these psychiatric features.

Limbic pathology in schizophrenia

Dr B. Bogerts, Department of Psychiatry, University of Dusseldorf, West Germany

In order to determine whether ventricular enlargement in schizophrenics is due to a general brain atrophy or to a more regionally confined reduction of brain tissue, CT scans and post mortem brains of schizophrenics were evaluated morphometrically.

(a) *CT scans.* The size of all cortical sulci, the Sylvian fissure, lateral, third and fourth ventricles of 54 schizophrenics (ICD 9 295.0-6) and 54 controls matched for age and sex were determined by blind planimetry. The left Sylvian fissure was most enlarged (by 110%, $P < 0.001$) followed by the right Sylvian fissure (80%, $P < 0.01$), the frontal sulci (35%, $P < 0.05$), third ventricle (25%, $P < 0.05$), and VBR (18%, $P < 0.05$), whereas the parieto-occipital sulci and the fourth ventricle were not significantly altered.

(b) *Post mortem brains*: Volumes of several parts of the basal ganglia and limbic system of 14 schizophrenics and 10 controls collected before the introduction of neuroleptics were determined by planimetry of myelin-stained serial sections. The hippocampus (-20% , $P < 0.01$), amygdala (-15% , $P < 0.05$), parahippocampal gyrus (-30% , $P < 0.05$), pallidum internum (-15% , $P < 0.01$), and the diencephalic periventricular grey matter (-19% , $P < 0.01$) were smaller in the schizophrenic brains, whereas all parts of the striatum, the pallidum externum, and all large thalamic nuclei did not exhibit significant alterations.

There was no correlation between any of the data and the duration of the disease, thus, brain atrophy seems to exist before the onset of the disease. CT scans and post mortem brain measurements indicate that the anterior parts of the temporal lobe and the limbic structures within the temporal lobe are most affected in the brains of schizophrenic patients. Hippocampus and amygdala are anatomically and functionally interposed between neocortex and hypothalamus. Therefore, it is suggested that at least some clinical symptoms of schizophrenia are psychopathological equivalents of a functional dissociation between neocortical (cognitive) and hypothalamic (emotional) activities.

Neurofibrillary tangles are located in somatostatin neurones in Alzheimer's disease

Dr G. W. Roberts, Division of Psychiatry, Clinical Research Centre, Middlesex

Alzheimer's dementia is a chronic, progressive neuropsychiatric condition of unknown aetiology. It is characterised clinically by global intellectual impairment and neuropathologically by the presence of numerous argyrophilic plaques and tangles. Neurochemical investigations on post-mortem tissue have established losses of cortical cholinergic afferents and cortical somatostatin containing neurones. Other aminergic cortical projections systems are also affected although these are thought to be secondary changes. The transmitters of other intrinsic cortical systems are largely unaffected.

Immunocytochemical techniques and post-mortem tissue were used to examine the relationship of somatostatin immunoreactivity neuropathological markers. Somatostatin containing neurones exhibited morphological changes consistent with neuronal degeneration and marked decreases in somatostatin immunoreactivity were observed. Classical and immunological methods for demonstrating plaques and tangles showed an association of somatostatin with both. Further studies indicated that other neurotransmitters (VIP, CCK, Acetylcholine) were associated with plaque formation but not tangle formation. The findings indicate that somatostatin-positive neurones are degenerate and associated with tangle formation. Thus, destruction of somatostatin neurones is an early and perhaps critical event in the onset of Alzheimer's disease.

Anorexia nervosa

Gastric functions and anorexia nervosa

Professor P. R. McHugh, The Johns Hopkins Medical Institutions, Baltimore, USA

A steady growth in the understanding of gastric emptying has demonstrated that it is a biphasic phenomenon under strict control from the receiving intestine. Initially, the gas-

tric contents spill into the intestine which then regulates the subsequent flow of intragastric material according to its caloric content. Patients with anorexia nervosa and bulimia have been studied in both the starved and weight recovered state and several distinctions from normal are found amongst them that may have significance to their clinical state and management.

Schizophrenia—the nature of the disease process

The retrovirus/transposon hypothesis of psychosis

Dr T. J. Crow, Clinical Research Centre, Northwick Park Hospital, Middlesex

Twin and adoption studies suggest a genetic component in schizophrenia, but the extent of discordance in MZ twins is compatible with a role for an environmental factor. This could be a virus, and the findings in some family studies do not exclude horizontal transmission.¹

In pairs of siblings age of onset is significantly ($X^2 = 42.45$, $P < 0.0005$) more often younger in the younger sibling than would be expected from a 50:50 expectation. However this is due to ascertainment bias rather than contagion because the age shift is not seen in elder sibling ill first pairs. This observation excludes horizontal transmission and also environmental factors shared by the siblings and occurring at a defined point in adult life.

The hypothesis is proposed that psychosis is due to a pathogen integrated in the human genome which is acquired either from a parent or by an integration/transposition event occurring early in ontogeny. It is suggested that the pathogen integrates at the site of the gene (a 'proto-oncogene') which determines cerebral dominance. This could explain selectivity for the left hemisphere.

¹Crow, T. J. (1983) *Lancet*, i, 173–175

Aspects of the epidemiology of schizophrenia

Dr E. H. Hare, The Maudsley Hospital, London

It is still a matter of opinion whether the diagnosis of schizophrenia is sufficiently precise for its epidemiological study to be worthwhile. The speaker gives his reasons for thinking the precision is sufficient, and that epidemiological studies of schizophrenia have suggested causal hypotheses which are worth further study.

The most firmly established finding is the seasonal variation of admission rates for schizophrenia to psychiatric hospitals. Seasonal variation in the birth rate of schizophrenia patients is also firmly established and, if it is of aetiological significance, puts a severe constraint on the nature of the causal agents. Variations in incidence rate with place and time are less well established but raise the possibility that schizophrenia may be a relatively new disease, rare before the 19th century.

Is tardive dyskinesia a marker of 'organicity' in schizophrenia?

Drs T. R. E. Barnes and P. F. Liddle, Charing Cross and Westminster Medical School, London

Investigation of the characteristics of schizophrenic patients with tardive dyskinesia may identify risk factors for the development of tardive dyskinesia, and further the understanding of the nature and cause of certain schizophrenic symptoms in those patients who exhibit the

condition. The known risk factors for tardive dyskinesia are the administration of antipsychotic drugs, and increasing age, which probably reflects degenerative brain changes. The appearance of disorders of involuntary movement in schizophrenia, independent of drug treatment, raises the possibility that the schizophrenic disease process may also be a factor. It has been postulated that both the movement disorder and some of the disturbances of higher mental function seen in schizophrenia may be related to subcortical damage, such as lesions of the basal ganglia¹. This postulate is consistent with recent reports that schizophrenic patients with tardive dyskinesia show more signs of organic brain disorder. Compared with their non-dyskinetic fellows, such patients show more evidence of cognitive impairment as seen in early dementia, more 'soft' neurological signs, larger cerebral ventricles and an increased mortality. They are also more likely to develop other movement disorders, such as akathisia and tardive dystonia, and show a higher prevalence of 'negative' or 'type II' schizophrenic symptoms². One recent study also suggests that patients with 'non-familial' schizophrenia, where organic antecedents may have a larger role than in 'familial' schizophrenia, might be more likely to develop tardive dyskinesia³.

¹JESTE, D. V., KARSON, C. N. & WYATT, R. J. In *Neuropsychiatric Movement Disorders* (eds D. V. Jeste & R. J. Wyatt). Washington: American Psychiatric Press, pp 119-150.

²BARNES, T. R. E., LIDDLE, P. F. Tardive dyskinesia, implications for schizophrenia? *Journal of Royal Society of Medicine*, (in press).

³BARTELS, M., MANN, K. & FRIEDRICH, W. (1985) Tardive dyskinesia: marked predominance of nongenetic schizophrenia. *Biological Psychiatry*, 20, 101-103.

Dyskinesia in schizophrenia: relative contribution of neuroleptic treatment and the disease process

Drs J. L. Waddington, Hanafy A. Youssef, David J. King, Stephen J. Cooper, Department of Clinical Pharmacology, Royal College of Surgeons in Ireland, Dublin 2, St Davnet's Hospital, Monaghan, Ireland and Holywell Hospital, Antrim, N. Ireland

In agreement with previous studies, we have found schizophrenic patients who develop late-onset involuntary movements (IM, 'tardive' dyskinesia) to have received neither longer nor more vigorous treatment with neuroleptics in comparison with patients without orofacial IM. Rather we find them to be more likely to show negative symptoms and intellectual impairment.

In further studies, schizophrenic patients were assessed for IM using the AIMS and evaluated neuropsychologically for intellectual function; one patient group had been previously investigated by CT scan and ventricle-brain ratios (VBR) determined.

In an older population (age 30-90 y, n=68), both intellectual impairment and the prevalence of muteness increased significantly with age in patients with IM, while no relationship was present in patients without IM. In a younger population (age 25-67 y, n=16) VBR's were significantly larger in patients with IM; intellectual impairment increased significantly with age in patients with, but not in those without IM. VBR increased with both age and extent of intellectual impairment, but again only in patients with IM, similar relationships were found substituting duration of illness for age in these analyses.

Preliminary results of a stepwise logistic regression analysis, using data from 88 patients, identified the presence of

marked intellectual impairment of muteness as the factor best discriminating patients with IM from those without; their presence was also associated with the severity of such movements.

These studies point towards an association between the presence of involuntary movement and a distinct pattern of progressive clinical deterioration and structural brain changes in schizophrenia. This work was supported by the Medical Research Council of Ireland and the Royal College of Surgeons in Ireland.

Neurochemical laterality in schizophrenia

Dr G. P. Reynolds, Department of Pathology, University of Nottingham Medical School, Queen's Medical Centre, Nottingham

There is a substantial and increasing body of evidence indicating a hemispheric abnormality in schizophrenia. This has developed from the hypothesis of Flor-Henry who, on the basis of the occurrence of schizophreniform psychosis in left-sided temporal lobe epilepsy, proposed schizophrenia to be a dysfunction of the left temporal lobe. Studies involving evoked potentials, skin conductance, brain imaging, neuropsychology and, more recently, post-mortem anatomical investigations have all lent support to a lateral asymmetry of the brain in schizophrenia.

Despite the strong pharmacological indications of a dysfunction in dopamine neurotransmission, only one report has indicated a lateral asymmetry of dopamine function in schizophrenia. Dopamine, measured in post-mortem brain tissue from schizophrenic patients, was found to be significantly elevated in the left amygdala but not abnormally increased on the right. The neurochemical and regional specificity of this finding has been investigated further. No increase in a range of neurochemical markers for noradrenaline, acetylcholine, GABA, and serotonin neurotransmitter systems was observed in the amygdala. Dopamine showed no asymmetry in either the caudate or accumbens nuclei, although a lateral asymmetry in dopamine concentration paralleling that in the amygdala has also been observed in the temporal cortex.

While influences such as that of neuroleptic medication cannot be ruled out, these results certainly provide a neurochemical indication of a left temporal lobe abnormality in schizophrenia.

Short papers

A controlled trial of carbamazepine versus lithium in mania

Drs N. C. Moore, B. Lerer, E. Meyendorff, S. R. Cho and S. Gershon, Lafayette Clinic, Detroit, USA

Controlled comparisons with chlorpromazine and placebo have reported antimanic efficacy for the anticonvulsant carbamazepine (CBZ). Lithium (Li) has been conclusively demonstrated to alleviate manic symptoms and may thus serve as an ideal reference drug for evaluating antimanic efficacy. In the context of a double-blind trial, 34 consenting manic patients fulfilling DSM III criteria, were randomly assigned to treatment with CBZ or Li for four weeks. Treatment commenced with CBZ 600 mg/day or Li 900 mg/day and was increased to achieve serum levels of 9-12 mg/ml for CBZ and 1.0-1.5 mg/ml for Li. Serum levels were within the projected therapeutic range. Rating scales administered at baseline and weekly thereafter were: Brief

Psychiatric Rating Scale, Beigel-Murphy Scale and Clinical Global Inventory (CGI). Twenty-eight patients completed the full protocol, 14 on CBZ and 14 on Li. Analysis of the data by ANOVA with repeated measures showed a significant antimanic effect for both treatments on all three rating scales. The overall therapeutic response of the Li-treated patients was superior to that of CBZ but this difference did not reach statistical significance. There was, however, a significantly greater preponderance of good treatment responders in the Li group, whereas the overall CBZ effect depended on two excellent responders (CGI change score of 5 points) and two good responders (CGI change 2-3 points) in that group. These findings suggest that CBZ has antimanic potential in specific bipolar patients whose clinical characteristics remain to be clearly elucidated. Detailed results are presented.

Platelet 5-HT receptor binding in depression

Drs E. Charig, P. J. Cowen and Mrs S. Fraser, University Department of Psychiatry and MRC Unit of Clinical Pharmacology, Littlemore Hospital, Oxford

Changes in 5-hydroxytryptamine (5-HT) receptor sensitivity have been implicated in both depressive illness and therapeutic effects of tricyclic antidepressant treatment (TCA). The platelet possesses specific binding sites for lysergic acid diethylamide (LSD) which probably represent functionally active 5-HT₂ receptors.¹ We report the effects of depressive illness and TCA treatment on platelet LSD binding.

Venous blood samples were taken from depressed patients and healthy control subjects between 09.00 and 10.00 hours. The depressed patients met DSM-III criteria for major depression and had been free of all medication except benzodiazepines for at least three weeks. The binding of iodinated LSD to intact platelets was measured using a modification of a previously described method.¹ Platelet LSD binding did not differ between depressed subjects and controls. However, following four weeks' TCA treatment the number of platelet LSD binding sites was significantly increased. This study of platelet 5-HT receptor binding, therefore, does not support the hypothesis that 5-HT receptors are altered in depressive illness; however, it appears that TCA treatment may increase 5-HT₂ receptor density. This latter finding is at variance with what would be expected from animal studies but consistent with our previous report of increased LSD binding on the platelets of normal subjects treated with desmethylimipramine.²

¹GEANEY et al (1984) *European Journal of Pharmacology*, 97, 87-93

²COWEN et al (1985) *Archives of General Psychiatry*. (In press).

Normal and abnormal variation in platelet 5HT transport: A comparison of control and depressed populations

Drs D. Healey, A. O'Halloran, P. A. Carney and B. E. Leonard, University College, Galway, Ireland

The original amine hypotheses of depression suggest deficiencies of central amines. Such hypotheses are inherently implausible. They were not supported by reliable clinical observations when first formulated and have since generated no reproducible clinical or biochemical evidence in their favour—with the single exception of platelet 5HT uptake which has been repeatedly reported to be lowered in psychotic depression. Such reports are of interest as many antidepressants purportedly inhibit 5HT uptake. There are

conflicting reports as to the effect of such agents on the lowered uptake of 5HT in depression.

We have investigated these issues further in a group of 28 psychotically depressed patients. Some of the group were treated with uptake inhibitors and the rest by other means. Uptake was measured at several points during the day and during the illness.

Our findings confirm reports of an abnormality in platelet 5HT transport but do not support the notion of amine deficiency or indeed of a primary aminergic abnormality. Other possible explanations are offered.

The predominant hypotheses concerning the psychological abnormalities in depression propose alterations in cerebral biogenic amine concentrations or functioning. Platelet 5-HT reuptake has been shown to be reduced in depression and is one of the few pieces of evidence from depressed persons, which has been consistently reproduced, that can be cited in favour of these hypotheses. In this study, platelet 5-HT reuptake was measured at three time points during the morning in depressed persons, before treatment, acutely after its institution and following chronic administration, and compared to controls. Control rates varied in a manner consistent with the presence of a circadian rhythm in uptake. Depressed persons showed no variation at these times of measurement. Clinical recovery was accompanied by a restoration of variation. This normalisation was not found after acute administration of antidepressants or ECT and was independent of the effect of treatment on the affinity of the transport system. These results are consistent with the notion that the pathophysiology of depression involves a disruption of circadian rhythms, yielding secondary abnormalities of biogenic amines rather than a primary monoaminergic disturbance. Among the depressed persons studied, those who were deluded displayed a qualitatively similar profile of uptake rates to the other depressed subjects but had quantitatively different rates at each time point.

This finding may be pertinent to the clinical experience that when depression is accompanied by delusions its response to treatment differs from that of depressions without delusions.

Is lithium justified after one manic episode?

Dr A. J. Mander, University of Edinburgh

A retrospective survey of first admissions with a diagnosis of mania revealed that almost half the patients concerned were discharged on prophylactic lithium. The decision to use lithium was influenced by a number of factors including severity and duration of illness, sex and social class. A follow-up study revealed that none of these factors, nor any others examined predicted outcome.

Although lithium was shown to be an effective prophylactic, the outcome of those treated with it was not significantly different from the untreated group. The reasons for this are discussed and some tentative evidence of a lithium withdrawal state presented.

Which depressives have blunted growth hormone responses to clonidine

Drs C. L. E. Katona, A. S. Hale, A. E. Theodorus, S. L. Davies, C. Tunnicliffe, R. W. Horton, E. S. Paykel and J. S. Kelly, St George's Hospital Medical School, London

Growth hormone (GH) response to the selective α_2 adrenoceptor agonist clonidine has been reported in several studies

to be blunted in depression.^{1,2} Our own preliminary report³ suggested that although there was no obvious blunting of GH responses in depressives overall, a subgroup showing dexamethasone suppression test (DST) non-suppression had significantly blunted responses. We report our investigation of the inter-relationships between the GH response following 1.3 ug/kg clonidine, the 1 mg, single sample DST, and a number of clinical variables in 41 depressed subjects with RDC major depressive disorder and a minimum Hamilton Depression Rating (HDRS) of 17. Our results are as follows:

Table I
Correlations with GH response (area under curve)

	Spearman's rho	P
Post Dexamethasone Cortisol (ug/100 ml)	-0.52	<0.01
Age (years)	-0.27	<0.1
Hamilton Depression Score	-0.02	NS
Drug-free Interval (days)	-0.15	NS
Feinberg-Carroll Score	0.07	NS
Newcastle Score	-0.10	NS

There is a highly significant correlation between post dexamethasone cortisol (PDC) and GH response; DST non-suppressors (n=17) as a group show significantly smaller GH response than suppressors (n=16; U=61.5; P=0.01) suggesting a negative relationship between GH response and age. PDC also correlates highly significantly with age (rho=0.4, P<0.01) but the PDC/GH response relationship remains significant after the effect of age is partialled out (rho=0.33, P=0.05). Neither severity of depression nor drug-free interval prior to testing correlate significantly with GH response or PDC. Of our measures of endogeneity, neither the Newcastle scale nor the Feinberg-Carroll discriminant index correlate with GH response or PDC. RDC definite endogenous depressives (n=32) however show a just significantly lower GH response than RDC probable and non-endogenous depressives (n=9; UO=90, P<0.05, 1-tailed) We conclude that a common neurochemical mechanism may be responsible both for blunting of the GH response to clonidine and of the cortisol response to dexamethasone in depression.

¹CHECKLEY, S. A. et al (1981) *British Journal of Psychiatry*, 138, 51

²SIEVER, L. J. et al (1982) *Psychiatry Research*, 6, 171.

³KATONA, C. L. E. et al *British Journal of Psychiatry*. In press

Genetic markers in late paraphrenia—A study of HLA antigens

Drs M. Naguib, P. McGuffin, R. Levy, H. Festerstein and A. Alonzo, *The Maudsley Hospital, London*

Thirty-one unrelated British patients with late paraphrenia were typed for HLA-A, -B, and -C. Increased frequencies of BW55 (P=0.036, relative risk 4.05), B37 (P=0.008, relative risk 4.34) and CW6 (P=0.056, relative risk 2.24) were found relative to controls. Unlike findings in paranoid schizophrenia there was no HAL-A9 association. This suggests that patients with paraphrenia may be biologically and genetically distinct from schizophrenics. No significant negative association was found with any of the HLA anti-

gens tested. Although the association found does not remain significant after correction for the number of antigens tested, the primary association seems to be with B37 which, despite occurring only in a minority of patients, shows the lowest corrected P value and highest relative risks. If the HLA-B37 association is confirmed in a new study it would make possession of this antigen a strong risk factor for developing the disorder.

Acute and tardive akathisia

Drs T. R. E. Barnes and W. M. Braude, *Horton Hospital, Surrey*

In a recent study¹ of consecutive acute psychiatric admissions receiving antipsychotic medication, acute akathisia developed in 25% as a strongly dose-related phenomenon. The signs and symptoms of mild, moderate and severe drug-induced akathisia were elaborated, and allowed distinction from agitation and restlessness associated with psychiatric illness. The relationship between akathisia, parkinsonism and response to anticholinergic agents was examined. In the majority of patients there was no association between akathisia and severity of parkinsonism, and anticholinergics were relatively ineffective. In the small group of patients with severe parkinsonism, akathisia was invariably present, and in this context both the akathisia and parkinsonism responded to anticholinergic treatment.

Diagnostic criteria for acute akathisia were derived, and applied in a sample of established schizophrenic outpatients.² Previous work³ had suggested that akathisia and oro-facial tardive dyskinesia commonly coexisted in such patients, and this finding was confirmed. A group of patients with 'chronic' akathisia were identified, which included cases with acute akathisia which had persisted, as well as cases of 'tardive' akathisia. This tardive akathisia variant is characterised by a late-onset, an association with oro-facial and trunk and limb dyskinesia, in that the condition appears or worsens on drug dose reduction or withdrawal, and tends to improve when the dose is increased.⁴

¹BRAUDE, W. M., BARNES, T. R. E. & GORE, S. M. (1983) *British Journal of Psychiatry*, 143, 139-150

²BARNES, T. R. E. & BRAUDE, W. M. *Archives of General Psychiatry*, 185, (in press)

³BARNES, T. R. E., KIDGER, T. & GORE, S. M. (1983) *Psychological Medicine*, 13, 71-81

⁴BRAUDE, W. M. & BARNES, T. R. E. (1983) *American Journal of Psychiatry*, 140, 611-612

Screening for alcohol problems in a non-clinical setting

Dr J. Spencer, *Director of Clinical Services, W. A. Alcohol and Drug Authority, Salvatori House, 1st Floor 35 Outram Street, West Perth, WA 6005*

With the realisation that early intervention and treatment carries a better prognosis, agencies face the problem of detection and referral of such patients. The problems of social norms and denial are of particular significance in offering help to those with drinking problems.

In an attempt to overcome these obstacles, the local Alcohol and Drug Authority has been using mobile alcohol screening and display units in suburban shopping centres. Included in these displays have been computerised terminals incorporating simple alcohol knowledge questions and the CAGE and SMAST questionnaires. The results indicate

that over 30% of respondents were drinking pathological amounts of alcohol and that many were doing so in ignorance of what is regarded as safe levels of drinking. There was a significant correlation between the CAGE and SMAST questionnaires thus confirming their validity in such community surveys. The paper discusses some of these issues and results.

A study of deliberate self-poisoning in the accident and emergency department

Dr D. W. Owens and Mr S. J. Jones, University Hospital, Nottingham

It is acknowledged that many accident and emergency departments do not admit all the patients presenting with deliberate self-poisoning. Little is known about such screening procedures and this study was carried out to quantify and make some evaluations of the practice in one department.

Every patient attending the Nottingham accident and emergency department over a four-month period in early summer 1982 with the presenting complaint of overdose was identified retrospectively. The case-notes of all 525 patients were examined for demographic data, drugs and alcohol taken and the initial management of the case. The Nottingham Psychiatric Case Register was examined to provide information about previous psychiatric contacts and use of psychiatric services over a two year follow-up period.

The study shows statistically significant differences between those patients admitted to a ward and those who return home from A & E, in respect of likelihood of psychiatric morbidity and use of psychiatric services. The results suggest that whilst one in three self-poisoning patients returns home directly from the accident department, there is some degree of appropriate selection in that department regarding factors of psychiatric importance.

Application of dexamethasone suppression test in dementia

Drs M. T. Abou-Saleh, E. M. Spalding and J. M. Kellett, Department of Psychiatry, St. George's Hospital, London and Dr A. Coppen, MRC Neuropsychiatry Laboratory, West Park Hospital, Surrey

The performance of the Dexamethasone Suppression Test (DST) was investigated in 60 demented patients including 14 patients admitted to a psychogeriatric assessment ward, 29 electively admitted patients and 17 long-stay patients in comparison with 37 middle-aged and elderly controls. It was shown that acutely admitted patients had the highest rate of DST non-suppression (86%), whilst electively admitted patients had a lower rate (55%), in comparison with long-stay admissions (65%), and controls (11%). In the electively admitted group abnormal DST results were associated with weight loss and depressive symptoms. There was no association between DST results and cognitive, behavioural and cortical dysfunction, including degree of cerebral atrophy as determined by CT scan. It is suggested that abnormal DST response reflects a biological dysfunction that cuts across the clinically established boundaries of psychiatric nosology and indicates a versatile component in psychiatric disturbance that could serve to predict or monitor the effects of physical and psychological intervention.

Poster presentations

Therapeutic factors in bulimia nervosa

Dr U. Schmidt, The Maudsley Hospital, London

The purpose of this study was to examine the usefulness of therapist-aided cue exposure to sight, smell and taste of food in the treatment of bulimic patients. Binge-eating was construed as an avoidance behaviour that helps the individual to reduce or avoid unpleasant moods (such as boredom, anxiety, and the discomfort or conflict produced by the desire to eat 'forbidden' foods). It was postulated that in binge-eating as in other avoidance behaviours cue exposure to food should result in response decrement (of the urge to binge). Four patients who fulfilled Fairburn's and Cooper's (1984) operational criteria of bulimia nervosa were each given 12 sessions of cue exposure to food. A single-case multiple-baseline strategy was used, with in-session and between-session measures. Three out of the four patients showed moderate to marked improvement at one-month follow-up which in two cases was maintained at six-month follow-up; one patient had partially relapsed. The fourth patient dropped out of treatment after nine sessions. Two of the improved cases showed in-session habituation and some degree of across-session habituation. The patient who dropped out showed within-session habituation but no between-session improvement.

The results of this small study are preliminary evidence that cue exposure to food might be a useful adjuvant in the treatment of bulimia nervosa.

The effect of a nurses' strike on the psychiatric care provision of a mental hospital

Drs M. Abdelkader, Hollymoor Hospital, Birmingham, J. Kogeorgos, Horton Hospital, Epsom and P. Fonagy, University College, London

Mental hospitals have been traditionally the main place of treatment for the psychiatrically ill, but in the past few years, their role has been questioned and there are current plans to close a substantial number of them.

We had the opportunity of evaluating the importance and pattern of psychiatric care provision of a mental hospital (Horton Hospital) during a six-month period in which this provision was drastically curtailed by industrial action of the nursing staff. The clinical, social and demographic characteristics of all patients admitted during this period (112) were analysed and compared with those of patients admitted during the corresponding period of the previous year.

In the study period the number of informal patients admitted dropped by 50%, but this was partly related to admission policies. The number of compulsory admissions remained the same but patients were admitted predominantly under emergency sections (Section 4 and 2) rather than Sections 136 and 37 as in the control period. A proportion of the compulsorily admitted patients in the study period would have normally come in informally and there was a significant overall excess of patients with a history of an organic psychosis and patients who had not been professionally supervised in the community.

We discuss the significance of our findings in relation to current government attitude and policies for future mental health care provision.

Multiple sclerosis: A correlative investigation of psychiatric, neurological and psychological impairment

Dr J. Kogeorgos, Horton Hospital, Epsom, Mr R. Brown, North London Polytechnic and Mr C. Kennard, The London Hospital

Mental changes are a well recognised feature of multiple sclerosis (MS) and have been intensively studied, but their exact nature and pathogenesis remain unclear. This reflects the complexity of the disease and related methodological difficulties, notably the restricted and often uni-dimensional approach adopted in many previous studies.

We present preliminary findings from a collaborative investigation of 82 patients (53 females, 29 males; age range 20–60; mean 35 years), with 'definite' MS by McAlpine's criteria. These patients were free from other major organic problems or psychiatric history prior to the onset of their MS and were selected consecutively from the chronic patients attending a busy neurological out-patient clinic. Their assessment consisted of systematic psychiatric (Goldberg's Standardised Interview and Beck's Short Depression Inventory), neurological and psychological evaluation, combined with objective investigation of cerebral function (computerised tomography of the brain, EEG and Visual Evoked Potential recordings), all tests on each patient being completed within a week. The results were analysed to identify relationships between these and other clinical variables including age, sex type and length of illness, and were compared with those from a control group of other chronic neurological out-patients of similar overall range of disability.

Both groups were cognitively impaired but the MS patients showed significantly greater psychiatric disturbance. However, this was non-specific and consisted mainly of affective changes especially depression, as well as irritability and a feeling of excessive fatigue. The psychiatric morbidity present showed little relationship to either physical or intellectual impairment.

An analysis of the elderly in a general psychiatric hospital

Dr S. M. Rea, Tyrone and Fermanagh Hospital, Northern Ireland

A cross-sectional survey assessed all elderly patients in a general psychiatric hospital in Co Londonderry, Northern Ireland. They comprised 42% of the total hospital population. The unusual finding of an almost equal number of male and female patients is discussed.

The cohort was divided into 'new long-stay' (22%) and 'old long-stay' (60%) as defined by Wing (1980). The majority of 'new long-stay' patients had a diagnosis of dementia while the majority of 'old long-stay' patients had a diagnosis of schizophrenia.

The Behaviour Rating Scale (BRS) of the Clifton Assessment Procedures for the Elderly (CAPE) (Pattie & Gilleard) was used to measure the 'level of dependency' of all elderly patients. A high degree of dependency was found in 60%. Only seven patients who did not have a diagnosis of dementia, and who had been in hospital less than five years, were identified in the continuing care wards as potentially suitable for a more independent existence outside hospital.

The Information/Orientation subtest of the CAPE revealed that the majority of non-organic patients also had very poor orientation scores. A more detailed analysis of the BRS estimated the degree of apathy and socially disturbed behaviour within the three main diagnostic groups.

These findings which confirm that the elderly in a psychiatric hospital are a severely handicapped group, have major implications in the development of the most suitable ward milieu and in the organisation of a psychogeriatric service. Government policy to replace large mental hospitals and the development of alternative care for the elderly in the community are also discussed.

Lesion size and outcome following stereotactic subcaudate tractotomy

Drs A. S. Hale, J. Bartlett and P. K. Bridges, Geoffrey Knight Psychosurgical Unit, London

Stereotactic subcaudate tractotomy (SST) is a psychosurgical technique for the relief of intractable depression which has been carried out in Britain since the mid 1960s.¹ Although the clinical effectiveness of the operation has been generally accepted,² it has not been possible to perform an adequate or ethically acceptable, double blind, or even open, controlled trial. Apart from anecdotal evidence from early work¹ there has been no examination of the effect of varying lesion size on outcome. The mechanism of action of SST is thought to involve the severing of projection fibres from the limbic system to the frontal cortex. There should thus be an optimal size for the lesion.

We report a prospective, double blind, non-controlled study of the effect on outcome of varying lesion size. The number of Yttrium 90 rods used thus varied, irradiating a range of volumes of brain. Both patient and assessor were blind to the number of rods used. This study is as near to the needed double blind controlled study as is ever likely to take place. Two hundred and fifty nine consecutive cases of intractable depression were given one of four lesion sizes on a cohort basis. 12, 16, 20 or 24 rods were used. Outcome was assessed blind to varying lesion size, one year after SST. Outcome was defined as: 1. Asymptomatic. 2. Well, mild residual symptoms. 3. Improved, significant symptoms remain. 4. Unchanged. 5. Worse. Results are shown in Table 1.

Table 1
Numbers of patients in each outcome group with varying Y90 rod number

		Number of rods			
		12	16	20	24
Outcome Category	1	11	6	14	1
	2	30	10	29	2
	3	36	16	21	2
	4&5	31	17	29	4

There is a sex difference in lesion size effect on outcome. For males, the optimal size was 20 rods, showing significantly more improvement than 16 rods ($P=0.01$), which in turn was significantly better than 12 rods ($P=0.04$). For females, this effect was weaker, only the 12 v 20 rod comparisons being significant ($P=0.04$). There was no significant relationship between age and outcome. Outcome was poorer in the small 24 rod group, this lesion size being abandoned because of frequent post operative confusion. The optimal lesion size was thus 20 rods. The data provides supportive

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evidence for the effect of SST being directly related to the production of the subcaudate lesion.

¹KNIGHT, G. J. (1965) *Journal of Neurology, Neurosurgery and Psychiatry*, **28**, 304-310

²GOKTEPE, E. O. et al (1975) *British Journal of Psychiatry*, **126**, 270-280

Prognostic factors and outcome after 20 years in anorexia nervosa

Dr R. H. Ratnasuriya, The Maudsley Hospital, London

Forty one patients admitted to the Metabolic unit Maudsley Hospital, consecutively between 1959 and 1966, were followed up. The mean time of follow up from admission was 20 years. Information was gathered on 23 patients (56.1%) by personal interview, one patient (2.4%) by telephone interview which was confirmed by mother, on six by postal questionnaire, on four from GP. Seven patients were dead. One patient who was living abroad did not respond.

Outcome was assessed on weight gain and menstrual function and socioeconomic functioning. A retrospective case note study was also done to find prognostic factors related to outcome.

29.3% had a good outcome, 31.7% an intermediate outcome and 19.5% had a poor outcome. 17.1% were dead. Neurotic traits, personality problems, disturbed relationships in the family, age of onset of illness, length of illness on admission, age on admission and maximum weight gained in hospital were significantly related to outcome. Other factors mentioned in the literature were not found to be significant. These results and their relationship to the results of a five year follow-up study of the same group of patients are discussed.

Sub-acute limbic encephalitis

Dr J. A. N. Corsellis, Runwell Hospital, Essex

The limbic areas of the brain have long held a particular fascination for those interested in organic psychiatry. Although they are reasonably well-defined within the medial depths of the cerebral hemisphere, few pathological conditions are well-enough confined to them to justify even primitive attempts to establish clinical and neuropathological correlations. One exception may be the way in which at least certain parts of the limbic grey matter become involved in a curiously selective, and fortunately rare, form of encephalitis (or encephalopathy) which has a close, but by no means, inevitable association with carcinoma, usually of the lung.

The present paper tries to outline the ways in which the condition has come to be recognised as one of the lesser known causes of marked intellectual impairment, possibly dementia, and perhaps most striking of all, a marked disturbance of recent memory.

The clinical and the neuropathological features of the disorder are summarised and the latter illustrated. Some of the questions raised by the nature and the localisation of the pathological process are raised.

The nature of chronic schizophrenia

Dr Eve C. Johnstone, Northwick Park Hospital and Clinical Research Centre, Middlesex

This paper is a summary of Northwick Park studies of schizophrenia which do not concern acute episodes. A survey of 510 in-patients with chronic schizophrenia showed widespread severe abnormalities which were not associated with past physical treatment but were significantly related to duration of illness/hospitalisation. Subsequent studies comparing this population with a sample of discharged schizophrenic patients and one of institutionalised manic depressives showed closer similarities between the two schizophrenic groups than between the two groups of in-patients. It was concluded that schizophrenia was frequently associated with serious defect and that this did not depend upon environmental circumstances. The degree of defect is highly variable and studies of the structure of the brain and of anterior pituitary function, while showing abnormalities in chronic schizophrenia, do not provide clear cut correlates with clinical state. Recent studies of early outcome indicate that severe and early defect is still commonplace. The influence of treatment upon this is discussed.

In vitro and in vivo studies of dopamine receptors

Dr F. Owen, Division of Psychiatry, Clinical Research Centre, Middlesex

Neurochemical studies in post-mortem brain material from schizophrenics have failed to provide any evidence for increased dopamine turnover in the central nervous system. However, there have been a number of reports of increased dopamine D² receptors in brains from schizophrenics compared with controls. It remains unclear whether or not this increase in D² receptors is entirely due to the effects of neuroleptic medication. More recently attention focused on assessing D² receptors in living subjects. Spiperone can be brominated at the para position on the benzene ring to produce parabromospiperone which retains its neuroleptic activity and D² ligand binding characteristics. When 77 bromine is used as the bromine source 77Br-bromospiperone (77 Br-BrSp) is produced which is a gamma emitter with a half-life of 56 hours and hence is suitable for clinical studies.

With 77Br-BrSp as ligand good computer reconstructed images of the radioactivity in human brain have been obtained using the IGE 400T gamma camera with a star computer system. When the technique was used to assess D² receptors in drug-free schizophrenic patients a small but significant increase, compared with controls, was obtained.

Winter Quarterly Meeting, 1986

*The Royal Society, 6 Carlton House Terrace, London SW1
(21–22 January 1986)*

Surveys of the development of psychiatric services for old people

Dr J. P. Wattis, St James's University Hospital, Leeds

Specialised psychiatric services for old people have grown rapidly over the last 15 years. This fact has been invisible in national statistics because psychiatry of the elderly is not treated as a subspeciality for statistical purposes. Three surveys have been undertaken to define this major new area of psychiatry. The first, in 1980, found 106 specialist consultants (though the real total was estimated to be around 120). Over half the respondents had started to work in the field in the preceding five years.

By 1983 there were at least 150 consultants working predominantly with old people though recruiting was depressed by a shortage of suitable senior registrar training posts. There were more services with more than one consultant and proportionately more full time appointments. The current survey expects to find 200 or more consultants in the field.

The studies have also examined staffing and facilities, and a synopsis of results is presented. The relationship of old age psychiatry to general psychiatry is also explored.

What should liaison psychiatry be doing?

Dr R. Mayou, University Department of Psychiatry, Warneford Hospital, Oxford

In North America, liaison psychiatry has rapidly become a sub-speciality which claims an expanding role in the psychiatric care of general hospital attenders, in training psychiatrists, and in teaching other professional groups. There has also been increasing interest in Britain, but services are poorly organised and usually provided by general adult or child psychiatrists, who feel that they have too little time to provide a satisfactory clinical service.

Review of the prevalence of psychiatric morbidity in general hospitals shows that it is common as (a) a response to physical illness, (b) psychiatric disorder presenting with physical complications (deliberate self-harm, alcohol, eating disorders), (c) hypochondriacal somatic symptoms. Many of these problems are unrecognised by physicians and surgeons and even when recognised, it is uncertain that they are adequately treated. Although much of the psychiatric disorder seen in the general hospital does not require specialist care, it is apparent that major problems are not being treated and that these cause considerable distress for patients and families, and limit the effectiveness of medical treatment.

Psychiatric liaison teams are only able to see a very small proportion of the general hospital patients. Although hospital doctors, nurses and other members of the clinical teams must continue to be responsible for the major part of psychological care, it is uncertain whether they have the necessary skills. The liaison psychiatry team has an important role in providing a well-organised emergency and consultation service and should, in addition, attempt liaison links with those general hospital units which have a particularly high prevalence of major psychiatric problems. Liaison psychiatry should be a part of every training programme for general and child psychiatry.

Alcoholic dementia

Professor W. A. Lishman, Institute of Psychiatry, London

The problematical concept of alcoholic dementia is discussed with emphasis on its uncertain nosological status. The lines of evidence which have a bearing upon it—all inconclusive of themselves—are presented, including recent evidence from psychometry and CT scanning. Possible pathogenic mechanisms are for discussion.

Can schizophrenia be localised in the brain?

Professor N. C. Andreasen, Department of Psychiatry, University of Iowa, USA

This presentation reviews several lines of evidence concerning the localisation of brain abnormalities in schizophrenia. It discusses hypotheses concerning diffuse versus specific localisation. Evidence based on CT scanning, BEAM, NMR, PET, regional blood flow, and neuropathological studies is reviewed. The preponderance of evidence appears to suggest a specific rather than diffuse localization. The specific site has not been identified, and in fact multiple sites may be involved.

Eli Lilly lecture: Mindlessness and brainlessness in psychiatry

Professor Leon Eisenberg, Harvard Medical School, Boston, USA

Brain and mind are as warp and woof to the fabric of psychiatry. Yet, psychiatric theories have tended to be, as with psychoanalysis, 'brainless' or, as with biological psychiatry, 'mindless'. The sources of this polarisation are explicated by the history of our speciality. Nonetheless, an informed review of the phenomenology of psychiatric disorders makes evident the inadequacy of unitary formulations.

Exclusively biological and psychological theories in psychiatry stand in relation to one another much as wave and particle theory do in subatomic physics. Both formulations are necessary to account for the universe of observation. The unique role of psychiatry in medicine lies in the extent to which it contributes to an understanding of the ways in which the mind is embodied and the body literally mindful. However far we may be from that goal, the striving for it is the *raison d'être* of our speciality.

Behavioural psychotherapy in general practice

Professor I. Marks, Institute of Psychiatry, London

Behavioural psychotherapy can often be given as successfully by general psychiatrists as by specialists. Clinicians need to delineate the clinical features of the problem, know when behavioural methods are applicable and their principles. Most behavioural approaches involve self-treatment by the patient with the clinician acting as guide and monitor. Using a suitable manual and diary, agoraphobics, social phobics and specific phobics improved as much with self-exposure by following a manual and keeping an appropriate diary as others who were given exposure instructions by a psychiatrist. Therapist-aided exposure added little to self-exposure + self-imposed response prevention for compulsive ritualisers, both approaches producing satisfactory gains. Much sexual dysfunction can also

be helped by general psychiatrists with simple instructions and monitoring. Anxiety and conduct disorders in children have also responded to simple guidance, as has enuresis. Specialist behavioural treatment is only indicated if behavioural guidance by general psychiatrists has failed.

Recent advances in prophylactic lithium treatment

Professor M. Schou, Aarhus University Institute of Psychiatry, Denmark

Now that the fear of progressive kidney damage during long-term, lithium treatment has been allayed, attention may be focused on appropriate information and instruction of patients, ongoing psychological support, clinical and laboratory monitoring, choice of optimal serum lithium concentration for maximum of benefit and minimum of side-effects, avoidance of situations involving risk of lithium intoxication, early detection and proper treatment of lithium intoxication, the significance of salt and water deficiency, interaction with other drugs, and course of action in case of incomplete or lacking response.

Patients who attribute their psychiatric symptoms to food allergy

Dr K. J. B. Rix, St James's University Hospital, Leeds

Wide publicity is being given to unsubstantiated claims that food allergy is responsible for all sorts of conditions in addition to the classical atopic diseases of asthma, eczema, urticaria and gastrointestinal food hypersensitivity. However, the most common reason for failing to tolerate foods is psychological and most patients with supposed food allergy present with psychiatric symptoms.

Although many lay people use the term 'allergy' to refer to any negative reaction to a substance and some professional groups apply it to all idiosyncratic or acquired adverse reactions, the term should be restricted to immunologically-mediated adverse reactions.

This lecture discusses the differential diagnosis of patients with supposed food allergy presenting to the adult psychiatrist. It encompasses distaste, food fads, food phobia, anorexia nervosa, organic hypersensitivity (including food allergy), psychological hypersensitivity and normal reactions to toxins and pharmacological agents. The diagnosis should only be reached after appropriate history-taking, examination, and, in some cases, dietary investigations, double-blind food challenges and immunological tests

Unorthodox investigations and treatments are mentioned.

Violence and psychosis: offenders and patients

Dr P. Taylor, Institute of Psychiatry, London

The best calculated estimates of risk suggest that only a very small proportion of psychotics become seriously violent. Nevertheless large numbers of them appear in prisons and their rate of violent offending is probably increasing. Violence is a key reason for admission to hospital for a substantial minority.

Reports differ in the importance they attach to the psychotic process as a precipitant of violence. Those based on prison samples tend to show the illness as less relevant compared with those based on hospital samples. In the only

large interviewed sample of psychotic offenders all but 7% were actively symptomatic at the time of their offence and nearly 50% had probably been driven by their delusions or hallucinations. Only 10% of this series were newly identified cases. Other work confirms that serious violence is rarely a presenting feature of the illness. Is violence by psychotics thus chiefly a manifestation of the failure of psychiatry and supporting services? Among men remanded to prison for a range of offences, 71% of schizophrenics, the majority of whom were said to be actively ill, failed to find a hospital disposal.

Psychiatric aspects of Parkinson's disease

Dr G. E. Berrios, University of Cambridge

A short historical incursion, showing that Parkinson did not mean to exclude 'psychiatric' disorder from his definition of the disease is followed by a discussion of some of the methodological difficulties affecting research in this area.

Preliminary results from the Addenbrooke's Study (based on 58 subjects under 70) are presented. Exploratory factor analysis extracted four factors: depression, cognition, illness/treatment and motor status. Hypotheses therein suggested were tested internally or by carrying out additional subprojects.

The following conclusions are drawn: 1. Evidence for affective and cognitive disorder can be found in a sample of PD sufferers under 70. 2. These disorders are not clinically coterminous with 'major depression' or 'dementia', respectively, as conventionally defined. 3. High doses of Ach compounds predict low free recall and affect the 'A' value in a signal detection task. 4. There is no correlation between severity of motor and affective disorder. 5. There is no correlation between severity of motor and cognitive disorder. 6. In general, motor symptoms have (apart from rigidity) low predictive value with regards to mental disorder. 7. PD subjects exhibit no conservative bias in a signal detection task but show memory impairment. 8. PD subjects show no frontal lobe involvement as defined by a verbal fluency task. 9. The Addenbrooke's Scale offers some advantage in the assessment of depression in the PD patient. 10. Longitudinal studies of PD cohorts are recommended. 11. Clinical and instrumental identification of primary neuropsychological abnormalities should help distinguish phenocopies from real psychiatric syndromes.

Life events and the onset of illness

Dr F. Creed, Manchester Royal Infirmary

Life events are clearly related to the onset of psychiatric illness and the relevant vulnerability factors are becoming better defined. The relationship of life events and the onset of physical illness is less clear and the vulnerability factors involved have yet to be elucidated.

Studies of life events and the onset of illness affecting the gastro-intestinal tract suggest that a different type of event may be related to the onset of definite organic disease than the onset of non-organic gastro-intestinal disorder. In the latter case the type of life event has similarities with those preceding deliberate self-poisoning. Some intervening variables between life event and somatic illness are suggested.

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Dementia

Ultrastructural studies in Alzheimer's disease

Dr D. M. A. Mann, University of Manchester

Ultrastructural (electron microscope) studies in Alzheimer's disease have to date concentrated on four aspects of pathology. (a) ultrastructural characterisations of senile plaques, neurofibrillary tangles and Hirano bodies; (b) cytological changes in nerve cells in relationship to neurofibrillary degeneration, (c) synaptic alterations; and (d) aetiological factors.

Senile plaques are thought to evolve through several pathological stages. They start out as primitive plaques consisting of clusters of dystrophic neurites, mature into classical plaques with amyloid core, astrocytic processes and many neurites and finally regress into burnt out plaques in which the neuronal elements are lost. Tangles comprise of sheafs of paired filaments, each of 10 nm diameter wound helically at a periodicity of 150 nm. Hirano bodies are paracrystalline arrays of filaments thought to comprise of actin.

Accumulation of neurofibrillary material in cortical pyramidal cells is associated with progressive reductions in the amount of useful cell cytoplasm, the size of the nucleus, the amount of rough endoplasmic reticulum and the number of ribosomes. Mitochondria are unaltered and lipofuscin is increased. Cells unaffected by tangle are unchanged in these respects. The protein synthetic capacity of nerve cells is thus diminished with accumulation of tangle; neurofibrillary degeneration may be the major cause of nerve cell loss in Alzheimer's disease.

Loss of synapses from temporal cortex averages over 50%, whereas neuronal perikaryal loss averages 30%; synapse to neurone ratio is therefore decreased by 33% in Alzheimer's disease, suggesting a primary, or at least a preferential, destruction of nerve terminals.

Electron microscope observations in Alzheimer's disease have identified various 'organelles' within nerve cells or neuropil, possibly indicative of pathological change relevant to the aetiology of the condition.

Selective cortical neuronal degeneration in Alzheimer-type dementia

Dr A. J. Cross, Department of Physiology, University of Manchester

Neurochemical studies on the post-mortem brains of patients with Alzheimer-type dementia (ATD) have described a marked loss of markers of cholinergic terminals in the cortex and limbic lobe, which correlates with the extent of clinical and pathological severity of the disease. Deficits have also been described in other ascending neurones, particularly those containing serotonin and noradrenaline. The loss of terminals of these ascending neurones cannot account for the extent of cortical degeneration present in ATD, and recent evidence suggests that these ascending neurones degenerate in a retrograde fashion.

A number of markers of intrinsic cortical neurones

have been studied in ATD. Of these it appears likely that somatostatin concentrations and serotonin S2 receptors are reduced in ATD, possibly reflecting a loss of a subset of cortical neurones. In addition, high-affinity glutamate uptake sites are reduced in ATD. The glutamate uptake site is a marker of neurones using glutamate as transmitter and includes cortico-cortical (association) neurones and corticofugal neurones. The relationship of these changes to the pathogenesis of the disease will be discussed.

Brain imaging techniques in the evaluation of dementia

Dr J. A. O. Besson, Department of Mental Health, University of Aberdeen

Two brain imaging techniques that have relevance in the clinical study of dementing conditions are nuclear magnetic resonance (NMR) imaging and single photon emission tomography (SPET).

NMR imaging involves the pictorial reconstruction of data derived from the behaviour of mobile H nuclei following excitation by magnetic forces. These magnetic forces are applied in the form of a series of pulses in different directions (90° or 180°) from the main magnetic field in which the subject is situated. The Aberdeen imaging system is based on an intercalated saturation recovery (90° pulse) inversion recovery (90° pulse preceded 200 ms before by an 180° pulse) sequence but other types of pulse sequences are possible.¹ Three types of data can be obtained by this method: proton density (PD), inversion recovery (IR), and spin lattice relaxation time (T). Images based on these data can be obtained for each section and transverse, coronal, and sagittal displays are possible.

In SPET the distribution of a gamma-emitting radio-labelled compound in an organ following intravenous injection is imaged by a camera made up of a series of gamma radiation detectors. The technique described in the current study images regional cerebral blood flow (rCBF) by detecting the distribution of 123 labelled *n*-isopropylmethylamphetamines (IMP),² which is taken up by the tissue on first pass through the brain following intravenous injection.

Diagnostic criteria based on DSM-III were used; patient groups studied were Alzheimer's disease (ATD), multi-infarct dementia (MID), Parkinson's disease with and without dementia, Korsakoff psychosis, alcoholic cortical atrophy (ACA) and Huntington's chorea. Hachinski ischaemic score was applied to the separation of ATD and MID groups. Age-matched normal controls were studied.

Changes observed in dementia using NMR are cortical atrophy and ventricular change (PD mode—best), periventricular white matter change (PVC) (increased T and IR, no change in PD), recent infarct (increased T and IR, no change in PD), old cystic infarct (increased T and IR and reduced PD), old gliotic infarct (normal T and increased PD).³ These changes may be quantified.⁴

In ATD, 50% of patients have elevated T lesions in the posterior watershed white matter areas on NMR⁵ and 90% of patients have bilateral reduction in cerebral blood

flow in the posterior perietal grey on IMP compared with none of the controls.⁶ In MID, 75% of patients had infarcts, PVC, or both together on NMR, and 100% of patients had focal or marked generalised reduction in rCBF on IMP, compared with none of the controls.

In ACA, cerebral atrophy only was seen on NMR and generally irregular radionuclide uptake on IMP, predominantly frontal. In pure Korsakoff psychosis NMR and rCBF appearances are normal, but where coexisting alcoholic cortical atrophy is present, predominantly frontal reduction in rCBF can be identified.

In Huntington's chorea increased intercaudate width and reduced volume of the head of the caudate can be seen, but no measurable change in T on NMR. Those with dementia do not show reduction in rCBF on IMP. Patients with depressive pseudodementia do not show abnormalities on rCBF imaging.

NMR and IMP are complementary in providing diagnostic and pathogenic information in organic brain disorders.

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Family stress and strain in carers

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Most dementing old people are cared for in the community by relatives. Little is known about caregivers' perceptions of dementia or how these perceptions affect coping. Nor is much known about the effects of many years of caregiving on marital and social relations, factors that influence psychological well-being, or whether or not service provision influences caregivers' willingness to continue giving care in the face of continuing deterioration. This paper considers these issues in light of findings from a small scale, intensive interview study of 24 co-resident and 24 non-resident caregivers. The implications of these findings will be discussed in relation to the current government's emphasis on community care.

Decentralised budgets and community care: the cost-effectiveness of this approach with particular reference to the management of the confused elderly

Dr David Challis, Assistant Director, Personal Social Services Research Unit, University of Kent

The community care approach, originally developed in Kent and subsequently replicated elsewhere, was devised to enable social services departments to provide effective alternatives to residential homes and long stay hospital care for frail elderly people. It involved the decentralisation of resources to individual social workers, who act as case-managers, with defined caseload and expenditure limits to ensure accountability, so that more flexible responses to need could be devised and the fragmented services integrated into a more coherent package of care.

The results of the evaluation indicated that when patients were followed over a one-year period more of those receiving the new approach had remained in their own homes and fewer were in long-term care settings. On a range of indicators of quality of life for both the elderly person and their carers there were significantly greater improvements than for those receiving the standard range of services. The costs to the social service department were slightly lower than, and those of the NHS similar to, those of standard provision.

Although the study was not specifically focused on the confused elderly, they comprised about one quarter of the cases. It appeared that the approach involving careful assessment, individual care plans and more flexible responses through control of a budget made it possible to manage the care of these people in their own homes more effectively than has usually been the case. However, the numbers were relatively small and a large study is required to test these observations.

Overall, the findings indicated that effective case-management is an essential feature of more effective home care of the frail elderly. The new approach appeared most appropriate and cost-effective for the extremely mentally and physically frail who were living with others, and also for the less frail, socially isolated elderly person prone to depression.

Forensic psychiatry

A survey of the regional secure units (opened and planned) in England and Wales

Dr P. R. Snowden, Prestwich Hospital, Manchester

In 1975 the Butler Committee on Mentally Abnormal Offenders recommended that each Regional Health Authority provide Regional Secure Units, and central government capital funding and partial revenue funding was made available. Some Regional Health Authorities opened Interim Secure Units by converting existing wards, while plans for Regional Secure Units were being developed. The first Regional Secure Unit did not open until 1980, and the next two units did not open until 1983. In order to investigate the reasons for the delays in opening these permanent secure facilities in England and Wales the plans for each Region were surveyed in detail between October 1982 and March 1984. The progress of each Regional Secure Unit development has been regularly updated, most recently in March 1986. Because of problems with staff recruitment—in particular suitable nursing staff—most of the Regional Secure Units that are currently admitting patients have only opened a proportion of their beds. The link between forensic psychiatrists and the Regional Secure Unit planning process is discussed, as is the relationship between Regional Secure Unit size and site and the type of forensic psychiatry service that can be provided in health regions with different needs, sizes and population densities.

Evaluating secure units—some methodological difficulties

Mr M. J. Berry, Department of Psychiatry, University of Birmingham

This paper illustrates some of the methodological difficulties in evaluating the secure unit programme, based on the work of the RSU Research Unit of Birmingham University. The study was established by the late Professor Gwynne

Jones in 1982 to carry out a three-year evaluation of interim and regional secure units in seven NHS regions in England. Data will be reported on 400 admissions across 11 units. Four hundred variables have been included in the study, but for the benefit of this paper only those associated with the admission pattern will be presented, to highlight both the similarities and differences between the units.

Data will be presented to show some of the characteristics of admitted patients (e.g. referral source, diagnostic label, ethnicity, sex, age, criminal and psychiatric history) and how they vary between regions, and various methods of evaluation of RSUs will be discussed.

The first two years of a Regional Secure Unit and service for adolescents—types of referral made

Dr Susan M. Bailey, Regional Forensic Psychiatric Service, Manchester

A total of 375 patients (264 male, 111 female; age range 11–18) were seen. An open referral system was operated; the majority of adolescents being referred by local Social Services Observation and Assessment Centres. Child, adolescent and forensic psychiatrists referred those adolescents who displayed the most disturbed behaviour.

Types of behaviour most likely to lead to court appearances in both sexes were stealing, damage to property, fire setting, non-school attendance, and (in boys) sexual deviance. Aggression to others (even of a serious nature) was unlikely, particularly in girls, to lead to a court appearance. Over two-thirds of those adolescents who were 'stealing' had no psychiatric illness, the remaining third having predominantly conduct disorders. Fire setters tended to display mixed emotional conduct disorders, as did the boys committing sexual offences. This latter group also contained a significant number of boys with mild mental handicap associated with minor physical handicaps.

Psychotic adolescents presented with a full range of disturbed behaviour, but sadly were not being recognised as psychotic when within the community and were therefore being referred via the penal system or the courts after committing serious offences.

Substance abuse was not a predominant reason for referral, but when assessed, up to 20% of younger adolescents gave a significant history of solvent abuse, and up to 40% of 16–18 year-old boys gave a significant history of alcohol abuse (the latter contributing to their pattern of offending). Surprisingly, few referrals were made because of problems of self-mutilation or abuse. The majority of such referrals were girls, with often a history of sudden maternal death in the preceding two years and subsequent unresolved grief reactions.

The 15 in-patients admitted over a twelve-month period (11 male, 4 female, average age 15½) spanned all diagnostic groups, behaviour leading to admission included violence to self and others, sexual offences, and arson. All our in-patients had histories of a disturbed and chaotic family background and, with the exception of our psychotic youngsters, had all had multiple last placements within the 'care' system. Minor physical handicaps were common as, on examination, were 'soft' neurological signs and abnormal electroencephalograms.

Although only a new service, we are beginning to see within our out-patients and in-patients the not unexpected finding that the greater the number of psychological and social 'insults' an individual receives from an early age the

more likely they are to present with disturbed behaviour in childhood and in adolescence, but perhaps more interestingly we are picking up evidence of previously unrecognised organic features in this patient group. These features may well be contributing to their behaviour, and must be an important factor in the likely prognosis for this group. It is interesting to note that seriously aggressive and dangerous behaviour, with the exception of sexual deviance in boys, was more common in the group of patients who did not have a 'forensic' label.

Changing patterns of admission to and discharges from special hospitals

Dr J. Higgins, Rainhill Hospital, Liverpool

This paper describes a study of patients from the Mersey Regional Health Authority catchment area who were resident in all four special hospitals on 1 January 1981 and those patients who were admitted from or discharged to the Mersey Region between then and 31 December 1985. Comparison is made between these patients and the expected characteristics patient derived from DHSS annual reports into patients in Special Hospitals, the Mersey Region being virtually 1/20th of the population for England and Wales.

On 1 January 1981 the expected total of patients in Special Hospitals was 98 (78 men and 20 women); the Mersey figures were only 84 patients (69 men and 15 women). The possible reasons for these initial discrepancies were reviewed; in view of the techniques employed they are unlikely to be due to sampling error.

Special Hospital patient numbers have declined over the study period, and the Merseyside total has declined in parallel. By 31 November 1985 the expected total was 84; the actual total was 68. The number of female patients has remained virtually static, but the number of male patients has dropped considerably. The changes in the Mersey male patient population are due primarily to the marked decline in those suffering from psychopathic disorder. The number classified as mentally ill (all chronic schizophrenic patients) and mentally handicapped remained virtually constant.

Over the study period 43 males and 10 females were discharged. Of these, 19 males and four females were admitted to the Regional Secure Unit, nine males and four females to mental illness and mental handicap hospitals, and eleven males and two females were discharged to the community. Two males were returned to prison and two males were dealt with otherwise. This pattern highlights the fact that the Regional Secure Unit is playing its role but not to the exclusion of the other appropriate agencies.

Over the study period 26 males and 11 females were admitted to Special Hospitals. Of the males, 15 are suffering from mental illness (two of whom were recalled); six males were suffering from psychopathic disorder, of whom three were recalled. The females were quite equally divided by diagnosis, and only one was recalled. A feature of both sexes is that the percentage admitted from the Regional Secure Unit has steadily increased between 1981 and 1986, in males at the expense of admissions directly from court and in females at the expense of admissions from other psychiatric hospitals. Although the figures are small, there is a clear trend towards the Regional Secure Unit being used as the placement of choice as a place of trial for discharged Special Hospital patients, the patients then being admitted

or returned to the Special Hospital if they proved to be chronic and dangerous.

Only those patients who were formally discharged from Special Hospitals were considered for the study; those sent to the RSU on trial leave were not included. As the patients remaining in Special Hospitals over the study period have become more chronic and more handicapped, there has been a change toward trial leave being the preferred mode of admission of Special Hospital patients to the RSU. This became more prominent in 1984 and 1985, thus making the number of patients remaining in Special Hospitals artificially low.

Child and adolescent psychiatry

Some psychiatric aspects of childhood epilepsy

Professor D. C. Taylor, University of Manchester

Epilepsy is a recurrent, transient brain dysfunction with altered consciousness associated with abnormal electrical discharges. It is best regarded as one form of brain dysfunction which may betoken the co-existence of other forms of brain abnormality, damage, or dysfunction. Epilepsy will often be associated with cognitive and behavioural disorder. Good prevalence estimates suggest that 1250 people will be in current care for epilepsy in the average district of 250 000 people. Half these people will have started epilepsy in childhood. Epilepsy is socially aversive. It is experienced by the public in a variety of guises; a transient severe fright, a wrecker of lives; or a killer. There are destructive, benign, and persistent forms, and the onset of epilepsy opens a variety of possible futures from the grave to the relatively trivial. These forms are discussed in detail.

Temporal lobe epilepsy is among the persistent forms, and is frequently related to lesions in the brain which provide a potential model for understanding hemispheric function in emotional, cognitive and behavioural spheres. Some of the evidence will be reviewed. Threats, losses and burdens abound in the care of children with epilepsy, and these are the stock-in-trade of psychiatric practice. Psychiatrists can improve adjustment. The nosology of the syndromes of cerebral dysfunction in childhood deserves attention.

Vulnerability factors, provoking agents and emotional disorders of school-age children

Dr I. M. Goodyer, Royal Manchester Children's Hospital

The association between environmental adversities and psychiatric disorder in childhood and adolescence has been the subject of considerable research over the past 20 years. Family, school and social factors have received varying degrees of attention according to the questions being asked and the interests of the professionals concerned. That adverse environments are significantly associated with emotional and behavioural disorders is firmly established.¹ However, the mechanisms by which these adversities exert their effects is less clear, and recent research has been examining this question in more detail.^{2,3} Recent research has established that recent stressful life events are associated with emotional and behavioural disorders as well as relatively long-standing adversities.⁴

The Manchester project on emotional disorders has looked at the relative contribution recent stressful life events make in the presence of other adversities, and one

aspect of our research has been to examine the role of adverse maternal factors in the presence of recent stress. A consecutive sample ($n=100$) of school-age children aged 7–16 years presenting with predominant features of anxiety or depression were compared with community controls free of psychiatric disorder and of the same age, sex and social class ($n=100$). We investigated four maternal factors (past and present psychiatric disorder, maternal supporting relationships, and the presence of potential attachment difficulties during pregnancy and in the first six months of the patient's life) and their association with emotional disorder in the presence and absence of recent stressful life events. All four of the maternal factors are significantly associated with emotional disorders before recent life events are taken into account. When the maternal factors are examined in the presence or absence of recent life events we found that (a) past and present psychiatric disorder in the mothers is associated with emotional disorder in both the presence and absence of events; (b) poor maternal support is associated with emotional disorder in the absence, but not the presence, of events; (c) potential attachment difficulties are associated with emotional disorder in the presence of life events but not in their absence (although nearly twice as many cases as controls report such difficulties without events). These preliminary results suggest that with the possible exception of early attachment difficulties the maternal factors examined in this study are more likely to exert a direct effect on disorder rather than be mediated through stressful life events, thus possibly operating as ongoing difficulties (i.e. provoking agents). The results do suggest that different maternal factors may exert different effects on disorder, and that further exploration of the relationships between maternal factors and recent stresses may reveal interactions of importance.

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Psychological aspects of children's consultations in primary care

Dr E. Garralda, Booth Hall Children's Hospital, Manchester

Work with children takes up a considerable amount of doctors' time in primary care, and it is generally assumed that general practitioners will undertake the total medical care of children in their practice, including both physical and psychological health. However, little is known about psychological disturbance in children presenting to general practitioners.

We carried out a study of psychological aspects of general practice consultations of children aged 7–12 years. Emotional or behavioural symptoms were the main presenting complaint in only 2%, but we found, from parental questionnaires and interviews, psychological disturbance to be present in a quarter. There was a trend for rates of

psychological deviance to be higher among attenders than in the general population, although this difference was marked only in the more socially advantaged areas. In the general practice sample psychiatric disorder was slightly more common in girls than in boys, and emotional disorders were diagnosed more often than pure conduct disorders. Some somatic symptoms were especially likely to feature as presenting complaints in disturbed children. Psychiatric disorder was associated with psychosocial disadvantage (e.g. broken homes or a family history of psychiatric disorder), and with high current levels of stress by the parents in relation to their children.

It is important to explore further the mechanisms which may underlie these consultations and the part primary care services could play in the recognition of childhood psychiatric disturbance and in interventions to alleviate the effects of this on the children and their families.

Anorexia nervosa in historical perspective

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Despite the topical importance and obscure aetiology of anorexia nervosa, systematic historical research has been very limited and the potential sources have never been evaluated critically. This study is based on an extensive search of a wide range of printed and manuscript material concerned with self-inflicted fasting and its effects, principally from Western Europe, from the 16th century onwards.

This information falls into three categories: (a) accounts of spiritual fasting and asceticism, which range from mediaeval saints to 19th century recipients of stigmata, whose resemblance to modern anorectics is notable; (b) descriptions of famous abstainers, freaks, and exhibitionists, including proven imposters and a small number of male 'living skeletons'. Such instances maintained a high level of public interest in fasting, and incredulity about its veracity, (c) a wide variety of medical cases and clinical material. Numerous descriptions of food refusal and abstinence, abnormal appetites and bulimia, mainly in women and associated with amenorrhoea, have been traced from the 16th century to the present. Virgin's disease (chlorosis), and the 'classic' descriptions of anorexia nervosa by Gull and Lasèque in the 1870s are set in historical perspective. A survey of 19th century hospital archives allows an analysis to be made of clinical disorders resembling anorexia nervosa. Brief reference is made to the re-emergence of anorexia nervosa as a psychogenic disorder in the 1940s, after diagnostic confusion with pituitary insufficiency.

Abnormal eating and very low body weights have attracted public curiosity and engrossed the interest of medical men for generations. The cumulative evidence suggests that modern anorexia nervosa is the current variant of a psychopathological entity which has spanned centuries. The central feature remains self-inflicted starvation in young women, but the syndrome has taken different forms, and its interpretation has altered, influenced by contemporary socio-cultural factors and developing medical knowledge. Such morphological changes have implications for present-day clinical practice. Fashionable aetiological ideas and therapeutic approaches need to be re-appraised in the light of these findings, whose relevance therefore exceeds mere antiquarian interest.

Psychopharmacology

Autonomic pharmacology in anxiety

Drs E. Szabadi and C. M. Bradshaw, Department of Psychiatry, University of Manchester

Although it is well recognised that anxiety is accompanied by increased sympathetic activity,¹ it is not known how this would affect the pharmacological responsiveness of sympathetically innervated tissues. We have studied the responsiveness of eccrine sweat glands, a tissue innervated by cholinergic fibres, to intradermally injected carbachol. A plastic paint impression method was used to count the number of active sweat glands.² In Experiment 1, the pharmacological properties of sweat glands were established: carbachol evokes a dose-dependent activation which can be antagonized by atropine.³ In Experiment 2, responses to carbachol were compared in a group of anxious patients and a group of sex and age-matched controls: in the anxious patients, more sweat glands, could be activated than in the controls.^{4,5} In Experiment 3, the effects of 'stressors' (heat, mental arithmetic) were examined in healthy volunteers; the stressors resulted in the same change as anxiety.⁶ In Experiment 4, the effect of diazepam (10 mg) on heat-evoked sweat gland hyper-responsiveness was examined; it prevented the development of hyper-responsiveness.⁷

These results are consistent with the hypothesis that the sensitivity of sweat glands to muscarinic receptor stimulants, such as carbachol, is modulated by sympathetic activity. The increased sympathetic discharge characteristic of anxiety states would sensitise the sweat glands to the agonist, and this response can be prevented by benzodiazepines, drugs known to reduce sympathetic output.

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Role of serotonin in punishment, depression and anxiety

Dr J. F. W. Deakin, Department of Psychiatry, University of Manchester

For many years abnormalities in brain 5-hydroxytryptophan (5HT) neurotransmission have been implicated in the

aetiology of anxiety and depression and in the mechanism of action of anxiolytic and antidepressant drugs. However, the precise role of 5HT remains controversial. Animal experiments suggest that 5HT neurones mediate effects of punishment on behaviour. These theories have generally failed to account for the various behavioural effects of punishment or to incorporate roles for different 5HT sub-systems. In response to these deficiencies it was proposed that: (a) spinal 5HT projections mediate unconditioned escape, withdrawal and analgesic responses to noxious stimuli; (b) 5HT projections from the dorsal raphe nucleus (DRN) to basal ganglia and amygdala function as a negative incentive system to guide the organism away from conditioned aversive stimuli; and (c) 5HT projections from the median raphe nucleus (MRN) to hippocampus mediate cessation of behaviour when expected rewards fail to materialise. More recently evidence has emerged for the existence of 5HT receptor subtypes. 5HT₂ receptors may be associated with DRN, and 5HT_{1A} with MRN functions. This theory predicts that anxiety and depression result from excessive operation of 5HT punishment systems.

To test this theory, a neuroendocrine index of 5HT neurotransmission has been evaluated in normal subjects and in depressed patients. In normal subjects infusion of the 5HT precursor tryptophan induced increased in growth hormone (GH) secretion which appear to be mediated by 5HT₁ receptors. In depressed subjects GH responses to tryptophan were markedly attenuated. These results suggest an association between reduced 5HT₁ neurotransmission and depression. Implication for 5HT-punishment theories are discussed.

Monoamine mechanisms in animal models of anxiety and depression

Professor J. A. Gray, Institute of Psychiatry, London

There is good evidence from studies of analogues of anxiety in a variety of animal species that this emotion is in part mediated by increased activity in central noradrenergic and serotonergic pathways, with a more limited activation of certain dopaminergic pathways. A particularly important role appears to be played by ascending and descending projections from the noradrenergic nucleus, the locus coeruleus, in the brain stem. Studies of the neurochemical mechanisms that mediate behaviour in animal models of depression are much more equivocal. They leave open two diametrically opposed views of the neurochemical basis of depression: (a) that this state reflects decreased monoaminergic (and especially noradrenergic) function; and (b) that it reflects increased monoaminergic function. It is possible that both views are correct, but in relation either to events in different regions of the brain or to different kinds of depression. According to the view that depression reflects decreased monoaminergic functioning, this state is at least to some extent the polar opposite of anxiety, according to the converse view, it is closely similar to anxiety. There are insufficient data to resolve these issues fully, but possible solutions are discussed.

The value of pharmacokinetic studies of neuroleptics in clinical practice

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Depot neuroleptics have been in clinical use for almost 20

years, during which time their clinical efficacy has been convincingly demonstrated. Pharmacokinetics of neuroleptics have been extensively studied, but the depot drugs, until recently, had not received as much attention. This paper reports on some recent studies conducted in Salford on the kinetics of depot neuroleptics. The three main areas discussed are (a) the blood level profiles of depot neuroleptics; (b) skeletal muscle factors affecting blood levels of these drugs; and (c) interaction with other psychotropic agents commonly used in psychiatry.

The findings suggest that blood level profiles of depot neuroleptics are not identical, and this may have clinical implications. Typical profiles of fluphenazine decanoate showed an initial peaking of blood levels within 12 hours of injection—a phenomenon not observed with flupenthixol. This observation may correlate with the reported occurrence of side-effects with this drug during this period. Withdrawal studies showed that these agents have a very slow elimination rate, which may be important in the timing of occurrence of relapses after withdrawal and the duration of side-effects when these develop.

The depot neuroleptics are injected into skeletal muscles, and local factors may alter the kinetics of these drugs. Three such factors were studied in a series of experiments. The data suggested that, contrary to the findings with aqueous injections, skeletal muscle activity and massage of the site of injection did not significantly affect blood levels. Nor did changing the site of injection have any effect on the blood level profiles of fluphenazine decanoate.

Finally, the study of concomitant administration of procyclidine and fluphenazine produced data suggesting a significant interaction between these two drugs. The significance of this cannot be underestimated in view of the fact that they are frequently prescribed together for the treatment of schizophrenia.

The overall relevance of pharmacokinetics studies to clinical use of depot neuroleptics is discussed.

Alcoholism: A doctor is required

Professor Neil Kessel, Department of Psychiatry, University of Manchester

Arguments are rehearsed why medical men must take a part in the assessment and treatment of alcoholics and not abdicate their necessary medical role to other professionals or to Alcoholics Anonymous.

Dimensions of neurosis

Professor David Goldberg, Department of Psychiatry, University of Manchester

A brief introduction is given to latent trait analysis, as a way of examining relationships, both between symptoms and between various kinds of research diagnosis. It is argued that two dimensions are sufficient to account for the relationships between common symptoms that occur in patients seeking help from primary care physicians. These dimensions are anxiety and depression, and they are shown to be highly correlated with one another. The diagnosis of 'depression' by general practitioners is shown to have too high a threshold on the depressive dimension, and to be unsatisfactory in another important respect as well.

Liaison with other disciplines

Psychological aspects of paediatrics

Professor David C. Taylor, University of Manchester and Professor R. D. H. Boyd, Royal Manchester Children's Hospital

The Professors of Child and Adolescent Psychiatry and of Child Health ran weekly clinical seminars for two separate groups of students. The students were in their fourth year, during a nine week resident paediatric term. The usual structure was of one or two clinical case presentations by students. Common problems in paediatrics were chosen, and the discussion amplified by cross-questioning of all the students to elicit and promote attention to the relevance to psychosocial factors. The sessions provided recurrent opportunities to orientate students towards the emotional, developmental and psychological aspects of their cases. In addition, the relevance of child psychiatry to child health was manifestly acknowledged, and for us the rounds provided an insight into the development of clinical scales and attitudes in students. These themes will be discussed, as will student understanding of: history taking; normal child development; attachment, threat and loss; and parenting and family structure.

Routine psychological screening of medical admissions by medical students

Professor J. M. Evanson, University of Manchester and Dr G. McGrath, University of Manchester

The large majority of patients with psychiatric illnesses present to, and are treated by, physicians. This study investigated whether such illnesses can be detected by a simple screening procedure administered by non-psychiatrists—in this case medical students. All patients admitted to the Professional Medical Unit of the University Hospital of South Manchester were screened for psychiatric symptoms using the 28-item version of the General Health Questionnaire, administered by medical students attached to the Department of Psychiatry. Patients with high scores were then interviewed by a medical student, using a semi-structured brief interview designed to elicit symptoms of minor psychiatric illness and relevant related social factors. The patient's responses also allowed psychiatric diagnoses to be made using operationalised criteria. Patients who had been interviewed were then seen by a staff psychiatrist on a regular teaching round held on the medical wards.

There was minimal disruption to the routine of the medical ward, and the regular contact with psychiatrists was welcomed by staff. Over one-third of patients were found to have a diagnosable psychiatric illness. The medical students' assessments were in broad agreement with those of the staff psychiatrist, although agreement was much better for the presence or absence of illness than for the strict diagnostic category. An additional benefit was the detection of a considerable amount of previously unrecognised, potentially treatable psychiatric illness in medical patients. The interview schedule could be improved by the addition of screening questions for alcoholism. Overall the study showed that the presence of psychiatric morbidity can reliably be identified using a simple screening procedure requiring little or no training. Whether detection of such morbidity leads to improvement in health care is yet to be determined.

Alcohol

A community alcohol team: A model for the future

Dr E. Owens, Kingswood Clinic, Manchester

During the past 20 years there have been major changes in treatment philosophies with increased recognition of the psychosocial aspects of problem drinking. Initially, treatment was thought to be best carried out in specialist units. Now it is recognised that, in the course of their work, primary care agents frequently come into contact with problem drinkers. Thus community agents could have an important role, not only with early identification and referral but also in carrying out treatment. Surveys, however, indicate that these agents have been slow to develop this role and that they are not even recognising the contribution of alcohol to the problems of their patients. Reasons for this are examined.

To try to overcome these problems, Community Alcohol Teams (CATs) have been set up in many parts of the UK. They are multidisciplinary, varying in size, composition, methods, and aims, but most seek to enable the primary care workers themselves to identify and manage patients. Different models and some aspects of their functioning are described.

Research findings are reviewed. Changes in knowledge and attitudes of the primary care worker have been demonstrated. Changes in professional practice of these agents have not yet been systematically evaluated, nor has the impact of the CAT on patients in terms of outcome. With regard to the secondary services, the CAT should be seen as part of a comprehensive response, not as an alternative.

Preventing alcohol problems at the local level

Professor D. Robinson, The University of Hull

Over the past 20 years, as alcohol consumption in Britain has doubled and every indicator of alcohol-related harm has shown a substantial increase, there has also been an increase in professional and public awareness of alcohol problems and of the need to prevent them. In recent years too, the prevention of alcohol problems 'at the local level' has begun to be taken seriously. In this short presentation I briefly review some features of the prevention debate, emphasise the potential for a local prevention strategy, outline the structure of a new guide to local action, indicate the range of topics covered by it, and in conclusion stress the complementarity of local and national prevention activity.

Liberalisation of licensing law: A Scottish success?

Mr S. Allsop, Research Fellow/Lecturer, Alcohol Studies Centre, Paisley College of Technology, Scotland

The liberalisation of Scottish Licensing Law over 1976 and 1977 involved a number of changes. These included an 'extra hour', whereby licensed premises closed at 11 p.m. rather than 10 p.m., and the opening of public houses on Sunday. These two changes moved the previously restrictive Scottish legislation into practice similar to that of England and Wales. What little evidence there is suggests that these changes were perceived to be popular, successful and were associated with the same amount of alcohol being consumed over a longer time period. A further change allowed premises to open throughout the afternoon. This has proved more controversial.

DRUGS

It has recently been suggested that there has been little increase in alcohol consumption since 1976, and that there have been similarly small increases in indices of harm. This has been the basis of claims that the new laws are an unqualified success. Unfortunately, these claims take no account of the effects of the recession, nor of the increase in the real price of alcohol since 1980-81. It is apparent that these latter factors have had an impact on alcohol consumption, and indices of harm, not only in Scotland but also in England and Wales where no changes in licensing law occurred. Many indices of harm in Scotland have, in fact, remained as high as in 1976 or are higher. Any decreases that have occurred are explicable in terms of the practice of the health and judicial services. It is clear that alcohol-related harm in Scotland is still greater than in England and Wales. This evidence is not consistent with claims that Scottish drinking behaviour is now more civilised as a result of changes in the licensing law: no benefits of the liberalisation have been demonstrated.

Given the recession and other economic effects on levels of consumption in Scotland, along with evidence that those countries with the most liberal licensing laws and easily available alcohol have the highest consumption and alcohol problems, the stage appears set for a rapid increase in problems. The claims of success of the 'Scottish experiment' and the case for liberalisation of licensing laws in England and Wales are without foundation.

Drugs

The drug scene: then and now (1960s London versus 1980s province)

Dr J. H. P. Willis, Liverpool Drug Dependency Clinic

This anecdotal paper compares and contrasts some of the clinical, psychosocial, demographic and pharmacologic aspects of diamorphine-dependent patients seen in the 1960s as opposed to the 1980s. A Drug Dependence Clinic at St Giles' opened in 1967; the author was Consultant Psychiatrist to that clinic until 1976. The demographic, clinical and psychosocial data of that clinic population had been described in the *British Journal of Addiction* (1978), 227 self-injectors of diamorphine were followed up from 1967 to November 1975. The patient population was noted to have a tendency towards a relatively long history of drug usage, e.g. 22% had been self-injectors for 2-5 years, 26% from 6-10 years and 10% for 10 or more years. The incidence for such amphetamine disorders as amphetamine-induced psychoses, hepatitis, pneumonia and septicaemia was high. 33% of the patients had had hospital admission for amphetamine-induced psychoses, 59% had had clinical hepatitis, 11% pneumonia, and 26% septicaemia.

The psychiatric background of the patients contained a high incidence of personality disorder; this was clearly diagnosable in 46%, and an additional 20% were clearly sociopathic, with persistent truancy, delinquency, etc., and there was a 13% death rate (the majority from barbiturate overdose). In other words, these were a group of extremely disturbed individuals in very poor physical shape.

After an absence of nine years from the UK the author returned to England to work in the Drug Dependency Clinic in Liverpool. Here the picture had changed dramatically. 95% of the diamorphine-dependent patients smoke heroin rather than inject it, although there is a hard core of

'old-fashioned' self-injectors—relics from the 1960s, the majority of whom are disturbed, unstable individuals who have never been able to contain control or manage their drug usage. The typical 'new' diamorphine user is aged between 19 and 21, smokes diamorphine by inhalation and shows a moderate pattern of dependency of no great severity—as evidenced by the fact that on admission to hospital such individuals display minimal withdrawal symptoms, require little or nothing in the way of medication, and achieve a normal sleep rhythm within one week.

At an impressionistic level they come across as perfectly normal youngsters who have acquired the heroin smoking habit and consequent dependence from peer group pressure or boredom. They do not have major psychological problems, and it is questionable whether they need psychiatric treatment. Their general state of physical health is greatly superior to that of their predecessors, their nutritional state is good, and they are well dressed and tidy in appearance and as far removed from the old-fashioned stereotype of the 'street junkie' as is possible to believe. Their presence in the community raises serious unanswered questions as to future management.

Out-patient treatment for heroin misusers; outcome with and without parental involvement

Dr P. K. Menon, Arrowe Park Hospital, Merseyside

Fifty patients who had reported physical dependence on heroin and had been treated as out-patients at a regional drug dependence service were traced between 6 and 12 months after entering treatment. All had received out-patient detoxification, by a 14-day methadone withdrawal regime, and counselling. The first 25 subjects had been counselled individually, the remainder with their parents. Forty-three patients were interviewed (23 without and 20 with parental involvement in therapy). Nine subjects interviewed outside prison reported abstinence, which was confirmed by urine testing. Parental involvement made no difference to outcome in respect to abstinence and other changes in heroin usage, or to criminal activity. Heroin smokers were no more likely than injectors to abstain or decrease heroin use. It is likely that abstinence and decreased heroin consumption were spontaneous changes unrelated to treatment.

Psychotherapy

The process of outcome in prescriptive versus exploratory psychotherapy: new findings from the Sheffield Psychotherapy Project

Dr D. A. Shapiro, MRC/ESRC Social and Applied Psychology Unit, University of Sheffield

The Sheffield Psychotherapy Project is a process-outcome study in which 40 professional and managerial workers with depression or anxiety received eight sessions of each of two treatments in a crossover design, with each patient seeing the same therapist throughout. As previously reported, outcomes were somewhat superior for prescriptive (cognitive/behavioural) than for exploratory (Hobson's conversational model) therapy, in a context of substantial overall clinical improvement; outcome was unaffected by the order in which the two treatments were undertaken. This paper presents our efforts to link process to outcome, and thus to identify the effective ingredients of the two treatments. Data

are presented which. (a) compare the trends in target problems rated three times weekly by patients throughout both types of treatment; (b) examine individual differences among patients and therapists as moderators of the outcomes of the two treatments; (c) detail verbal process differences in both client and therapist behaviours in the two treatments and relate these to session impacts; and (d) compare the helpful and hindering impacts reported by patients in the two treatments using a new and efficient method of interpersonal process recall

Substance abuse in the community

Professor Lee N Robins, Washington University School of Medicine, USA

After a long history of epidemiologic investigations of alcohol and illicit drugs limited to assessing patterns of use, the frequency and patterns of dependence and abuse are now being assessed. One such study is the Epidemiologic Catchment Area project, carried out in five sites in the USA. This set of surveys covers both household and institutional populations. It asks about history of problems resulting from substance ingestion over the lifetime, making it possible to study precursors of first involvement with substances and predictors that early problems will continue.

Many of the findings confirm results of much smaller and less representative follow-up studies: conduct problems in childhood and parental substance abuse are powerful predictors; substance use beginning early in adolescence has a much more serious prognosis than does later onsets. But unlike follow-up studies, community-wide surveys allow comparison of experiences of different age cohorts. Disturbing evidence for rising rates is found, particularly among women, for disorders that are already extremely common.

Because the samples are so large, it is possible for the first time to describe groups too rare to be studied in most general population samples. Thus we have adequate samples of cocaine and heroin users, and describe their demographic characteristics and treatment experiences.

Teaching interview methods

Teaching psychotherapeutic interviewing to trainees in peripheral hospitals

Dr G. P. Maguire, University of Manchester

A teaching package of three videotapes with accompanying written material was developed to provide introductory teaching in a method of psychotherapy. Twelve trainees from non-teaching hospitals were compared with 12 teaching hospital trainees previously studied. The research showed that both groups of trainees showed significant changes in interview behaviour both in style and content after this brief teaching introduction.

A further comparison within the group from non-teaching hospitals between trainees taught by teachers who were experienced or inexperienced in using these structured videotapes showed that there were no significant differences attributable to the trainer's experience.

It is concluded that the teaching package is an economic and effective way of introducing basic psychotherapy skills to all psychiatry trainees before they receive clinical supervision. However, the key role of the psychiatric clinical tutor in developing this training in practice is emphasised.

Traps, dilemmas and snags—Focusing in brief psychotherapy *Dr A. Ryle, St Thomas' Hospital, London*

Behavioural, cognitive and psychoanalytic methods of time-limited psychotherapy have all been shown to be effective. A cognitive model is proposed within which methods from the different sources can be combined. The main specific feature of the approach is in the method of reformulating the patient's problems. Therapist and patient together elaborate during early sessions a list of distinct, accurate descriptions of the repeated mental and behavioural sequences underlying the patient's problems. These target problem procedures are the basis for defining the aim of therapy. Cognitive and behavioural tasks and transference work are related to these formulations, and patients have a specific homework assignment of learning to recognise and modify these procedures as they occur. The balance of different methods tried can vary according to therapist and patient preferences and capacities. The method has been applied successfully to an NHS out-patient population.

Problem-orientated interviewing in general practice

Dr Linda Gask, Withington Hospital, Manchester

The problem-orientated approach to interviewing is briefly described on video tape. It is shown that the interviewing skills of general practice trainees can be significantly changed as a result of training in this model, and that this also results in an increased accuracy in detecting psychological illness. These findings strongly suggest that such teaching can be effectively carried out with GP trainees in a group setting.

Care of new long-stay patients

Evaluation of a hostel ward in Manchester

Dr Keith Bridges, Manchester Royal Infirmary

A controlled cost-benefit evaluation of Douglas House is described, and preliminary results referring to the first two years since it opened presented. Although it has been found unsuitable for some patients, the results suggest that the care of chronic psychotic patients is better in the hostel ward and is cheaper than providing care for similar patients in a psychiatric unit of a district general hospital.

Day care of the new long-stay

Dr Digby Tantam, University of Manchester

Psychiatric day hospitals have an important place in future planning of district mental illness services, and there have been several recent re-assessments of their value. Psychiatric day hospitals have historically served two main purposes: for the treatment of mainly neurotic patients who do not require hospital admission, and for the after-care of mainly psychotic patients who have had in-patient treatment.

The Withington Day Hospital is an 80 place DGH which notionally caters for both types of problem. A census by Dr Graeme McGrath and myself demonstrated, however, that the day hospital was dominated by the after-care function and that many of the new long-stay or the potentially long-stay were day and not in-patients. A rehabilitation service was instituted for these patients, and has been evaluated by Dr McGrath and myself. The results of the evaluation show: very small gains in the patients who remain in the day hospital; that a greater proportion of

patients were discharged than were discharged from a group of matched controls; that short-term in-patient admission was more common in both discharged and non-discharged rehabilitation patients; and that discharge had not resulted in symptomatic or social deterioration at the end of the follow-up period.

It is suggested that the results of this study throw doubt on the value of day hospital attendance for many long-stay day patients. It is argued that day-hospital attendance with the negative goal of preventing deterioration is particularly likely to lead to dependency on the day hospital, and to the augmentation of the new long-stay population in day hospitals. Day treatment may be more successful if it is only to be continued for as long as it produces an improvement in the patient's condition.

A survey of long-term users of psychiatric beds in City and Hackney Health District

Dr Cathie O'Druscoll, Team for the Assessment of Psychiatric Services (TAPS), Friern Hospital, London

City and Hackney Health District has been providing locally-based psychiatric services without mental hospital back-up for over 10 years. The district has acute wards and psychogeriatric assessment wards but no long-stay beds except for ESMI.

Over the past few years, a wide range of accommodation has been set up in the district by the Community Psychiatry Research Unit for chronically disabled patients. Despite these efforts patients were remaining in hospital wards for more than six months, and this survey was commissioned to look at the characteristics of this group. Thirty such patients were identified. The findings are described and the survey recommendations outlined. The survey found that only 20% of the patients were felt to need hospital care on the census date. The others fell into three groups: the elderly psychologically infirm, those with organic brain damage, and resistant or multiply-handicapped functional psychotics. Suitable provision for these patients is currently being planned within the district, and the survey will be updated at regular intervals.

Psychiatry of the puerperium

Maternal depression in the year following childbirth and mother-child interaction

Dr Alan Stein, Warneford Hospital, Oxford

Possible associations between postnatal depression and mother-child interaction were investigated by assessing two groups of mothers and their 19-month old babies. An index group of 49 mothers had experienced emotional disorders (46 depressive and 3 anxious) in the year following childbirth, while a control group of 49 individually matched mothers had been free from psychiatric symptoms in the same period. When the babies were 19 months old, all mothers were re-examined psychiatrically. A week later, mothers and children were assessed at home in three ways: observation of mother and child while carrying out standardised tasks; developmental assessment of the child; and a semi-structured interview with the mother. The assessors of the mother and child were blind to the findings of all the mothers' mental state examinations.

A number of differences between the two groups were found. For example, index mothers scored lower on

measures of positive contribution to their children's activity ('facilitation'), while their children showed less 'affective sharing' (smiling and showing toys to mother, or smiling and vocalising during a joint play). When currently depressed mothers were excluded from the analysis some of these differences persisted, although less strongly.

Within the index group chronic social difficulties appeared to be related to some aspects of mother-child interaction. For example, mothers who had experienced chronic social difficulties showed less warmth and facilitation than those who had not experienced such difficulties, while their children showed less affective sharing.

Maternal mental illness: Behavioural analysis of mother-infant interaction

Dr R. Kumar and Miss Carol Gambles, Institute of Psychiatry, London and Dr E. Melhuish, Thomas Coram Unit, London

Childbearing is associated with a sharp rise in the incidence of psychiatric disorder in women; between one and two out of every thousand recently delivered mothers require admission to psychiatric hospital. Most such women are found to be suffering from post-partum psychotic illnesses, although some mothers with postnatal depressive neurotic disorders are also admitted,¹ possibly because of additional social and interpersonal problems and severe difficulties in coping with the demands of a new baby. In Britain, separation of mother and child is usually avoided by joint admission either into a specialist mother and baby unit or into a general psychiatric ward with improvised facilities.^{2,3} There have been very few systematic studies of mother-infant interactions in such clinical settings in which both the maternal mental illness and the milieu may exert important short, and possible long-term influences on the developing infant and on the relationship between mother and child.

Margison⁴ has described some of the pioneering work which was carried out at the Manchester Mother and Baby Unit in which the clinical management of mothers was considerably facilitated by detailed regular observations by nurses of mothers and babies together. The preliminary research which is described here is an attempt to further systematise clinical observations of mentally ill mothers and their infants. The main aims of this research have been:

- (a) To discern and describe different patterns of interaction and to examine possible correlations with maternal psychopathology.¹
- (b) To make more informed management decisions e.g. concerning regulation of access and the prediction of risk to the child.
- (c) To establish a data base upon which to draw in future studies of the psychological development of children who were admitted as infants together with their mothers, to psychiatric hospitals.
- (d) To try to test whether joint admission to an institution of itself carries disadvantages for the developing child that outweigh possible benefits.

A rating scale has been developed at the Bethlem Royal Hospital which attempts to quantify perceptions by staff of selected aspects of mother-infant interactions. Preliminary results are described and illustrated by a series of case reports.

¹MELTZER, E. S. & KUMAR, R. (1985) Puerperal mental illness, clinical features and classification: A study of 142 mother and baby admissions. *British Journal of Psychiatry*, 147, 647-654.

- ²MARGISON, F. & BROCKINGTON, I. F. (1982) Psychiatric mother and baby units. In *Motherhood and Mental Illness*, pp 223-238 (eds I. F. Brockington & R. Kumar) London: Academic Press
- ³KUMAR, R., MELTZER, E. S., HEPPLEWHITE, R. & STEVENSON, A. D. (1986) 'Mother and baby' admissions to psychiatric hospitals in the South East Thames region: a survey of facilities. *Bulletin of the Royal College of Psychiatrists*, 10, 169-172.
- ⁴MARGISON, F. (1982) The pathology of the mother-child relationship. In *Motherhood and Mental Illness* pp 191-222 (Eds I. F. Brockington & R. Kumar) London: Academic Press

Long-term consequences of post-natal depression on child development

Dr H. L. Caplan, Consultant Child and Adolescent Psychiatrist, University College Hospital, London

Ninety-four women and their first-born children took part in a longitudinal study of maternal mental health during pregnancy and after delivery. The children's cognitive functioning and behaviour were assessed using respectively the McCarthy Scales and the Behaviour Screening Questionnaire, but without knowledge of the mothers' psychiatric history or current health. Significant intellectual deficits, averaging about 10 points, were found in the four-old children of the women who had suffered with post-natal depression. Girls performed slightly better than boys and children from middle-class and professional families did better than children from working-class homes, as did children whose mothers had achieved at least one 'A' level in their high school education. The presence of maternal depression correlated with lower cognitive ability but only when the depression occurred in the first year of the child's life. Behavioural disturbance in the four year-old children correlated with concurrent maternal depression but not significantly with maternal depression in the first year post-natally. The presence of marital conflict and a history of paternal psychiatric problems were independently linked with both lower cognitive test scores and behavioural disturbance and these were the only factors which contributed by interaction to the deleterious effect of maternal post-natal depression.

The effects of parents: What can non-human primates teach us?

Dr M. J. A. Simpson, MRC Unit on the Development and Interaction of Behaviour, Cambridge

The study of monkeys, apes and other animals can offer us principles to consider and test in clinical scientific work with people. Experimental work with rhesus monkeys has yielded some clear-cut and provocative findings. Thus a rhesus monkey reared by her mother out of sight of others is unlikely to accept her own baby easily, unless she sees other mothers and babies sometime in the first 2½ years. Such non-tactual perception can then be enough to engender an accepting attitude which allows her to learn from her own infant. A video-tape of a rhesus monkey giving birth to her first infant illustrates how mother and infant can co-operate.

Studies of human mother-infant interaction are probably now more sophisticated than most studies of non-human primates. The view that very young human infants can contribute substantially to intricate and well-meshed interactions with their mothers was present in work by Middlemore in the 1930s and Klein in the 1950s, before the rise of ethology and primate studies. However, the evidence is only now becoming compelling. For example, Murray and

Trevarthen arranged for mothers and infants to communicate face to face through a video set-up, and could replace the mother's 'live' contribution with part of one of her earlier sequences. When faced with a no longer responsive sequence the infants showed detached unhappy puzzlement. When the mother put on a blank face, the infants worked to reinstate a mutually responsive interaction. The study suggests that the infants used expressions of feeling to assist the mother in establishing mutual awareness, and it acknowledges the debt of infant studies to the ethological traditions of studying expressive communication. Similarly, Bowlby's views of the instinctive bases of attachment system grew out of his discussions with Hinde (a bird ethologist before a primatologist), as well as his psychoanalytic background.

If animal studies have contributed something to the study of loving co-operative exchanges, they also contribute an evolutionary view of individual selfishness, and of preferential treatment to others in proportion to how closely they are related, and/or likely to reciprocate. Infanticide, especially towards unrelated infants of potential mates, and conflict between parents and infants over how much is to be invested in the infant through weaning, are no longer viewed as surprising or necessarily pathological. If we expect weaning infants to conflict with their parents, we should also expect earlier development to lay suitable foundations for coping with conflict, and special weaning problems for those whose parenting was not good enough.

We are looking at the individual development of rhesus monkeys living in captive groups of two to five mothers and their young, and a single adult male. To what extent does an individual develop special ways of coping with its particular social situation, and how is such development guided? For example, status differences between mothers are clearcut and consistent, and result in different maternal and social environments for their infants. Subordinate infants are more often harassed and chased, and have to be much more circumspect in how they move toward favoured companions or foods. By the eighth month when tested with their mother as the only adult present, subordinate infants are more likely to persist in foraging or exploring out of their mothers' reach in situations with novel features which elicit fear and cautious approach in most infants. Such enterprising behaviour is also seen in subordinate jackdaws and reef fishes, and is adaptive to a life in which the opportunity to get near a potentially attractive object may last only while the setting is novel enough to cause others to hang back. If nothing is ventured by one subordinate, something may be gained by another. The dominant infant, in contrast, needs venture forth only when the subordinate has proved the situation safe, and can then easily gain control of whatever goods remain. While subordinates may become enterprising because they often have opportunities to take risks, there may also be constitutional differences in enterprise, and in the degree to which early mother-infant interaction encourages its development.

Draft Code of Practice

Mental Health Act 1983: Draft Code of Practice

Dr J. R. Hamilton, Broadmoor Hospital, Berks

The paper discusses the background to the Code of Practice, originating as it did in the consent to treatment

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provisions of the Mental Health (Amendment) Bill. The remit of the Code of Practice was changed whilst the Bill went through Parliament to provide guidance on the treatment of all patients, in hospital and elsewhere, suffering from mental disorder. The purpose of a Code of Practice is to provide guidelines on practice which Acts of Parliament and Regulations cannot do. The legal status of the Code is discussed together with the possible consequences of non-compliance. The question is raised as to whether the Mental Health Act Commission has overstepped its remit by including certain matters in the Draft Code such as non-clinical research. Considerable confusion was added to views on consent to treatment last year when the Commission sent to Health Authorities a document on consent to treatment which again has a questionable status. Much of the Draft Code is legalistic and has an anti-medical bias; examples of this are given. No guidance whatsoever is given on the interpretation of the urgent treatment section of the consent to treatment provisions nor on who should be responsible for carrying out the recommendations of a Mental Health Review Tribunal when these are disputed by the responsible medical officer. No guidance is given on treatment in the community for patients on leave of absence. (Mention is made of the problems in treating those who have been conditionally discharged from hospital and are on guardianship and there is a suggestion for legislation to allow for an extension of the consent to treatment provisions to patients in the community: comment is made on the administrative problems surrounding such an extension and how to resolve the potential problems of non-compliance).

It is concluded that much consideration will have to be given to the Draft Code during this consultation stage and that the final Code should not include contentious matters on which there is no consensus. The document should be reduced drastically in size and should not repeat what is going in Acts or the DHSS's memoranda. The final Code should give reasonable and realistic guidance on ways to promote optimum care and at the same time recognise the importance of clinical judgement. Unless this is done there is a danger of the Code being seen as a mixture of patronising and unrealistic advice which will not attract the confidence or credibility of psychiatrists.

Who wears the chains now?

Dr C. J. Thomas, Leicester General Hospital

The Mental Health Commission have drafted a provisional Code of Practice which they wish to impose on the psychiatric and other related professions. The feelings of the vast majority of psychiatrists in current clinical practice is that the draft Code of Practice is totally unacceptable. Most of us had envisaged a code of practice clarifying certain legal aspects of the Mental Health Act (1983) that would make the use of the Act more easily understood. However, the Commission seem to have given themselves a much wider remit and it appears as if they see themselves as determining how psychiatry should be practised in all areas. In their biannual report they admit they wish their powers to be extended to informal as well as formal patients. One must assume that the Commission have the intention in the draft Code of Practice of obtaining ultimately a better service for the mentally ill; unfortunately, it seems as though they believe this should be done by putting excessive controls on the practice of psychiatry. This naive approach is likely to greatly hamper the therapeutic and caring aspects of

psychiatry and will therefore in all probability lead to a considerable deterioration in the quality of care provided for the mentally ill.

The whole concept of a Code of Practice being imposed upon a profession by an outside body with only limited knowledge and experience of the problems and requirements of professional practice is unacceptable. If a Code of Practice has to be drawn up then it should be in a drastically different form than the current draft Code and should be one that is largely produced by the psychiatric profession itself and is acceptable to the members of that profession.

Poster presentations

A survey of blood alcohol levels in self-poisoning cases

Drs R. Varadaraj and J. D. Mendonca, St Thomas, Ontario, Canada

One hundred and fifty-eight cases of overdose were studied for alcohol use following their admission to an accident and emergency department. Of these patients 41% had consumed alcohol and 29% had blood alcohol levels exceeding 17.4 mmol/L (80 mgm/100 ml). Impaired overdose patients were found to be significantly older than non-impaired patients. There was a tendency for higher blood alcohol levels to be more associated with weekends, than weekdays, this association being stronger for males. More female overdoses were impaired on weekdays. Blood alcohol levels of women arriving during 'risk' time (2200-0659) were significantly higher than those of women arriving at other times. These findings confirm that alcoholism assessment and treatment services need to be integrated with accident and emergency services.

A comparison of plasma beta endorphin and ACTH in depression and chronic pain

Drs R. J. Smith, K. H. Simpson and A. G. Oswald, Department of Psychiatry and Anaesthesia, University of Leeds

Depression and chronic pain are interrelated and patients with intractable pain often become depressed. Patients with depression show diminished sensitivity to experimental pain stimuli. Endogenous opioids may be involved in pain perception, however, their role in affective disorders is not clear. Measurement of CSF and plasma opioid activity has produced conflicting results.

Neuroendocrine tests have been used as diagnostic and prognostic indices of depressive illness. Many depressed patients show increased activity of the hypothalamic-pituitary-adrenal axis in response to dexamethasone, and it has been suggested that this may be regulated by endogenous opioids.

Twenty-two patients with chronic pain and 20 patients with primary depressive illness completed a battery of psychological rating scales, and venous blood was withdrawn for beta endorphin and ACTH measurement using radioimmunoassay. The two groups of patients showed subtle differences. The relationship between beta endorphin and ACTH was the most powerful discriminator.

A critical review of research into neurotic hypochondriasis

Dr G. Adams, Department of Psychiatry, Leicester Royal Infirmary, Leicester

This paper was presented in a slightly different form at the 15th European Conference on Psychosomatic Research in

London in 1984. It examines briefly both the pragmatic research which has been done on hypochondriasis and the theoretical bases which have been used in attempting to comprehend this difficult area. It proposes that more phenomenological research is needed and discusses ideas on how this could be organised.

The efficacy of oxypertine in tardive dyskinesia

Drs J. S. Bamrah, H. L. Freeman and S. D. Soni, Research Department, Prestwich Hospital, Manchester

Tardive Dyskinesia (TD) is characterised by abnormal involuntary movements which often involve the oro-facial musculature and sometimes the trunk also. The exact aetiology of the condition is not known although it is thought to be related to dopamine receptor hypersensitivity resulting from prolonged neuroleptic therapy.

Several pharmacological treatments have been tried in an attempt to alleviate the symptoms of TD with conflicting results. One approach would be to look at agents that do not block post synaptic receptors but act through depletion of dopamine by presynaptic mechanisms such as oxypertine. A six-month double-blind, placebo controlled study was undertaken in Salford to evaluate the efficacy of oxypertine in the control of symptoms in patients with established TD. The results of this long-term study were at variance with our previous findings. The data are presented together with its implications which, we feel, extend to all trials for the treatment of TD.

Hormonal responses to i.v. tryptophan in depression

Drs I. Pennell, and J. F. W. Deakin, Department of Psychiatry, University of Manchester

The role of 5HT in the aetiology of depression and in the mechanism of action of antidepressant drugs remains controversial. The repeated finding of reduced csf 5HIAA concentrations and the possible antidepressant efficacy of 5HT reuptake blockers suggest a deficiency of 5HT neuro-

transmission in depression. In contrast, the immediate or delayed 5HT receptor antagonist properties of many antidepressants suggests excessive 5HT neurotransmission may be associated with depression. To discriminate between these theories we have evaluated a neuroendocrine test of 5HT neurotransmission in normal subjects and depressed patients. In normal subjects tryptophan infusions (100 mg/kg) induced increases in growth hormone (GH) and prolactin (PRL) but not cortisol. These responses were not antagonised by high doses of ritanserin, a 5HT₂ receptor antagonist. This suggests that 5HT₁ receptors normally mediate GH and PRL responses to tryptophan.

Twelve patients meeting DSMIII criteria for major depressive disorder were given tryptophan infusions. Their responses were compared with those in nine healthy controls. In depressed patients GH responses were significantly attenuated. Prolactin responses were generally more variable than GH responses to tryptophan and differences in PRL between controls and depressed patients did not reach significance. The results suggest reduced 5HT₁ neurotransmission is associated with depression.

Modification in family therapy with Chinese families

Dr U. Freeston, Senior Specialist in Child Psychiatry, Canberra, Australia

A family approach is now standard practice with Western child psychiatry, but its techniques and attitudes are derived from work with Western families. In what way would these be altered when working with a culture different in traditions, values, patterns of communication and child rearing practices? Specific effects of these differences on family life and on the attitudes of the family to therapy are examined. An attempt is made to identify specific alterations in techniques for these differences. Areas include: economic, money, work, housing, etc., rural to urban—old attitudes and new needs; patterns of communications in the family; values—security versus freedom.

Annual Meeting, 1986

Boldrewood Conference Centre, The University of Southampton
(8–10 July 1986)

Seasonal affective disorders

Light treatment of normal volunteers: effects on mood performance and melatonin rhythms

Drs J. Arendt, Department of Biochemistry, University of Surrey, J. Broadway, Department of Biochemistry, University of Surrey and British Antarctic Survey, Cambridge and S. Folkard, MRC Perceptual and Cognitive Performance Unit, Laboratory of Experimental Psychology, University of Sussex

The treatment of seasonal affective disease (SAD) with bright light in a short, or long 'skeleton' photoperiod (daylength) derived from the observation that human night-time melatonin production is only suppressed by a light intensity > 2500 lux. We were concerned to establish the effects of skeleton photoperiod bright light in normal subjects as part of a study of human photoperiodism. The Antarctic (Halley base, 75°S, 25°W) provides an interesting environment for such studies both with regard to the natural, extreme changes in daylength and to the long period (three months) of winter darkness where the only available illumination is artificial (< 500 lux).

For six weeks following the winter solstice the artificial photoperiod was manipulated with bright or dim artificial light: two groups of five male subjects were exposed from 0800 to 0900 h and from 1930 to 2030 h to either bright (2500 lux, Vitalite, Durotest Corporation) or dim (< 500 lux) supplementary lighting. Plasma samples were taken from volunteers via indwelling venous cannulae at hourly intervals for 25 h at equinoxes, solstices and at the end of light treatment. The samples were assayed for melatonin by radioimmunoassay.

Subjects recorded mood (on 18 visual analogue scales), oral temperature, and performed logical reasoning, letter cancellation and pegboard transfer tests six times daily (0900, 1100, 1400, 1600, 1830, 2200 h) during the week before light treatment and during the last week of light treatment. No major differences were found in mood, temperature or performance during light treatment. Both season and light treatment influenced melatonin. Bright light in the winter induced a two-hour phase advance in the melatonin rhythm (overall analysis of variance pre-compared to during light treatment, $P < 0.00001$; statistical difference in phase position, $P = 0.0402$; dim light treatment: no effect). A similar phase advance was seen in summer compared to winter (overall analysis of variance, $P = 0.0247$).

We conclude that in this exceptional environment a clear physiological response—phase-shifting of the melatonin rhythm—is seen during bright-light treatment given as a 12.5 h skeleton photoperiod, with no major concomitant effects on mood, temperature or several performance variables. Seasonal changes in human melatonin production are probably dependent on photoperiod.

Seasonal affective disorder (SAD—Does it exist?)

Drs G. Isaacs and C. Thompson, Charing Cross & Westminster Medical School, London

SAD, originally described in 1984 by Rosenthal et al, is a condition of regularly recurring autumn-winter depressions, separated by spring and summer remissions. It occurs predominantly in women who complain of fatigue, hypersomnia, overeating, carbohydrate craving, weight-gain and sadness.

In the first part of the paper, the first British sample ($n = 51$) to be described were compared in terms of their clinical and demographic characteristics with American and Swiss groups. They were shown to be remarkably similar in terms of their atypical symptomatology despite this not having been part of the inclusion criteria. The main differences were a lower incidence of the Bipolar II diagnosis and less frequent family history of affective disorder in the British sample.

In the second half of the paper, an intervention study in 11 patients was described. The aims of this part of the study were to investigate whether the total amount of light or extension of the photoperiod was the important variable. To this end, three different treatment regimens with full spectrum lighting were used in each patient. The treatments were given in a balanced randomised order for one week, followed by a withdrawal week. They were photoperiod extension by four hours with either bright light at 2,500 lux or dim light at 300 lux, and four hours of incident light augmentation with bright light at 2,500 lux given within the photo-period so as not to alter its length.

Observer ratings of depressions by a blind rater, using the 17-item Hamilton Depression Rating Scale and a specially compiled atypical symptom rating scale, showed augmentation to be significantly better than photoperiod extension by bright or dim light. However, self rating, showed photoperiod extension with bright light was significantly better than the other two treatments. Thus the superiority of bright over dim light was replicated while the blind observer ratings suggested that photoperiod extension was not essential to the action of phototherapy.

Mood disorders, infradian rhythms and seasons

Professor M. R. Eastwood, Clarke Institute of Psychiatry, Toronto, Canada

Recently there has been a developing interest in the relationship between affective disorders and season. Epidemiological data from disparate centres suggest that affective disorders (and suicide) are seasonal in that they peak in the spring and fall. This finding excites interest since it implies that this type of illness is predictable in part—clearly significant for prevention. Furthermore, the finding may be linked with the current interest in changes of light, mood and melatonin. Our group has shown that the affective disorders and alcoholism (which may well be a reflection of mood disorder) are the only psychiatric diagnoses to show seasonality. Furthermore, the team has examined long mood cycles—*infradian* rhythms—to see whether they exist in those with and without major affective disorders. A lengthy study showed that, of the *infradian* rhythms, 90 day cycles were the most common and occurred in both cases and controls. The cycles differed only in amplitude. This

suggests that affective disorder, like many medical illnesses, is a pathological variant of a physiological variable.

The purpose of the presentation is to link seasonal mood variation with the patterning of the infradian cycles. A group of patients suffering from major affective disorder and a healthy comparison group rated their mood daily for approximately 14 months. The two groups exhibited very similar mood patterns with infradian rhythms synchronising with calendar seasons.

The effect of daylight saving time on the incidence of psychiatric presentations

Dr P. A. Bick and Ms A. L. Hannah, University of Dundee, Scotland

A specific seasonal affective disorder has recently been described. This disorder, typically a winter depression, may be caused by changes in the period of natural daylight (photoperiod) at different times of the year. These photoperiod changes are thought to cause disturbances in circadian rhythm homeostasis which lead to the expression of the seasonal mood change. This postulate is supported by evidence that seasonal depressions can be readily treated with a three to six hour exposure to artificial light and that resolution of the disordered mood is accompanied by resolution of phase disturbed circadian rhythms. We wondered if a sudden change in apparent length of daylight consequent to a one hour time shift (caused by the Daylight Saving Time change) might lead to a phase disturbance in circadian rhythms and an associated increased incidence of affective disorders for a short time following the change.

There have been no previous reports of Daylight Saving Time causing clinically significant changes in behaviour. The authors investigated the effect of five Daylight Saving Time changes on the incidence of psychiatric presentations before and after the time changes at a busy crisis centre. 1,521 patient contacts were reviewed by a rater who was blind to the dates of the records. There were significantly more patient contacts during the week following the time changes. This increase was due to a specific rise in depression and psychosis related presentations. A hypothesis is presented that Daylight Saving Time causes a short term disturbance of circadian rhythms, leading to the expression or exacerbation of psychiatric symptoms.

Influence of sunshine on the prevalence of mania

Dr P. A. Carney, Regional Hospital, Galway, Ireland

Monthly admission rates for patients with a diagnosis of mania to the Department of Psychiatry in Galway Regional Hospital were examined for the five year period 1980-1984. Monthly variation and admission rates were compared with monthly levels of sunshine. The number of admissions for each month over the five year period correlated significantly with the average total hours of sunshine for each month in the same time. It is suggested that the presentation of mania in this fashion is due to an abnormal response to light in patients with the disorder. The data also demonstrated a large increase in the number of admissions over the study period.

The effect of light on CNS noradrenaline and serotonin receptor function

Dr R. Mason, University of Nottingham

Seasonal affective disorder (SAD) is characterised by recurrent episodes of depression which occur annually

during the winter months, corresponding with the periods of shorter daylength and reduced light intensity. Interestingly, the use of bright artificial light is reported to be an effective treatment for SAD.^{1,2} A common result of antidepressant drug and ECT treatment in animal studies is altered 5-hydroxytryptamine (5-HT) and noradrenaline (NA) B-adrenoceptor sensitivity in the CNS.^{3,4} If light therapy is effective in depression then it might also be expected to induce similar changes in 5-HT and NA neurotransmission.

Pigmented male rats were kept on lighting cycles of either LD(12 hr:12 hr), LD(18 hr:6 hr) or under continuous light (LL) for 6-12 weeks. Five-barrel micropipettes were used for extracellular recording from the hippocampus and cerebellum and for the microiontophoretic application of 5-HT, NA and GABA. The sensitivity of single neurones to 5-HT, NA and GABA was evaluated by both the I.T50 and threshold ejection current methods.^{3,4} Both continuous light (LL) and LD(18:6) light exposure resulted in the development of a 2-3 fold supersensitivity to iontophoretic 5-HT (hippocampus) and 2-3 fold subsensitivity to NA (cerebellum) compared with LD(12:12) conditions. The response of neurones to iontophoretic GABA was similar in both LD and LL rats. Altered sensitivity to iontophoretic NA and 5-HT was not found following short periods (1-3 days) of exposure to light. Rats kept under LD(12:12) and treated daily with imipramine HCl (10 mg/kg, i.p., for 3 weeks) also showed a pronounced subsensitivity to iontophoretic NA and supersensitivity to 5-HT compared with saline treated controls. No effect on GABA sensitivity was found.

The similarity between the effects of light-induced altered neurotransmission in the CNS and those induced by antidepressant drugs on 5-HT and B-adrenoceptor function may provide a basis for the recent reports of the beneficial effects obtained with bright artificial light in the treatment of seasonal affective disorder. The light-induced effects may, in turn, be mediated via retinal projections through the suprachiasmatic nuclei (SCN), central structures for the regulation of the circadian and circannual rhythmicity.^{5,6}

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WHO and mental health research

Community response to alcohol problems: results from Scotland, Zambia and Mexico

Dr E. B. Rutson, Royal Edinburgh Hospital

The world is producing and consuming more and more alcohol. Total production of alcohol rose by almost 50% between 1965 and 1980. There is evidence that alcohol consumption has increased rapidly over the same period. Existing evidence shows a close relationship between total

consumption and the extent of harmful consequences. Increasing alcohol consumption is, therefore, a major health concern throughout much of the world. Most existing research on alcohol consumption is based on Western industrialised countries and when communities in Africa, Asia or Latin America have been the focus of a study the emphasis has usually been on an anthropological rather than an epidemiological perspective.

The research described here was a collaborative project involving the countries concerned and promoted by the World Health Organization. It aimed to describe the extent and nature of drinking patterns and alcohol related problems in a number of selected communities, alongside a study of that community's attitudes towards alcohol and their current response to alcohol related problems. The overall objective was to provide an information base within each community that would facilitate the fuller development of a more appropriate response to alcohol related problems. It was also envisaged that the experience within these communities would stimulate similar endeavours within the country concerned and within neighbouring countries.

Urban and rural communities were selected within countries of widely differing characteristics; Mexico, Scotland and Zambia. Epidemiological studies within each community revealed very different patterns of consumption in each of the countries involved, although the urban rural differences within countries were less than anticipated. Scotland proved to be a country of near universal alcohol consumption. Attitudes towards adult drinking were tolerant and two thirds of the men and the vast majority of women who had been drinkers reported no problems arising from their drinking in the past year. In Mexico 55% of the women were abstainers as were 15% of the men. In Zambia 32% of the men were abstainers and 69% of the women. In both these relatively abstinent countries attitudes towards drinking were much less tolerant, particularly amongst Mexican women, and the pattern of drinking was more episodic and intense in both countries. Among drinkers in Mexico and Zambia, many of whom consume much less than the Scots, there was a surprisingly high level of reported problems.

The research gave priority to obtaining data which were of principal value to the development of a community response to alcohol problems in the communities involved. There are, however, implications for cross national studies and one community may benefit from the lessons learned in another. The problems of designing and maintaining a project of this kind in widely differing settings are also discussed.

Incidence of schizophrenia: results of the first phase of an international study

Professor J. E. Cooper, University of Nottingham

The International Pilot Study on Schizophrenia (IPSS), conducted in eight Field Research Centres between 1966 and 1972, raised the possibility that patients with schizophrenia living in developing countries (Africa and India) had a more favourable two year and five year outcome than similar patients living in developed countries, (UK, USA, Denmark, USSR). The IPSS was not an epidemiologically based study, so this finding could not be regarded as definite, even though a similar finding was evident in several other studies. Another WHO collaborative international study was therefore undertaken, with a design that allows

more definite conclusions to be drawn. Most of the field research centres in the IPSS took part again, plus several new centres (11 in all). In each centre, attempts were made to identify and follow up for two years all patients presenting to the psychiatric services (and other helping agencies, defined for each centre), serving a defined catchment population, with a first ever psychiatric illness that might be schizophrenia. Screening was on a symptomatic basis, rather than relying on local diagnostic habits. Extensive social and personal data were collected for each patient, since the IPSS showed that the predictive power of social information was at least equal to that of clinical information about the illness itself.

The incidence rates and some of the clinical characteristics of the patients are presented for those centres for which rates can be calculated. They show a surprising similarity, in contrast to the wide geographical and cultural variations found in the incidence of most physical disorders. The relationship of the study (known as the 'WHO Collaborative Study on determinants of outcome of severe mental disorders') to other WHO studies on schizophrenia and social disability is also discussed.

WHO multicentric studies of treatment of selected mental disorders

Professor Max Hamilton, The University of Leeds

Three of these studies have already been published and the next will appear soon. Because of the difficulties and expense in organising such trials, they have to be very comprehensive and this makes analysis and interpretation anything but simple.

Schizophrenia. The study is well known. It found that, on the whole, with adequate definitions, it is easy to agree on the diagnosis of schizophrenia. The features are similar in different cultures.

Schizophrenia follow-up. The most important finding is that in the Third World, the outcome is very much better.

Anti-depressant dosages. The symptoms of primary depressions are remarkably alike across cultures. The higher dosage of drug is very slightly better than lower, but the difference was not significant statistically.

Sedatives and counselling in the treatment of 'somatized' anxiety. This complex study included analysis of the details of counselling and the assessment of life events. Drugs had little effect on this type of patient, but counselling was clearly beneficial.

Biological aspects of depression

Propranolol and depression—causative or coincidence?

Dr R. Manchanda, University of Western Ontario, Canada

Several reports suggest that propranolol commonly used in non-psychiatric patients may cause symptoms of depression and this may be dose dependent. On the other hand, other investigators have argued this relationship to be insignificant. The author reviews the available evidence from case reports, manufacturers' reports of adverse effects, data from hypertension clinics and the tricyclic anti-depressant use among patients on antihypertensives. The findings of propranolol-induced depression may be coincidental as the results are derived from uncontrolled and blind studies and hence are not very reliable. Some investigators have used high doses of propranolol for the treatment of anxiety, mania and schizophrenia. Change in

the ratings of depressive symptoms in controlled trials of schizophrenic patients using high doses of racemic (dl) and dextro(d) propranolol do not lend support to the claim that propranolol causes depression.

Stimulus seizure issues in electroconvulsive treatment

Professor R. McClelland and Drs M. Headley, S. Cooper, J. Lumsden and G. McAllister, Queen's University of Belfast

While electroconvulsive therapy (ECT) has been established as an effective treatment for depressive illness a number of questions remain regarding the optimal form of administration and patient selection. Underlying such questions is the need for a fuller understanding of the kinetics and mode of action of ECT and the development of reliable methods of patient monitoring. It is now generally accepted that the *grand mal* seizure is a necessary concomitant for the therapeutic effects. Several parameters of the induced seizures show considerable inter-subject and intra-subject variability, particularly duration and distribution. While it is less clear that these contribute significantly to outcome, present evidence suggests that local fits, unilateral fits, and fits which last only a few seconds are not as effective as generalised convulsions lasting more than 20 seconds. With the use of muscle relaxant drugs, behavioural observations are not reliable in detecting and quantifying the seizure events. Multichannel EEG monitoring gives a more certain and precise assessment of the occurrence, distribution, duration and characteristics of the induced fit and many would regard neurophysiological monitoring as mandatory in further scientific studies of ECT. In a recent series of double-blind studies we have been investigating the seizure characteristics and clinical efficacy of ECT. This has included a comparison of the Ectron Mk 4 Sine-wave constant voltage stimulus unit (CV) and the Ectron CCX constant current pulsed stimulus unit (CC). Preliminary findings from these studies suggest that the standard stimulus outputs of these two units do not significantly affect seizure duration. Small but significant delays were observed in the onset of bisynchronous seizure activity using CC ECT. Spectral analysis of the seizure events is presently being undertaken.

Major discrepancies were observed between the visually observed and EEG monitored seizures using both the CC and CV stimulus methods. Visual fits were significantly shorter than EEG seizures. Six of 82 (7%) of visually monitored seizures were reported as 'missed'. The corresponding EEG events confirmed the occurrence of seizures in all cases.

Tyramine conjugation deficit, a trait-marker for endogenous depression

Professor M. Sandler, Institute of Obstetrics and Gynaecology, University of London

A highly significant decrease in ability to metabolise an oral load of tyramine to its sulphate conjugate compared with controls and neurotic depressives was identified in patients with endogenous unipolar depressive illness. The biochemical deficit persists after clinical recovery and is present in about half the non-depressed first degree relatives of endogenously depressed probands. Thus it is likely that the abnormality is a trait-marker for depressive illness and may be useful in practice as a predictor of vulnerability to this disease. The tyramine test is superior to the dexamethasone suppression test in both sensitivity to, and specificity for,

endogenous depression. Patients who responded to tricyclic antidepressants had significantly lower pretreatment tyramine excretion values than non-responders.

Brief dynamic psychotherapy

Intensive short-term dynamic psychotherapy: trial-therapy model of initial interview as a selection criterion

Dr H. Davanloo, McGill University, Canada

Two major factors leading to the extreme difficulty and great length of dynamic psychotherapy are *resistance* and *transference*. In Davanloo's technique it is possible first to hasten the onset of these two phenomena and then to meet them head on and resolve them, in such a way as to gain direct access to the unconscious within a single interview.

The process begins with pressure towards the patient's avoided feelings. The patient responds with an increase in resistance, which takes the form of a series of defences. In contrast to traditional methods of dynamic psychotherapy, these defences are not *interpreted* but *challenged*. The result is a rapid rise in transference feelings which the patient wishes to avoid, resulting in a further intensification of resistance. The resistance, now in the transference, is challenged in the same way as before. Eventually, the defences become exhausted and the patient is able to experience his transference feelings directly and to acknowledge them to the therapist.

The effects are marked and dramatic. There is a major drop in tension and a mobilisation of the therapeutic alliance and the patient often makes a communication that throws light on central aspects of his neurosis.

At this point, and not before, the therapist begins to use the main intervention of traditional dynamic psychotherapy, namely interpretation. The residual resistance is systematically analysed by interpretation of the two triangles: defence, anxiety and hidden feeling (the 'triangle of conflict'), and current, transference, and past relationships (the 'triangle of person'). This leads eventually to a phase in which for the time being resistance is almost completely in abeyance and the therapist can achieve direct access to the unconscious without further reference to the transference. He is then able to bring the patient to the experience of some of the most painful or anxiety-laden feelings that lie behind his neurosis, which sets the whole therapeutic process in motion.

All this can be achieved in a single initial interview lasting from 1½ to 4 hours. As a result the patient's whole psychic system is permanently loosened; and any patient who responds in this way can be selected with confidence for intensive short-term dynamic psychotherapy in which exactly the same technique is used. Thus the response to this form of *trial therapy* constitutes the main selection criterion. The number of sessions required varies from 1 to 10 for simple symptomatic neuroses, through 10 to 20 for more complex cases, to 20 to 40 for character disorders of increasing severity and complexity.

Care of the chronic patient in the community

Family management of mental illness: Clinical, social and economic benefits

Dr I. R. H. Falloon, Buckingham Mental Health Service

The role of the family unit in the care of the chronically ill in the community is threefold, providing supportive

housing, providing informal nursing care, and providing stress management. The capacity of families to meet such extensive demands varies considerably, with very few able to perform such a role without substantial training coupled with long-term professional assistance. The family stress associated with the shift towards community management of all mental illness is often extreme and likely to reduce the long-term viability of this key community resource.

Current approaches to the family management of schizophrenia have focused on enhancing the efficiency of the family in the management of stress. This has included the everyday stresses encountered by the index patient and his or her family caregivers, family members' stresses independent of the index patient and stressful life events that impinge upon all family members. It is assumed that for any family to cope with a member who has developed a chronic or recurrent illness exceptional efficiency in problem resolution is needed.

The efficacy of one approach to family management that includes optimal medication, education about schizophrenia, behavioural family therapy and specific coping strategies is presented. Significant benefits have been found in terms of clinical, social and family morbidity. These benefits are discussed as well as the costs to the community of family care.

A case management programme for difficult-to-treat patients *Professor L. I. Stein, University of Wisconsin Medical School, USA*

There is general agreement that persons with chronic mental illness living in the community require 'case management'. This paper reviews the conceptual foundation for case management, describes its functions and discusses several case management models. One model discussed in greater detail is one that has been researched and found to be effective in working with difficult-to-treat patients. This patient population shares some or all of the following characteristics: an unwillingness to regularly come in for services, poor medication compliance, a need for structured daily activities, a poor ability to self-monitor and frequent crises. The special case management programme developed to serve these patients is staffed by a multidisciplinary team that operates two shifts per day, seven days a week. Much of their activity takes place out in the community rather than in the office. Patients are carefully evaluated and monitored, barriers to community tenure are identified and worked with. No time limit is established—patients are worked with for as long as they need the programme—this may be for a lifetime. Results clearly show a decrease in symptomatology, an increase in stability and a marked decrease in use of the hospital.

Long-term patients in the community. An Italian experience *Professor Michele Tansella, Istituto di psichiatria, Verona, Italy*

The Italian model of community psychiatry, as prescribed by the 1978 psychiatric reform, is described. Three distinctive features of this model are worthy of note. First, the phasing out of mental hospitals was intended to be a gradual process—by means of a block on first admissions and subsequently on all admissions—rather than an abrupt deinstitutionalisation of chronic patients. Secondly, the

new services were designed to be alternative rather than complementary or additional to mental hospitals. Thirdly, it is hospital psychiatry (which, with the passage of time, is increasingly located in general hospitals rather than in mental hospitals) which is considered complementary to community care, and not vice versa.

Evidence collected in areas where the reform has been correctly implemented indicates that the 'new' community services are able to cope with the problems presented by the whole range of psychiatric patients resident in the catchment area. Moreover case-register data from three of these areas (Pavia, Portogruaro and South Verona) show that most of the patients are treated outside the hospital only. The rates of long-stay patients (≥ 1 year in hospital) have been decreasing over the years in all three areas, while rates of 'new' long-stay are very low and there is no evidence that such patients are accumulating in any of the three areas. The South Verona experience will be described in some detail. South Verona is an area of 75,000 inhabitants where a comprehensive and well integrated community psychiatric service has been set up and organised according to the provisions of the new law. In the organisation of this service, effort has been directed towards ensuring therapeutic continuity, long-term support and care to patients and their families, as well as integration between different kinds of intervention (made by the same team). In South Verona while the number of long-stay patients is decreasing, the number of 'long-term users of psychiatric services' (those in contact with some form of psychiatric service—not necessarily the same one—for a year or more without a break of more than three months) is increasing. The results of a two-year follow-up study of a cohort of long-term patients is presented.

Schizophrenia and the quality of life

Professor P. R. A. May, University of California, USA

A limitation of our current concepts of treatment is that we have tended to focus on rather narrow concerns such as relief of symptoms and return to community living. The time has come to think of other things, and particularly of quality of life (QOL). As an offshoot of some other research, we came to consider QOL systematically. Life is more than doctors, pills and symptoms, more than psychotherapy or having your behaviour modified by the experts. It has many aspects. We each value them differently.

Originally we identified 16 main components of quality of life: (1) physical health; (2) mental health; (3) material resources; (4) education; (5) employment; (6) recreation; (7) daily services; (8) welfare services; (9) non-material: art, relationships; (10) knowledge/education; (11) safety and security; (12) beauty of nature, (13) inner QOL: spiritual, religious, contentment; (14) national esteem; (15) national defence; (16) peace.

Later we identified 12 main areas that seemed suitable for assessment of the QOL of schizophrenic patients: (1) housing; (2) material; (3) environment; (4) safety; (5) travel and communication; (6) physical and mental health; (7) work; (8) leisure; (9) education/knowledge; (10) relationships; (11) inner experience; (12) religion.

These were set out in a checklist from that could be filled in by an interviewer to portray the patient's QOL from either the patient's or the staff's or the family's view of it

This proved to be a useful tool for identifying areas of a patient's life that needed attention. In subsequent work, Malm and Skantze have rearranged the items as a questionnaire to be filled in by the patient. It is clear that many aspects of patients' QOL are not under the control of the health care system at all. We need to point out to other agencies that they too have some responsibilities for the patient's QOL and that we expect them to do something about it.

Aggression

Premorbid personality of depressive, bipolar and schizophrenic patients, with special reference to suicidal issues

Professors Jules Angst, Psychiatric University Hospital, Zurich, Switzerland and Paula J. Clayton, University of Minnesota, USA

The personalities of 6,315 Swiss conscripts of the army were assessed in 1971 by the Freiburg Personality Inventory (FPI), together with social attributes and consumption of tobacco, alcohol and drugs. New factor analyses of the FPI produced three scales, extraversion-introversion, autonomic lability and aggression. Up to 1983, 185 psychiatric patients were identified who were treated as in-patients or out-patients in the same geographic area. A sub-sample of 87 patients were chosen for a blind diagnostic study applying Feighner criteria to the records. Their premorbid personalities are compared to controls from the same group. The results are preliminary, the diagnostic groups are small.

The bipolar patients do not deviate significantly in any personality dimension from the controls. They differ significantly from the unipolar depressives by a lower aggression score. Unipolar depressives score high in aggression and in autonomic lability. The same is true for the socio-paths. Aggression scores are also high in those who committed suicide or died by accidents. Independent of the diagnostic classification, subjects with suicidal attempts score high in aggression, whereas subjects with suicidal ideation score low in aggression.

Biological aspects of suicide and aggression

Dr S. A. Montgomery, St Marys Hospital, London

There are many cultural factors which may affect suicide and suicidal behaviour: religion, war, economic factors—and the rates vary widely from country to country. In spite of this variation there has been a consistent demonstration of a relationship between a biochemical measure and suicidal behaviour. A number of centres in different countries have now reported a significant association between low levels of 5HIAA, the serotonin metabolite in the CSF, and a history of suicidal behaviour. In those studies that have reported HVA, a significant relationship between low levels and suicidal behaviour may also be seen. These studies were originally conducted on depressed patients. Subsequent studies on difficult US naval personnel indicate a similar relationship in personality disorders. In the centres that graded the violence of the suicidal act it would appear that violent suicidal acts appear more particularly associated with low 5HIAA and HVA. In the US naval personnel a history of aggression correlated with CSF 5HIAA. A review of intervention studies demonstrates the failure of general psychiatric follow-up, or social or psychotherapeutic

measures to lower the repeat rate. The significant reduction in suicidal behaviour seen on a low dose neuroleptic suggests that possible pharmacotherapy should be investigated more thoroughly.

Impulsivity and the serotonin system

Professor J. J. Lopez-Ibor Jr., University of Alcalá de Henares and Ramon y Cajal Hospital, Madrid, Spain

Biological research in affective disorders has evolved in recent times from the research of neurochemical substrates of the disease or of its clinical subtypes into the correlation of laboratory data and specific symptoms or behaviour patterns ('target symptoms'). The serotonin system has been shown to be involved in suicidal behaviour not only in affective disorders but in schizophrenia and personality disorders. Serotonin has also been claimed to be involved in aggressive behaviour, even in non-psychiatric cases, in bulimia and other disorders. It has been suggested that the common link of these and other symptom patterns is a lack of adequate control of impulsivity. But correlations between behaviour patterns that are not specific to a disorder or diagnostic category and that may be present in normal psychology as traits of individual coping styles, put forward very important methodological problems.

The psychoanalytic theory of aggression

Professor J. Sandler, University College, London

The notion of aggression is an important one in psychoanalysis, but there is no one psychoanalytic theory of aggression. Freud developed several theories of aggression during his psychoanalytic lifetime, and further psychoanalytic formulations have been made since. At present there are psychoanalysts who see aggression as a fundamental drive which parallels sexuality, and who postulate the existence of aggressive as well as sexual energies as motivating forces in mental functioning. Other psychoanalysts adhere to and amplify Freud's theory of the death drive. There are also those who reject the idea of a primary aggressive drive completely, and this group overlaps with those who do not accept the energy model in psychoanalysis but incline more towards a systems theory approach.

A short historical overview shows how very different Freud's theories of aggression were. These range from the idea of aggression as a 'self-preservative' drive to that of a primary death drive. There are a number of important distinctions which are relevant to the psychoanalytic approach to aggression. These include the distinction between aggressive behaviour and unconscious aggressive impulses. The view is put forward that emphasis should be placed on the consideration of the aggressive wish as opposed to the aggressive drive, as many different stimuli, both internal and external, can evoke an aggressive wish. The relation of the aggressive wish to effects such as anger has to be reconsidered, and wish-fulfilment needs to be looked at from a different point of view than that of energy discharged. The particular psychoanalytic formulations put forward in this presentation have important clinical implications, some of which are touched upon.

The symptom of irritability and related concepts

Dr C. M. Taylor, St James's University Hospital, Leeds

The symptom of irritability is related to the concepts of anger, hostility and aggression. When the relationship between irritability and the affects of anxiety and

depression is explored in neurotic patients, irritability behaves as if it were an independent variable. This suggests the possibility that it could represent a distinct and separate mood state. The different aspects of this state are explored and it is suggested that this could represent an altered state of autonomic reactivity. Some relevant literature is described and the nosological status of irritability is discussed.

The psychology of malignant disease

Bereavement and cancer: Results from the OPCS longitudinal study

Dr D. R. Jones, St George's Hospital Medical School, London

The results of initial analyses comparing cancer registration and death rates following widow(er)hood with corresponding age-sex specific rates in a large sample representing the whole national population have already been reported.¹ Data were derived from the OPCS Longitudinal Study, in which routinely collected data on cancer registrations, deaths and deaths of a spouse occurring in a 1% sample of the population of England and Wales in the period 1971-1981 are linked together and with the 1971 Census records of sample members. There was little evidence of an increase in cancer registration rates following widow(er)hood and only a weak suggestion of increased cancer death rates, offering little support for the hypothesis that stress (of bereavement) is implicated in the aetiology of cancer.

In this paper, patterns of post-widow(er)hood cancer morbidity and mortality are explored in several more detailed analyses. In particular, the dependence of mortality ratios on factors such as sex, age, social status, economic activity and measures of social and familial support, including household structure and family size, are investigated.² In general, the differentials observed are similar to those to be seen in general mortality.

Variations of mortality ratios with time since bereavement are also considered. Although cancer mortality ratios in the period immediately following widow(er)hood are much less elevated than those for other causes such as accidental and violent deaths, there is some evidence of excesses of cancer deaths after longer periods, e.g. an excess of about 50% from breast cancer three or more years after widowhood. Other aspects of post-bereavement cancer morbidity and mortality analysed include the patterns of causes of death in spouse pairs. Concordance of cancer deaths in spouse pairs lies close to the expected value; in other words, there is no evidence that cancer is the cause of death of both members of a spouse pair more often than is to be expected.³

¹JONES, D. R., GOLDBLATT, P. O. & LEON, D. A. (1984) *British Medical Journal*, **289**, 461-464.

²— & — (1986) *Stress Medicine*, **2**, in press.

³— & — (1986) *Journal of Biosocial Science* 1986, in press

Psychotherapy with cancer patients

Dr Pamela Ashurst, Royal South Hants Hospital, Southampton

A diagnosis of cancer constitutes an emotional crisis for the individual concerned, and for his/her immediate family, which is frequently aggravated by the management and

treatment of the condition. Cancer patients are under a special and severe form of stress and the price of survival may be considerable. Cure, if possible, must be the aim of both patient and clinician; nevertheless the good physician must have a concern also for his patient's *feelings* about the disease and its treatment. The severity of the condition, the nature of the treatment and the possible consequences for body image and function, and the broader psychosocial concerns which may deeply affect the quality of life, will all influence the patient's reaction.

The individual with cancer faces his own mortality in a very special way, and other life issues may be brought into sharp focus when physical survival is under threat. This paper draws on clinical experience of psychotherapy with patients in whom cancer has been diagnosed.

Tardive dyskinesia

Non-neuroleptic aspects of tardive dyskinesia

Drs D. J. King, A. Wilson and S. J. Cooper, The Queen's University of Belfast and Holywell Hospital, N. Ireland; Drs J. Waddington, Department of Clinical Pharmacology, Royal College of Surgeons in Ireland, Dublin and H. A. Youssef, St Davnet's Hospital, Monaghan

It has been difficult to demonstrate a relationship between the presence of tardive dyskinesia (TD) and the history of neuroleptic drug exposure in clinical studies.¹

This report summarises three surveys of TD in chronic schizophrenia in two hospitals. The first included 68 patients (mean age 65.1 years), 28 (41%) of whom had TD according to the criteria of Schooler and Kane.² The second and third studies looked at 16 patients (mean age 44.4 years) who had previously had CT scans as part of another study. Four (25%) of these had TD on both occasions (persistent TD).

No association could be demonstrated in any of these three studies between either the prevalence or severity of AIMS and any measure of exposure to neuroleptic drug treatment (age of onset, duration, mean daily dose, life-time intake, or current dose). Negative symptoms, however, (mutism and affective blunting) and cognitive impairment were consistently associated with TD. Patients with persistent TD had significantly larger cerebral ventricles (VBR) than those who did not ($P < 0.05$). AIMS scores were also highly correlated with the presence of neurological soft signs ($r_s: 0.914, P < 0.01$).

Thus chronic schizophrenic patients with persistent TD are characterised by negative symptoms, cognitive impairment and large VBRs. Neuroleptic drug exposure would appear to be neither a necessary nor a sufficient explanation for TD in these patients.

¹CUNNINGHAM OWENS, D. G. et al (1982) *Archives of General Psychiatry*, **39**, 452-461.

²SCHOOLER, N. & KANE, J. M. (1982) *Archives of General Psychiatry*, **39**, 486-487

Tardive dyskinesia: Which patients are at risk?

Dr T. R. E. Barnes, Charing Cross Hospital & Westminster Medical School, London

The majority of patients receiving antipsychotic drugs do not develop tardive dyskinesia. The identification of factors contributing to individual vulnerability to this condition

should be of help in its prediction and prevention. With a paucity of long-term, prospective studies testing the predictive value of both treatment variables and patient variables of possible relevance, evidence from cross-sectional and retrospective studies, and short-term follow-up investigations, of tardive dyskinesia is examined. Almost invariably, a statistically significant relationship has been found between the main treatment variable, the administration of antipsychotic drugs and risk of tardive dyskinesia, although no such relationship has been convincingly demonstrated for dose, duration or type of drug. There is little evidence to implicate the chronic use of anticholinergic agents, or prolonged hospitalisation, as important risk factors. The patient variable predicting, most consistently, the appearance, severity and persistence of the condition is advancing age. Other patient variables that may be associated with an increased likelihood of developing tardive dyskinesia include a susceptibility to drug-induced parkinsonism and akathisia, a history of brain damage and a diagnosis of affective disorder. Also, recent findings, including preliminary data from our own work (Barnes, T.R.E. & Liddle, P.F., unpublished), suggest a significant positive relationship between the presence of negative symptoms in schizophrenic patients and tardive dyskinesia.

Measurement of extrapyramidal symptoms and involuntary movements by electronic instruments

Professor P. R. A. May, University of California, USA

Abnormalities of the extrapyramidal system (EPS) and involuntary movements (IM) such as tremor, rigidity, and dyskinesia are of great importance in psychiatry. They occur in a variety of organic brain disorders and, even more importantly, as side effects from neuroleptic drugs, in particular, dyskinesia, dystonia, akathisia, akinesia, rigidity, and lack of emotional expression. These create misery and suffering for the patient, and the problem has serious implications for all patients who are receiving neuroleptic medication.

A major problem is that EPS and IM are difficult to measure. Reliance has been placed largely on rating scales that are heavily dependent on the rater's subjective judgement, and whose inter-rater agreement leaves much to be desired. Measuring instruments have been used in some research studies, but the methodologies in their present form are not suitable for general use. Hence, research and clinical work in this area have been limited by lack of precision.

During the last eight years, the investigator has initiated development of a battery of prototype instruments to measure and analyse EPS-IM directly, objectively, non-intrusively, and with a high degree of precision, that are easy to administer, and that require only a relatively low degree of co-operation from the subject. Substantial progress has been made. Prototype instruments (systems) have been developed to measure tremor and involuntary movement of various kinds in different parts of the body, in particular, resting, sustention and intention tremor; facial, leg and trunk, and general body movement; and blink rate. Can examples and pilot data indicate that such equipment would be useful for early recognition, accurate diagnosis, long-term monitoring, documentation, to guide appropriate adjustments in drug therapy, and to stimulate staff education in this area?

With any laboratory test, the ideal system should combine the skills of the clinician with the capability of a machine. A machine can measure the degree and frequency of movements better than a skilled clinician. But a clinician is still essential to classify/identify the type of movement and to differentiate artifacts. In other words, the machine's function is to assist and extend the capabilities of clinicians, not to replace them.

Caring for the carers

The contribution of the psychogeriatric service from the carer's viewpoint

Dr P. Coleman, University of Southampton

As part of an investigation into the factors underlying effective collaboration between services for the elderly mentally infirm, interviews were carried out with a cross representation of staff in social, geriatric and psychiatric services in two health districts, one with and one without a specialised psychiatric service for the elderly. These indicated the contribution of the psychogeriatric service to the education of other professionals, and improved liaison between them, as well as the provision of a more effective service to patients and their carers, with a prompt response to referrals and reliable follow-up and support. Interviews with a small group of carers of demented elderly people referred to services provided evidence consistent with this evaluation, but also indicated points for further investigation. These included the counselling role of the community psychiatric nurse, the need for the right kind of day care, and the value of relief admissions.

Sitting service for demented patients

Dr H. P. Rosenyunge, Moorgreen Hospital, Southampton

In 1983, a sitting service for the elderly patients suffering from dementia was set up with the aim of offering home based support for the relatives living with them. A salaried, part-time co-ordinator recruited and matched paid sitters with the cases referred by the psycho-geriatric unit. Finance was obtained from Joint Funding and was administered by the local branch of MIND. At the end of the three year pilot study, 99 patients had received sitting service support. This paper describes the setting up of this service, the types of patients and families receiving support. An attempt to analyse its effect on the families studied and on the psychogeriatric service is made. A reduction in long-stay bed usage is shown.

Caring aides as an extension of the community psychiatric nursing service in Bournemouth and East Dorset

Drs M. A. Rowton-Lee and C. J. H. Williams, St Ann's Hospital, Dorset

Caring aides (patient aides) are unqualified persons recruited locally to support and relate to the community psychogeriatric nurses caring for ESMI patients in the community, concentrating mainly on those with brain failure. They may or may not have previous nursing experience: personality factors are important—lots of common sense and a liking for looking after people.

Bournemouth and East Dorset is a district with a high loading of elderly people. In a population of approximately 400,000, nearly 25% are over the age of 65. The projected population over the age of 75 is 11% compared to 6.8% for

the national average. A large proportion of the elderly in Bournemouth and East Dorset have migrated into the area on retirement away from their extended family and live alone and unsupported. The district is under-resourced in terms of beds available for the care of Brain Failure and this falls well below the recommendations in *Priorities for Health and Social Services in England* (HMSO 1976). Based on a planning norm of 2.75 beds for 1000 of the population over the age of 65, the district was undermanned by 100 beds.

It is with this background that the concept of caring aides, also known as patient aides, was developed. The number of beds available was simply inadequate. The community psychogeriatric team had been developed and its effectiveness was therefore extended by the addition of the patient aides. In 1977, Christopher Williams, general practitioner and hospital medical practitioner, prepared a paper for the Dorset Area Health Authority *The King's Park Hospital at Home—An Option for the East Dorset Health Care District*. In it he reviewed the application of the 'hospital at home' concept to the needs of patients with brain failure being cared for in the community. This coincided with the realisation of the clinical team of which he was a part that there was a need to extend the caring capacity of the domiciliary team. The notion of caring aides attached to the community psychogeriatric nurses and through them to the clinical team was developed and provided a substitute for the extended caring tribal family.

A pilot project was established in 1981 and the work of four patient aides, two in Bournemouth and two in Poole was monitored with the help of the Health Services Organisation Research Unit at Brunel. The report on a year's work *The Extended Care Project for Psychogeriatric Patients*, validated the view already held at team level that the patient aide was a valuable extension to the community team, increasing the quality of care for dementing patients in the community and that they improved the quality of life of both patients and carers.

Patient aides receive in-service training. Their job description is flexible and allows them to cross the artificial boundaries of Health and Social Services. It is important that they co-operate with home helps, concentrating more on the care of the person rather than the care of the environment. In practice if the selection of the right kind of person has been carried out properly, this interaction takes place without fuss. Difficulties mostly occur if administrators define the job too narrowly, the concept works best with flexible action and positive thinking. Lines of communication are important and the patient aide is responsible to and relates with community psychogeriatric nurses, who themselves maintain important lateral links with the Social Services and primary health care teams. The close contact between the patient aide and the clinical team allows the patient aide to feed in information rapidly about a deteriorating clinical situation.

Patient aides were shown to perform 'basic looking after work'. This included attention to physical comfort (including toileting); physical safety, psychological comfort (including structured social stimulation and orientation to personal and social reality), attention to feeding as well as the ordinary ministrations which are needed when an individual cannot look after himself. Attention to physical comfort may include giving a bath or tidying up a room. Attention to feeding may include actual feeding or helping

a patient to prepare food, either as a part of seeing that patients get food or as a part of a programme of rehabilitation, social stimulation may range from chatting to therapeutic reminiscing or even taking them to a pub for a drink. Duties include help with incontinence aids, facilitating the hospital to community laundry service, supervising medication and assisting with shopping, collection of prescriptions and pension, payment of bills and providing a granny-sitting service to enable relatives to get 'time off'.

Assessment of the project leads us to believe.

- (a) That the length of time for which a patient can remain out of institutional care is extended and the quality of life is improved.
- (b) The quality of life for caring relatives and supporting friends is also improved and not only is there increased willingness for them to continue to support but support in the community becomes the preferred form of management.
- (c) Other supporting services are prepared to continue to support the patient at home and tend to produce more positive input (responding to increased support by the clinical unit) instead of withdrawing their services because they feel overwhelmed, isolated and in difficulties.
- (d) Crises are averted and coped with
- (e) The expertise of the in-patient unit becomes available to a greater number of the elderly mentally infirm, and can be, at least theoretically, extended to all. Because people are being managed in the community instead of being institutionalised, the unit beds and particularly the assessment beds are available to patients (all of whom are equal number 1 on the waiting list) to come in when they are ill, or there is a crisis, and in-patient care is therefore available when most needed.

The concept of the caring aide was not devised to be a cheap alternative to inpatient care, but it has in fact proved cost effective. The project demonstrated that the service provided at relatively little financial cost enormous benefits to patients, relatives and other caring agencies. Patient aides experienced considerable job satisfaction. Often important bonding occurred between aides and patients and families they were supporting. Born out of necessity, helped by the caring aide, the community support became the caring service of choice for brain failure.

Support group for carers

Mr N Moffat, Branksome Clinic, Poole

An evaluation is provided of the setting up and running of a support group for relatives of patients with dementia.¹ The first stage reports a survey of known carers in Britain. Following this a survey of the needs of local carers was carried out and a 10 week programme chosen from this needs questionnaire. The initial evaluation involved 17 carers who were interviewed before and after being on a waiting list or participating in a support group. Participants provided positive feedback on all meetings. There were non-significant trends for a reduction in carers' expressed need for information, and in scores on the General Health Questionnaire. There were no significant changes in the Ways of Coping Questionnaire, and changes on the Personal Questionnaire were more related to respite care than to attendance at the support group. The small sample size of the initial study is recognised and the results from a further sample is reported. The formation of a monthly

outing for carers is described as a follow-up to the short-term structured groups.

¹TRIGG, A. (1984) Evaluation of a structured support group for relatives of patients with dementia. BPS Diploma.

Short papers

Attempted suicide by deliberate self-poisoning in four immigrant groups

Dr J. Merrill, All Saints Hospital, Birmingham

Rates and characteristics of deliberate self-poisoning in four immigrant groups, Asian, West Indian, Irish and Scottish were compared with those of white English-born resident in an inner-city area of Birmingham. Irish and Scottish-born were over-represented in all age/sex groups. Asian and West Indian males and older females were under-represented. Substantial differences in the characteristics of self-poisoning were found between immigrant and control groups. Alcoholism was increased in Irish and Scottish groups. Asians frequently suffered from marital problems and culture conflict. Teenage pregnancy and separation from parents in childhood were more common in West Indians. The relevance of the findings to the assessment and management of self-poisoners is discussed.

Deliberate self-harm: correlates of suicidal intent and severity of depression

Drs G. O'Brien, A. Holton, K. Hurren, L. Watt and F. Hassanyeh, Nuffield Unit for Child Psychology and Psychiatry, Fleming Memorial Hospital and Royal Victoria Infirmary, Newcastle-upon-Tyne

It is well-recognised that people who commit acts of deliberate self-harm vary widely in their suicidal intent and severity of depression. This study sought to identify characteristics which correlate with suicidal intent and depressions and also to assess how well some of the available rating scales correlate with each other, in this population.

Ninety-eight consecutive referrals for deliberate self-harm were given a semi-structured clinical interview. Cases were also assessed on Spitzer's Research Diagnostic Criteria (RDC), on the Pearce Suicide Intent Scale, on the Hamilton Depression Scale and on the Newcastle Diagnostic Scale for Depression. In addition, the interviewing clinician made a clinical decision on diagnosis.

The findings showed that the RDC 'cases' had higher suicidal intent and were more significantly depressed on Hamilton Scale than 'non cases'. Diagnosis of 'Endogenous' type depression on the Newcastle Scale had a high correlation with Hamilton Score and a moderate correlation with suicidal intent. In addition to high correlation with RDC 'caseness' and Newcastle diagnosis of endogenous depression, there was a high correlation of Hamilton Score with suicidal intent. With respect to early parental loss or bereavement, there was no significant correlation with any of the above measures.

Rape: The behavioural and psychological sequelae in the partners of the victims

Dr A. W. Bateman, The Royal Free Hospital, London

The plight of the rape victim has been given increasing

attention over the last decade but very little emphasis has been placed on the reactions of their partners. Over the last year the partners of rape victims referred to a victim support scheme have been interviewed and counselled. A characteristic response by victims' partners has emerged.

The liaison with a voluntary community organisation will be discussed and the behavioural and psychological changes in the male partner of the rape victim described in detail. Illustrative cases are used to show how the psychological changes in a victim's partner may not only have severe consequences for the individual but also interfere with any help offered to the victim.

Delusional and non-delusional depression in late life: Evidence for district subtypes

Dr R. C. Baldwin, Manchester Royal Infirmary

The study tested the hypothesis that delusional and non-delusional depressive illness are distinct clinical entities in late life and carry different prognoses. From a larger retrospective study (in press) aimed at clarifying the long-term outcome of major depression in old age, 24 patients were identified with late onset delusional depression and were matched to 24 late onset non-delusional controls with depression. At the index admission delusional cases were significantly more depressed, were hospitalised for longer and received significantly more by way of physical treatments, especially ECT and major tranquilisers. However, the clinical status at discharge was no different from the controls, neither was the outcome, over a period of between 42 and 104 months, in terms of relapse rates, deaths and overall prognosis for depression. One striking difference was the finding that whilst relapses in the delusional group were frequently characterised by further delusions, this never happened for the non-delusional controls. The latter finding, coupled with the severity and treatment differences mentioned, suggests that the view already widely held for younger patients and based on similar evidence, namely, that delusional depression is distinct from non-delusional forms, is also true for the elderly. These findings do not, however, support recent work which suggested a very poor prognosis for delusional depression in late life.

Personality and emotional disturbance in rheumatoid arthritis

Drs F. McGregor and P. Jauhar, Duke Street Hospital, Glasgow

One hundred and eighty-two patients with rheumatoid arthritis were studied to examine the distribution of personality scales and the incidence of psychiatric morbidity and to explore the effects of pain, disability and serology upon these indices. All the patients completed a set of questionnaires including the Eysenck Personality Questionnaire, the Hospital Anxiety and Depression Scale and the General Health Questionnaire. Seventy-six patients were interviewed using the Clinical Interview Schedule. Patients were more likely to be introverted than population norms, women were more likely to produce socially desirable results and men were more likely to have a tendency to neuroticism and were significantly more likely to be emotionally disturbed. Pain and disability were found to be associated with psychological disturbance in women, but only disability was an important factor in men. The importance of other factors in these findings is discussed.

Peter Scott Scholarship 1984. Help for the victim of violence: are there lessons to be learnt from American rape crisis programmes?

Dr G. C. Mezey, The Maudsley Hospital, London

I have recently completed a study at the Institute of Psychiatry investigating the effects of rape on the victim. As a result of this work, I was funded by the Peter Scott Scholarship to visit hospital-based rape crisis programmes in the United States earlier this year. This paper is the outcome of the visit and describes how the care of rape victims in the States has become a very integral part of the total Health Service provided by hospitals.

The American Rape Programmes were set up in general hospitals in the early 1970s (McCombie et al 1976)¹ and have since proliferated in number, so that almost every major city now has at least one such programme operating. The programmes are run by a multidisciplinary team and provide for the physical, emotional as well as the practical needs of the victim. They function for 24 hours a day and provide both acute and follow-up services.

Staff training in all disciplines reflects the importance given to the concept of 'psychic trauma'. Victims of violence are seen as having as legitimate a claim to the health services provisions as patients who present with 'physical' emergencies. The paper describes the functioning and set up of rape crisis programmes in one large city in N America: Boston. It shows how these programmes link up with other, 'community' based services, with the police and legal bodies. I describe some of the recent laws passed relating to rape victims, perhaps a reflection of the increasing seriousness with which this problem is viewed. Finally, I discuss some of the current victim research that is being done by the Boston groups and the implications this may have for our work as psychiatrists in Britain.

¹McCOMBIE, S., BASSUK, E., SAVITZ, R. & PELL, S. (1976) Development of a medical centre rape crisis intervention programme. *American Journal of Psychiatry*, 133, 418-424

Opiate withdrawal: A comparison of in-patient and out-patient programmes and of preferred versus random assignment to treatment

Drs A. Johns, M. Gossop and Mrs L. Green, Bethlem Royal Hospital, Kent

This prospective study looks at the relative effectiveness of an in-patient and an out-patient withdrawal programme for opiate addicts. Addicts were more likely to achieve a drug-free state on the in-patient withdrawal programme than on the out-patient scheme: success rates were 18% (in-patients) versus 17% (out-patients). This in-patient/out-patient difference could not be attributed to pre-treatment, drug-related factors, nor to social or psychological differences between the groups. The results have clinical and policy implications for the treatment of opiate addicts and some of these are discussed.

Factors associated with compliance in the follow-up treatment of alcoholism

Dr B. Fleming, Shaftesbury Square Hospital, Belfast and Professor Stuart Lewis, Queen's University, Belfast

This study set out to examine factors associated with compliance in the follow-up treatment of alcoholism. Twenty-five patients who dropped out of treatment within six months were compared with 25 patients who completed

a two-year treatment programme. The two groups were compared for socio-demographic factors and personality traits using the EPI and Cattell 16PF. Personality traits did not discriminate between the two groups. However, as in previous studies significant correlation between low socio-economic class and dropping out of treatment was found. Marital instability and a family history of alcoholism also tended to correspond with dropping out of treatment, although this tendency was not statistically significant.

High section 136 admission rates: A challenge to community psychiatry

Drs T. Fahy, The Maudsley Hospital, London, D. Bermingham, St Thomas' Hospital, London; J. Dunn, Tooting Bec Hospital, London and P. Joyce, St Augustine's Hospital, Kent

Section 136 admission rates to St Augustine's Hospital, Canterbury and Tooting Bec Hospital, London were reviewed for the two-year period following the introduction of the 1983 Mental Health Act. Rates of three and six times the national average respectively were detected. There were many similarities between the groups despite major demographic differences. Factors influencing admission rates differ between regions. The St Augustine's group was surprisingly heterogeneous for several variables. Absence of out-of-hours social work support was judged to be a crucial determinant of their high rate. By contrast the Tooting Bec sample were heavily weighted for a large number of variables including sex, age, ethnic origins and certain diagnostic categories. In both groups over one third of patients received a diagnosis which was not classifiable under the Mental Health Act definition of a mental disorder. An intensive community based response to the problem of high Section 136 admission rates would be well positioned to tackle important aetiological factors and may contribute to reduce the number of such admissions.

The micro-computer in rehabilitation

Dr A. McDonald, The State Hospital, Carstairs

The rehabilitation of schizophrenic patients is one of the major problems of psychiatry. The illness renders a person peculiarly susceptible to stress and unable to participate fully in day-to-day relationships and living. For many, thought disorder and lack of motivation prohibit the full use of their intellect when more florid psychotic symptoms may have disappeared.

This study sought to examine the use of a micro-computer by long-term schizophrenic patients as a means to motivate them, to allow them to function at an appropriate intellectual level and to facilitate interaction with other people. Although no such previous work has been done this multi-disciplinary project drew heavily from experience in the field of special education.

The use of the computer by 11 chronically ill, several incapacitated, schizophrenic patients is described. No particular type of programmes were found to be of preference but it was crucial that a therapist shared the experience with the patient. Most important in its use as a facilitator is the concept that the mastery of the computer is not an end in itself but rather a rehabilitative aid in the schizophrenic's relationship with other people. Whilst some delusional interpretations were expressed in relation to the computer it did not become an object of paranoia and showed itself to be a good motivator, facilitating interaction with other

patients and staff which generalised beyond the computer setting. Further research would seem to be indicated although the progress obtained does not lend itself readily to quantitative assessment.

Early mother/baby contact: positive consequences

Dr de Wet S. Vorster, Plymouth Nuffield Clinic, Plymouth
Winnicott stressed the 'holding' of the infant as of great import especially relevant to the more severe psychiatric illnesses in later life. A few prospective studies have indicated the import of early mother/baby contact concerning later healthy psychobiological development. Klaus indicated the import of the first half hour postbirth contact and follow up at ages three and five years showing a better mother-child relationship. Attempts to replicate these studies have not always succeeded; the improvement in the experimental group (close contact) may have been due to other factors. However, de Chateau's work revealed no major differences in personality characteristics of mothers in both control and close contact groups.

As studies have involved small numbers, a larger sample was pursued in the study described. Findings from the present ongoing study of 300 mother/baby pairs at the six week developmental testing will be presented.

An early mother/infant contact questionnaire was completed by clinical medical officers. Key questions included duration of contact postbirth and in first months, most successful cry cure, and possible relationship between early contact and subsequent symptoms in mother and baby. Results mothers and babies who had 20 minutes or more contact postbirth presented fewer problems e.g. crying, feeding disorder, at age 6 weeks, 10 minutes or less contact presented the same percentage of problem behaviour as for those separated at birth; more problems occurred in the latter two groups to a significant degree statistically.

To verify the memory of mothers concerning the infant contact questionnaire represented by medical officers at six weeks, further work has included a mother/baby bonding chart completed by midwives on a second group of 300 mother/baby couples recording the length of time the baby was in proximity to mother immediately postbirth, as well as recording Apgar ratings and obstetric factors, destination of baby after leaving mother and reasons for interruption of holding time. Babies' response to holding, amount of crying and type of feeding were recorded. Results indicated that only a third of mothers and babies were together for 20 minutes or more; crying was not frequent in this group of babies; breast feeding occurred in the same percentage of both groups and obstetric factors failed to account solely for different holding time. Results of later follow-up are presented.

Television violence and childhood aggression: The field experiments

Drs K. D. Gadow and J. Sprafkin, State University of New York, USA

For over two decades the viewing of television violence has been implicated as playing a role in childhood aggression either as an etiological mechanism, an environmental stimulus that exacerbates symptoms, or both. Although laboratory studies are generally interpreted as providing overwhelming support for these notions, actual acts of symptomatic behaviours are typically not recorded in these studies. Rather, child behaviour is generally evaluated with

regard to reactions toward inanimate objects or playing games. The more ecologically valid field experiments (i.e., conducted in natural settings) are also considered to support the aggression-induction hypotheses. A careful reading of the 15 field experiments conducted to date (four of which were recently completed by our research team) provides only weak support for this interpretation. In fact, the majority of these studies show that viewing non-aggressive material is generally more aggression inducing than viewing aggressive shows. Moreover, our own research on emotionally disturbed (conduct disordered) and learning disabled children has clearly shown that watching aggressive television programmes is *not* more likely to exacerbate the symptoms of conduct disorder than non-aggressive content. Interestingly, while existing research efforts do not appear to support the aggression-induction hypothesis, they do indicate that television is not a neutral medium. Compared with baseline levels of antisocial behaviour, many studies show that, regardless of content, television viewing does lead to higher levels of negativistic interpersonal interaction. The findings from all of the field experiments are discussed with regard to their clinical relevance for mental health professionals.

First episode schizophrenia: Effect on the family

Dr M. G. Livingstone, University of Glasgow

A sample of 49 schizophrenics in their first episode of the illness was recruited. Data were collected on psychopathology, cognitive functioning and CT scans, together with an assessment of the relatives' psychiatric and social functioning. This paper reports on the relative' psychosocial status throughout the year following initial episode of schizophrenia. An association between the relatives' adjustment and patient's symptoms is described.

Auditory hallucinations and subvocal speech in schizophrenia

Drs P. A. Bick and M. Kinsbourne, University of Dundee

Hallucinations have variously been regarded as an expression of excess (disinhibited) brain activity or products of adaptive behaviour, either searching for organisation in a chaotic array or motivated by dynamic imperatives. We have examined whether auditory hallucinations arise from brain dysfunction, or compensate for it, with respect to the recurrent claim that these are accompanied by subvocalisation.

We carried out a study in 18 schizophrenic patients who complained of voices. Each patient was asked to perform three tasks, one of which (holding the mouth wide open), has been shown to prevent subvocalisations. The control tasks were to make a fist with both hands and squeeze tightly and a facial control manoeuvre, screwing the eyes tightly. The interviewer was unaware of the experimental hypothesis. Fourteen of the patients reported that their voices went away when they undertook the manoeuvre that precluded subvocalisation.

To investigate the generality of this phenomenon, 35 normal subjects were hypnotised and given the suggestion to hear voices. Twenty-one accepted this suggestion. Subjects were instructed to perform the same manoeuvres as the schizophrenic group. The mouth opening manoeuvre abolished hallucinations in 18 of the subjects. This finding aligns the schizophrenic's experience with behaviour to which normal subjects may 'unconsciously' resort.

These results confirm that subvocalisation accompanies

auditory hallucinations in schizophrenics. Previous studies have included no specific or controlled manipulation of this phenomenon, and it could not, until now, be excluded that schizophrenics shadow in whispers, voices that they hear. Our data demonstrate that schizophrenics actually generate voices, they do not merely shadow them. A theory can be propounded that hallucinations represent verbal thoughts, disinhibited into vocal activity. This might explain why neuroleptics are often ineffective in counter-acting voices. This might also explain why the patients in this study were profoundly unimpressed by the revelation that they could easily abate their troublesome voices. Treatment strategies for troublesome hallucinations need to be reassessed in the light of this finding.

Cerebral perfusion in schizophrenia

Drs R. J. Mathew and W. H. Wilson, Duke University Medical Centre, USA

Regional cerebral blood flow (rCBF) measurements were obtained with the ^{133}Xe inhalation technique in 100 patients with a diagnosis of schizophrenia and an equal number of normal controls matched on age, sex and cerebral dominance. The patients were screened with Schedule for Affective Disorders and Schizophrenia and the diagnosis was based on DSM-III criteria. Psychopathology was quantified with Brief Psychiatric Rating Scale (BPRS) and a standardised scale for positive and negative symptoms of schizophrenia. All cerebral blood flow measurements were conducted in a semi-dark, quiet room under identical conditions. Thirty patients were kept drug-free for a minimum of two weeks before the rCBF measurements. All subjects were required to abstain from caffeine and tobacco for at least three hours before measurements. Normals showed increased frontal perfusion in comparison to the rest of the brain. The patients, however, showed reduced frontal-posterior gradient in cerebral perfusion in comparison to the controls. The relationships between rCBF and such clinical variables as duration of the illness, family history of schizophrenia, number of previous hospitalisations, symptoms of schizophrenia and treatment with neuroleptics and relevant physiologic variables (CO_2 , hematocrit) were examined.

Brain changes during ECT using magnetic resonance imaging

Drs A. J. Mander, A. Whitfield, D. M. Kean, M. A. Smith, Miss R. H. B. Douglas and Professor R. E. Kendell, University of Edinburgh

Fourteen patients satisfying RDC criteria for a major depressive episode had an NMR scan before and immediately after the first and approximately the last treatment of a course of ECT. Subsequently three of the patients were scanned at intervals over the following 24 hours. No progressive changes in the T1 relaxation time were noted over the course of ECT, but acute changes did occur after each individual treatment, reaching a maximum 4–6 hours following the fit. The changes appeared to be generalised in nature and there was no evidence of lateralisation in those patients given unilateral ECT.

The data are consistent with the observation that the blood brain barrier breaks down during a fit and it is of particular interest that the maximum change occurs some hours later. The results must be viewed as encouraging, since there was no evidence of permanent or progressive structural change as a result of treatment. More work is

now needed on the temporal changes of T1 with a larger patient sample and whether these changes are related to improvement or side effects. The underlying physiological basis of the change in T1 is still to be elucidated.

Minor psychiatric morbidity in people with epilepsy: Evidence for a gender difference

Professor G. W. Fenton, University of Dundee

All the patients with epilepsy in a total practice population of 75,200 were identified. Those agreeing to participate in the study were reviewed by their GP and visited at home by a research health visitor. Those aged 16 years or over completed the Crown Crisp Experimental Index (CCEI). A control sample of non-epileptic patients from the same practices, matched for age, sex and social class was also examined. The male epileptic patients had a higher prevalence of certain neurotic symptoms and traits. This gender difference related to seizure frequency and employment status, but not to polytherapy, serum anticonvulsant levels nor class of epilepsy.

Mood disorders in epilepsy: A survey of psychiatric patients

Drs S. S. Palia and M. A. Harper, Glan Rhyd Hospital, Wales

In-patients and day-patients with epilepsy in a general psychiatric service were surveyed. Fifty-three patients able to co-operate in a detailed assessment were studied using the Hamilton Rating Scale and the Beck Depression Inventory. About one-third (32%) of the patients had serious depressive illness mostly classified as non-endogenous on the Newcastle Index.

Neurophysiological factors were overshadowed by the psychological and social problems generally found in depressed patients. Almost all of the depressed epileptics gave histories of repeated previous deliberate drug overdose and a past history of depression. The multifactorial nature of depressive states in epilepsy is emphasised.

It is suggested that the occurrence of intense short-lived depressive moods associated with suicidal behaviour in epileptics may be due to the release of affect secondary to fluctuations in consciousness in vulnerable individuals rather than to specific neuro-physiological or pharmacological factors.

Psychopathology of the Gilles de la Tourette syndrome

Drs M. Robertson, M. Trimble and A. Lees, The National Hospital, London

The history of an association between psychopathology and the Gilles de la Tourette Syndrome (TS) is long, particular reference being made to obsessive compulsive behaviour, aggression, dysphoria and changes in neuro-psychological function.¹ However, most studies are anecdotal and clinical, often relying on a small number of cases. One group attempted a systematic evaluation of psychopathology among TS subjects and not only failed to find an increased amount when compared to a general psychiatric outpatient population, but reported nothing specific.² We have carried out a study of 90 patients with TS, using standardised psychiatric rating scales and specialised tests including neuropsychological testing, EEGs and CAT scans. Psychopathology was prominent. In particular, patients exhibited depression, hostility and obsessiveness. Depression was not related to the taking of medication, while aggression, hostility and obsessiveness

were significantly associated with some core features of the syndrome, namely copro- and echophenomena. Links between neurological and EEG abnormalities were minimal.

¹TRIMBLE, M. R. & ROBERTSON, M. M. (1986) The psychopathology of tics. In *Modern Trends in Neurology* (eds C. D. Marsden, and S. Fahn) Guildford: Butterworths (In press).

²SHAPIRO, A. K., SHAPIRO, E. S., BRUUN, R. D. & SWEET, R. D. (1978) *Gilles de la Tourette Syndrome*. New York: Raven Press.

Organic pathology and psychiatric morbidity in neurological referrals to a consultation/liaison service

Drs A. Schofield and A. Duane, Cork Regional Hospital, Ireland

This study analyses the patterns of referral of neurological in-patients to find out in what way they differ from referrals from the whole general hospital. The symptomatology of those patients with functional neurological illness is explored and the psychiatric morbidity of those patients with organic illness is evaluated. Neurology referrals differ from general hospital referrals in reasons for referral and in patterns of diagnosis. The commonest neurological illnesses referred are epilepsy, Parkinsonism and cervical spondylosis. The commonest conversion symptoms seen are pain and secondarily pseudoseizures. The psychiatric diagnosis made in those patients with functional pain varies with the site of the pain. These two conversion symptoms, pain and pseudoseizures have differing ages of onset and prognoses. A number of patients were given no psychiatric diagnosis and the reasons for this are discussed.

It is concluded that for the psychiatric treatment and management of neurological referrals to a psychiatric consultation/liaison service, a detailed knowledge of the patterns of symptomatology and psychiatric morbidity found in these patients is of benefit.

An electrophysiological study of senile dementia of the Alzheimer type

Drs P. A. Williams and G. H. Jones, University of Wales College of Medicine, Cardiff

Event-related potentials were studied in senile dementia in 20 subjects and compared with normal controls. The auditory evoked potential to a target stimulus consists of the N100, P200 and P300 components. The N100, N200 and P300 are all delayed in latency in this group of subjects. During the procedure the subject is required to press a button to detect the target stimulus and reaction time data are collected also, the latter is also delayed in dementia. A number of authors have also recently published data indicating delays in the P300 components in presenile dementia and there has been work on reaction time experiments in the literature. The authors describe their results and attempt to discuss them in terms of a perspective of information processing theory as a means of understanding cognitive processes in dementia.

The effect on the hypothalamus of an oestrogen provocation test in pre-operative transsexuals

Dr M. T. Haslam, Clifton Hospital, York

Donner working in Berlin noted that the differentiation into male and female hypothalamic types can be distinguished with reasonable reliability using an oestrogen provocation test and monitoring FSH and LH output for four days

thereafter. He noted an increased number of female type responses in male homosexuals whom he described as of effeminate type. It was hypothesised that in transsexuals and possibly some transvestites this phenomenon might also occur, thus confirming the transsexual's belief that he has a female brain functioning in a male body.

To test this hypothesis transvestites and transsexuals living in the York/Leeds area who were not on hormones and were prepared to take the test were screened with an oestrogen provocation test. A small control group was also utilised to form a comparison. The results of this trial are presented. It is noted that the incidence of female-type responses, while not reaching statistical significance in view of the smallness of the group, nevertheless show a considerably greater number than would be expected by chance. The significance of this result is discussed.

Increased bromide levels in serum and hair during lithium treatment

Drs C. A. Campbell, Hellesdon Hospital, Norwich; M. Peet, Northern General Hospital, Sheffield and N. Ward, University of Surrey, Guildford

Lithium has been shown to affect serum levels of a number of elements, such as calcium and magnesium.¹ A recent report has suggested an elevation in serum bromide levels in patients on long-term lithium carbonate treatment.²

In the study to be presented, the concentration of bromide was measured by neutron activation analysis in samples of serum from 32 patients on maintenance lithium carbonate and from 32 age and sex matched healthy controls. Whole blood and hair samples were also taken from a number of patients and controls. Bromide levels were found to be significantly higher in the serum and hair of patients in the lithium group compared to the control group (serum bromide $P0.05$; hair bromide $P0.0001$ two-tailed t test). The possible effect of this increase and the mechanism causing it is discussed.

¹CHRISTIANSEN C. et al (1978) *Acta Endocrinologica*, 88, 528-534

²HANDORF, C. R. et al (1985) *Journal of Clinical Psychiatry*, 46, 9-10.

Alcohol and drug problems in the elderly

Alcohol and Old People

Dr John P. Wattis, St. James's University Hospital, Leeds

Despite the increased proportion of old people in Western societies since the last World War and despite upward trends in alcohol consumption associated in part with industrialised production and marketing methods, doctors still do not expect to see elderly alcoholics or drug abusers. Following a recent upsurge of interest in this area it is becoming evident that alcohol abuse in old people is more common than was previously apparent. Evidence is also accumulating that the clinical presentation of alcohol abuse in old people is often different from the presentation in younger people and that alcohol abuse in old people far from being a harmless consolation leads to serious physical and psychological harm. With the information available it is now becoming possible to look at possible strategies for the prevention, early detection and treatment of alcohol abuse in old people.

Surveys have repeatedly shown that in different societies

old people consistently drink less alcohol than their younger counterparts. One study in Scotland showed, for example, that only 43% of elderly men and 22% of elderly women were 'regular drinkers' compared with 74% and 46% of the total male and female populations. This and similar studies in other countries can be criticised on two main counts: (a) the definitions of 'regular' or 'heavy' drinking are the same for young and old, male and female, despite evidence that women and old people tolerate alcohol less well than young men; (b) it is quite possible that old people under-report alcohol consumption for a variety of reasons. Epidemiological studies of the prevalence of alcohol abuse as opposed to heavy drinking suggest that in some cultures at least, female prevalence for alcohol abuse peaks in the over-70 year-old age group. This finding concurs with clinical studies which have suggested that there is a late-onset group of female alcoholics who presumably take to excessive drinking for the first time in response to the stresses of ageing including bereavement and loneliness.

The clinical presentation of alcohol abuse in old age is also distinctive. Although some elderly alcoholics are longstanding survivors of chronic excessive drinking, others present with problems such as repeated falls, self-neglect and confusion, typical 'non-specific presentations' of disease in old people. In these people in particular the true diagnosis is easily missed.

The harm that results to old people who drink excessively is similar to the harm that occurs to younger drinkers. Old people may be more at risk for alcoholic dementia and for problems associated with poor nutrition. In addition they may have different patterns of drinking and supply than younger people. They may be dependent on others to supply alcohol within the home or may even be encouraged to drink by other younger alcoholics who regard the old person's pension as a useful source of alcohol for their own consumption.

The detection of alcohol abuse in old people thus demands a high level of awareness on the part of medical and social services staff and on public awareness that heavy drinking in old people is harmful and should not be dismissed with such phrases as 'it's all she's got left'. Treatment is in two phases. The acute phase involves alcohol withdrawal with sedative cover usually as an inpatient where associated vitamin deficiencies and other medical problems can be dealt with. Following this it is essential whenever possible to alter the patient's social support network so that excessive drinking is no longer encouraged. Prevention of alcohol abuse in old people raises similar issues to prevention in younger people though it may be that additional social measures such as more constructive opportunities in life for recently bereaved old people could have a part to play in the prevention of the late onset type of alcoholism.

The little explored area of drug abuse in old people is briefly discussed. The problems of active and passive abuse of prescribed drugs is considered and the positive role of the medical profession in reducing this problem is stressed.

A case register study of elderly alcoholics in Edinburgh

Dr K. J. B. Rix, University of Leeds

The Edinburgh Psychiatric Case Register was used to identify all alcoholics aged 60 years or more who made a new contact with the Edinburgh psychiatric services over a

six month period. A sex-matched comparison series of non-elderly alcoholics was obtained in the same way. Subjects were interviewed and questioned concerning demographic information, alcohol consumption, possible alcohol-related disabilities, symptoms of alcohol dependence and social functioning. Assessments were made of cognitive functioning. Thirty-four elderly and 32 non-elderly alcoholics were notified. At least 90% in both groups were traced and interviewed. The elderly alcoholics differed from the young alcoholics in the following respects: diagnosis of chronic brain disease, source of referral, service in which treated, outcome of marriage, type of accommodation, occupational status, alcohol consumption, history of parasuicide, alcohol-related crime, age of onset of alcoholism, social range, number and range of current long-term difficulties and experience of difficulties with work, police and finances. The differences in performance on cognitive tests disappeared when elderly alcoholics with a diagnosis of chronic organic brain disease (i.e. dementia or Korsakoff's psychosis) were excluded.

Understanding alcohol abuse in the older person

Mr C. J. Goodman, Social Services Department, London Borough of Richmond-upon-Thames

The elderly population is increasing and those responsible for the resources available will need to be more aware of the particular problems surrounding the process of growing old. One of the consequences of growing old is the increasing likelihood of loss. Losses can include the loss of home, status, children, job, independence, spouse, etc. Due to the paucity of services and time available, the counselling and understanding of the processes of loss are often insufficient to allow the old person to cope adequately. As a result alcohol is being used by the old person to cope with the stress caused by the loss.

There are, however, two types of elderly drinker, defined as the long-term user and the 'solution' drinker. The long-term drinker is far more difficult to help and is more likely to be labelled as 'hopeless alcoholic', due largely to the carer's inability to effect any change. The 'solution drinker', however, can be helped by the appropriate use of counselling and support and therefore provides us all with a chance to effect change in lifestyle. Although these two categories of drinker are different and their response is different, the use of the term 'alcoholic' does not help either the old person or the carer to understand the cause of the drinking or to help in the process of change. Indeed one function of the labelling process is to provide a comfortable definable status or sub-group in which the bearer can receive the comfort and support of a similar group of people. The effect of the label 'alcoholic' is to ensure that the person so described will behave in a way which will justify his inclusion into such a group. As professional carers we must be careful that the language we use to our clients does not exacerbate the 'condition' with which we have been asked to help.

Group therapy with elderly female alcoholics

Dr E. Guthrie, Manchester Royal Infirmary

Approximately 1 in 15 patients referred to alcohol treatment units are over 65. There is conflicting evidence as to whether these patients' pattern of drinking and treatment needs are different to those of younger patients with alcohol problems. A group for elderly female alcoholics was set up

on the ATU at Withington Hospital in 1983. The setting up of this group, its course over two years and the practical problems that were encountered are described. The main themes of the group are outlined and suggestions for future management of such patients are made

Suicide

Some observations on the epidemiology of suicide

Dr P. Sainsbury, Emeritus Consultant Psychiatrist, Graylingwell Hospital, Chichester

The singular value of suicide statistics to psychiatric epidemiology is illustrated by: (a) recent trends in England and Europe, (b) the relations between these trends and changes in socio-economic conditions in Europe, (c) some differences between social and other characteristics of cases of suicide and a sample from the general population which indicates who is at risk, and the (d) extent to which suicides have a recognisable and treatable psychiatric illness and the preventive implication of this.

The recent epidemiology of suicide in England and Wales

Dr G. M. G. McClure, Westminster Children's Hospital, London

The suicide rate for both sexes in England and Wales reached a post-war peak in the early 1960s and then declined until 1975. This decrease was associated with a fall in the number of suicides by poisoning with domestic gas, which was being detoxified during this period. The greatest decrease in suicide rate was for people over 45 years. Between 1975-1985 the suicide rate for females levelled off, but there has been a steady rise in suicide rate for males during this period. The greatest increases occurred for poisoning with vehicle exhaust gas and by hanging, strangulation and suffocation, while there was a decrease in poisoning with solids and liquids. There has been a substantial increase in suicide rate between 1975-1985 for males aged 25-44 years.

Prison suicides

Dr S. Beckett, Royal Edinburgh Hospital

A recent study has shown that suicide is the largest single cause of death in prison and accounts for approximately a third of the total number in this setting. Concern has been expressed about these deaths and the question most frequently asked is whether they could have been prevented. When considering possible preventive strategies, one has to ask if the characteristics of those who commit suicide in prison are similar to those in general population and whether any groups or sections of the prison population appear to be particularly vulnerable.

This paper addresses these questions using data from studies carried out both here in the UK and the United States. It also includes results from a study which examined the previous psychiatric contact of a group of prisoners who subsequently committed suicide.

The epidemiology of violent and unnatural death in one catchment area

Mrs E. King and Dr B. M. Barraclough, University of Southampton

Victims of violent death were identified from death registration information sources. Demographic, social, circum-

stantial and medical data were extracted from inquest files and hospital case notes. Patient status at the time of the event leading to death was ascertained for each referred psychiatric patient. In the eight years up to the end of 1981 there were 412 potentially self-inflicted deaths among catchment area residents which were subject to inquest by one of 22 coroners in England and Wales. 245 suicide (S), 126 accidental death (A) and 41 undetermined death (OV) verdicts were returned. The 1981 Census population of the Southampton Department of Psychiatry Catchment area was 297,000 (237,000 aged 15+). Between 1974 and 1982 the average annual catchment area female violent death rate (S+A+OV) of 15.3/100,000 population aged 15+ was identical with the corresponding England and Wales rate; the catchment area male violent death rate of 28.5% was 19% higher than the England and Wales rate with the increase entirely in the suicide rate.

Of the whole group of 412 violent death victims. (a) 56% (60% S; 50% A; 54% OV) were known from records to have been referred to a psychiatrist at some time; (b) 20% (21% S; 18% A; 27% OV) were actually receiving psychiatric care when they died; (c) 4% (6% S; 1% A; 2% OV) were psychiatric in-patients; (d) 16% (15% S, 16% A; 24% OV) were current psychiatric out-patients. During any year less than 2% of the catchment area population was recorded by the Southampton Psychiatric Case Register as being in contact with its psychiatric services.

Brief psychotherapy

Assessing and treating families of in-patients

Drs B. K. Rosen and P. Jarrett, Guy's Hospital, London

Work with families of in-patients requires adaptation of family therapy practice which has largely concentrated on intact families. The obvious difference is that one member of the family has been identified as ill by both the family and the helping organisation. The hospital has a special relationship with the sick member which must be addressed in work with the family. Perceptions of family relationships are likely to be distorted by the admission and an initial task for the unit is to develop an assessment which takes this into account. We have found a useful tool in the McMaster Model of Family Function which provides a systematic and structured assessment procedure. The McMaster model leads to the construction of a problem list which can also be incorporated into ward management and treatment procedures. Interviewing families in this way allows joint planning of treatment goals including task setting for weekend leave and discharge. Particular aims of family therapy include the following: (a) registration of the status of the patient and his illness within the family; (b) countering the family's sense of failure, guilt and suspicion of the treatment setting; (c) effecting change within the family system and sub system which can be maintained after discharge; (d) organising ward treatment programmes which are specifically targeted at the patient's problems within the family.

Assessing the individual

Dr A. Ryle, St Thomas's Hospital, London

It is proposed that a suitable first intervention for most neurotic and personality disordered patients is a focused time-limited (12 to 16 sessions) individual therapy. If adequately focused this will be sufficient treatment for most

patients and a suitable assessment procedure for those with more severe disturbances. Patients with severe drug or alcohol abuse or psychotic illness will be excluded and some patients may be better treated in family or marital therapy. The aim of the psychotherapy is to alter those patterns of relationships and self-attitudes underlying the patient's distress. Some patients, for example those with severe eating disorders or marked obsessive-compulsive disorders and some phobias, may need prior or concomitant behavioural treatment. Patients with major depressive symptomatology may need prior antidepressant medication.

For those taken on for the brief therapy the central task of the assessment phase, which usually lasts for four sessions, is to derive clear descriptions of the patient's repeatedly used maladaptive procedures. These descriptions may include hypothesising some mental processes of which the patient is unaware. By 'procedures' is meant the sequence of belief, anticipation, choice, action and evaluation used by the patient to organise purposive action; shorthand descriptions of neurotic procedures have been described as traps, dilemmas and snags. Explanations and descriptions of these are listed in the 'psychotherapy file' which patients are given to read after the first session. In reformulating patients' problems, therapists will draw upon clinical data, both historical and observational and on psychological test procedures and on the work which patients themselves will do. This will include self-monitoring of symptoms, other forms of writing and paper and pencil tests as well as the patients' reading of the psychotherapy file. By the fourth session it is usually possible for the patient and therapist to agree to a written list of target problems (TPs) representing usually the presenting complaint and of target problem procedures (TPPs). These lists identify what it is that therapy aims to change. Subsequent therapeutic work, whether behavioural, cognitive or interpretive or some combination of these, will always be related to these descriptions. Patients are given the task of focused diary keeping in relation to these descriptions to enable them to recognise and hence change their recurrent use of these ineffective procedures. Change in respect of the defined problems and procedures will be rated by patients regularly through the rest of therapy.

Target problem procedures define for the patient his appropriate responsibility for his difficulties while diminishing his sense of powerlessness, confusion and irrational guilt and they serve to emphasise that both conceptual and behavioural change are called for. For the therapist, the list provides a clear guide to his interventions and a basis for choosing methods and for assessing progress. In more disturbed patients, only selected identified problems may be tackled in the course of the brief therapy but the patient's ability to work and the effects of such work, will provide a basis for deciding whether further treatment is necessary or helpful. This approach minimises the risk of regressive dependency on the part of the patient and guards against a premature or unnecessary commitment to longterm therapy.

Brief psychotherapy in crisis intervention

Dr Shakir Ansari, St Thomas' Hospital, London

The work of a 'crisis intervention team' involving domiciliary assessment and management is described, as part of a comprehensive district-based service. A wide spectrum of psychiatric disorders are seen and treated, ranging from

psychotic illness, to major depression, neuroses and adjustment reactions. Treatment is broad-based, including medication and psychological methods.

It has been noted that the use of psychotherapeutic techniques are of value, being commonly employed. Using the crisis team at the mental health advice centre as a model, it can be seen that psychotherapy, especially brief or time-limited, fits in well with the idea of a comprehensive district-based psychiatric service.

A trainee's experience and opinion

Dr B. J. Brockman, Guy's Hospital, London

The paper addresses the historical setting of a 7 year training period in South East Thames as preparation for work as a general adult psychiatrist. The difficulties arising in training will be discussed, paying particular attention to personal and temporal issues. The format of therapy training will be discussed with reference to different therapeutic techniques, supervision, formal theoretical teaching and a research project. The application of the therapeutic techniques will be discussed in relation to work as a general psychiatrist. In conclusion, the relationship of therapy training and work in the NHS setting is discussed.

Poster presentations

Psychological sequelae of kidney donation: A 5-10 year follow-up study

Drs Vimal Kumar Sharma, Fazakerley Hospital, Liverpool and M. David Enoch, Royal Liverpool Hospital, Liverpool

Twenty-three out of 30 potential kidney donors were followed up after a period of 5-10 years. Fourteen were donors and the other nine subjects who were not allowed to donate kidneys on medical grounds constituted the control group (non-donors). Psychiatric morbidity was assessed by using the Present State Examination (PSE) Interview Schedule. CATEGO-ID Programme identified one case in the donor group and one case in the non-donor group. The psychiatric problems of both the cases were unlikely to be related to the kidney transplantation. The majority of the donors, irrespective of the outcome of the transplant procedure, expressed positive feelings towards kidney donation. This study suggests that the kidney donation does not cause adverse long-term psychological sequelae.

The use of electroconvulsive therapy in the elderly

Drs S. M. Benbow and D. J. Jolley, Central Manchester Health Authority

A retrospective study was carried out of 122 patients who received 193 courses of ECT under the care of the psychogeriatric service at the University Hospital of South Manchester between 1976 and 1982. The characteristics of the patients, their treatment details, response and outcome are described. In most cases (96%) the major diagnosis was a depressive illness. Bilateral ECT was administered throughout 95% of courses with a mean of 8.3 treatments per course. Although many patients had a physical problem (51%), serious complications of treatment were rare and 80% of patients were discharged well or substantially improved. About one-third of patients had another one or more courses of ECT during the follow-up period. Subsequent courses of ECT were not likely to be longer than the initial course nor was ECT less likely to be effective. It is

concluded that ECT is an effective and safe treatment in depressed elderly people.

Brain perfusion during epinephrine induced anxiety

Drs R. J. Mathew and W. H. Wilson, Duke University Medical Center, USA

Regional cerebral blood flow (rCBF) was measured with the ^{133}Xe inhalation technique twice at an interval of 30 minutes in 30 drug-free patients with a diagnosis of generalised anxiety disorder (based on DSM-III and SADS) and an equal number of normal volunteers. One-half of the patients and controls received intravenous infusion of 0.2 mcg/kg per minute of epinephrine in saline for five minutes immediately before the second measurement. The remaining subjects received saline infusion between the two measurements. The subjects were assigned to epinephrine and saline groups on a random basis and infusions were given under double-blind conditions. All measurements were carried out in a semi-dark, quiet room under identical laboratory conditions. During each rCBF measurement blood pressure, pulse rate, end-tidal levels of carbon dioxide, state anxiety (State Trait Anxiety Inventory), somatic symptoms of anxiety (with a standardized rating scale) and hematocrit were measured. The two groups of subjects were compared on these indices and their inter-relationships were examined. The anxiety patients who received epinephrine showed a significant increase in state anxiety and decrease in both right and left hemispheric flows during the second run. This finding persisted even after the effects of carbon dioxide changes were partialled out. The rCBF decrease was most pronounced in the left hemisphere. Correlations between anxiety related clinical and physiological indices and rCBF (resting and after epinephrine) were computed in both groups of subjects.

A double-blind trial of carbamazepine and haloperidol in mania

Dr D. Brown, Professor T. Silverstone and Dr J. Cookson, St Bartholomew's and the London Hospitals

Is carbamazepine (CBZ) as effective as haloperidol (HP) in the treatment of acute mania and is it as well tolerated? To answer these questions 17 hospitalised manic patients fulfilling DSM III criteria for mania were included in a double-blind trial of CBZ (starting dose 400 mg/day max. 1600 mg/day) and HP (starting dose 20 mg/day max. 80 mg/day). Procyclidine (PCD) up to 30 mg/day was allowed for extra-pyramidal symptoms. If required, chlorpromazine (CPZ) could be used as a 'rescue' drug. Clinical ratings using the Young and the Petterson mania scales were made on days, 0, 1, 3, 7, 14, 21, 28 and 42.

Seven of the nine patients on HP compared to two of the eight on CBZ failed to complete the four week trial. Failure to complete occurred either because of side effects (3HP) lack of efficacy (2 CBZ, 3 HP) or a switch to depression (2 HP). HP appeared to act more quickly; the mean Young score fell from 29 to 20 in three days compared to no change in the CBZ group. Thereafter the CBZ group improved steadily with the Young score falling from 31 to four in four weeks. Extra-pyramidal side effects were common on the HP group (accounting for three dropouts); gastrointestinal symptoms were more prominent in the CBZ group, but did not lead to any drop outs.

While the two drugs were broadly similar in terms of efficacy, tolerability was noticeably greater with CBZ. CBZ

is thus likely to prove a useful drug in the management of mild to moderate mania

Anticonvulsant medication in a mental handicap hospital: 1972-1982

Dr L. C. Sheppard, University of Dundee

The prescription sheets for all patients in a mental handicap hospital on one day in 1972 and on the same date in 1982 were examined to identify trends in anticonvulsant medication over a 10 year period. There was a reduction in size of the hospital population associated with fewer very young and mildly handicapped persons. The prevalence of anticonvulsant prescribing fell slightly over the decade under study with a dramatic fall in the use of phenobarbitone. This decline is paralleled with an increase in carbamazepine prescribing. Other changes include the use of sodium valproate in a significant minority of patients and the occasional use of clonazepam in 1982, while phenytoin remains a first rank anticonvulsant throughout the period under review. The proportions of patients on polytherapy have not changed over the 10 year period, though the contribution of phenobarbitone to the combined drug regimes has reduced significantly.

Practice in a provincial drug dependency clinic

Dr J. Marks, Halton General Hospital, Runcorn, Cheshire

Widnes and Runcorn (pop. 150,000) have had a drug dependency clinic offering a maintenance opiate management for several years. Bootle had no such clinic until 1985, but counselling, withdrawal and detoxification had been available. It was hypothesised that illicit drug use would have spread wider in Bootle and Widnes because of this difference in service provision and that referrals would reflect this. This was found to be the case. Follow-up of chronic drug addicts in Widnes is reported. This study confirms Vaillant's view of drug dependency as a chronic relapsing condition with a remission rate of roughly 10% per annum. If the patient can be sustained at a drug clinic, the prognosis reaches a 50% likelihood of recovery after 10 years.

Treatment of alcoholism in Kuwait: A prospective follow-up study

Drs A. M. Bilal, J. Kristof, A. Shaltout and M. F. El-Islam, Kuwait University

The outcome of routine drug supportive psychotherapy treatment of 100 patients with alcohol-related problems in Kuwait's only psychiatric hospital was evaluated on four six-month intervals. Patients were assessed on nine post-treatment questions which reflect patient's improvement or deterioration. Correlation of follow-up changes to pre-treatment patient data shows that non-Kuwaitis do better than Kuwaitis in resuming employment and social re-integration. Patients on chronic benzodiazepine prescription were less likely to become abstinent, had poor social re-integration and more re-admissions. Good relationships with the family of origin were clearly associated with a better treatment outcome.

Scores of weighted follow-up findings were calculated to assess the patient's status at each follow-up. The patient's progress during the whole follow-up period was measured by the direction of slope calculated from linear regression model for individual scores. The relationship between pre-treatment conditions and patterns of follow-up was

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evaluated cross-sectionally (mean scores) and longitudinally (direction of slope). A pattern of shift emerged where different patients' groups crossed over to the opposite end of score scale. Kuwait society's indulgent attitudes towards Kuwaiti patients with alcohol related problems in employment have to be changed and utilisation of the role of the Arab extended family in therapy is stressed.

Psychiatric emergencies at King Khalid University Hospital *Dr A A El-Gaaly, Faculty of Medicine, Riyadh, Saudi Arabia*

In this study, 100 consecutive emergency psychiatric cases are studied. These were all new referrals from the accident and emergency department to the psychiatric team. Demographical, clinical data, as well as management offered are compared for males and females. The total group are compared with the results of a British study. Differences are discussed. The findings suggest that a psychiatric emergency service should be available to all referrals 24 hours a day. It is not only a needed service, but it also offers important learning opportunities to residents and staff.

Minor physical anomalies as a biologic marker for behavioural disorder

Dr J C Pomeroy, SUNY, New York, USA

The importance of subtle neurologic or neurophysiologic abnormalities as predictors of childhood and adult psychiatric disorders or indicators of underlying organic brain disorder in behaviourally disturbed children is of con-

siderable dispute. Minor congenital deviation in physical characteristics, believed to represent first trimester insults to the foetus, have shown a relationship with childhood behavioural and educational problems as well as psychosis in all ages. Early enthusiasm for the measurement of minor physical anomalies to indicate future disturbance and need for intervention has been somewhat tempered by more recent studies.

In the course of a comparison study between children designated emotionally disturbed or learning disabled by the educational system, an opportunity arose to measure physical anomalies in this population. The school nurse rated 93% of the 139 pupils for physical anomalies in the centre for emotionally disturbed grade schoolers. Sixty-eight per cent of this population were also rated by a second examiner. Scales assessing behaviour, medical history and social characteristics were completed by the teachers, social workers and school nurse. Demographic information as well as intellectual and educational evaluations were available from school records.

Nineteen girls and 111 boys aged 5-12 years (mean 9.4 years) with a mean IQ of 88 (ranged 42-126) were examined. All were designated emotionally disturbed. Inter-rater agreement for the presence of anomalies was comparable to other studies but exposed the subjectivity of a number of the measures. The level of anomalies was not as high as had been found in clinically diagnosed groups, but using the data available from this study, and from a review of the literature, an assessment of the relevance of physical anomalies for specific behavioural syndromes will be addressed.

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